

**Final Report to Fundação Bial on the Project
“A Controlled Analysis of Subjective Paranormal Experiences
in a Neuropsychiatric Population”
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John A. Palmer, Ph.D., and Vernon M. Neppe, M.D., Ph.D.

We completed the first stage of our project in June, 2001. The results of this stage were included in the interim report we mailed to the Foundation at that time. Dr. Palmer presented this interim report as a paper at the 2001 Convention of the Parapsychological Association, which was held in New York City August 1-4. The slightly revised interim report comprises Part I of the detailed report accompanying this letter. Part II, which begins on page 17, describes the work completed since the submission of the interim report. Part II of the report will be submitted for presentation at the 2002 Convention of the Parapsychological Association, to be held in August in Paris. A poster describing the entire project will be presented at the Foundation's 4th Symposium to be held April 4-6, 2002 in Portugal. We also anticipate journal publications based on this research.

To summarize briefly, each of 100 patient files were coded for reported subjective paranormal experiences (SPEs), including ESP experiences, apparitional experiences, and out-of-body experiences, and for 4 distinctive markers of temporal lobe dysfunction (TLD) in the brain: (i) specific symptoms as reported by the patient on the INSET questionnaire, (ii) EEG abnormalities, (iii) response to anti-convulsant drugs, and (iv) predisposing conditions, including head traumas such as concussions and brain tumors, and use of some recreational drugs. Scores on these 4 elements were summed, and the sums were used to divide patients into a TLD group and a control group. As predicted, the TLD group reported significantly more SPEs than the control group, but this result was entirely attributable to the INSET questionnaire, which by itself predicted SPEs to a highly significant degree. It was also found that females both were more likely to be classified as having TLD and to report more SPEs than males.

The second stage of the project consisted primarily of logistic regression analyses that were used to predict subjective ESP (S-ESP) experiences, one category of SPEs, from subcategories of the predictors used in the first stage. These included gender, age, the 16 individual INSET items, specific EEG variables reflecting the location and type of abnormal EEG waveforms, use of specific recreational drugs, head traumas, and measures of brain laterality. The final model indicated that S-ESP was positively associated with females, right laterality (left hemisphere dominance), jamais vu experiences, and visual/auditory hallucinations. An interaction with gender was found indicating that EEG abnormalities restricted to the temporal lobes, and sometimes extending to the frontal lobes, was positively related to S-ESP in females but negatively in males. This finding confirms our hypothesis of a positive association of TLD and SPEs, but only for females. The EEG analyses indicated that S-ESP experiences are most prevalent among right-handed females with high frequency EEG abnormalities or spikes in the temporal area of the dominant (left) hemisphere. This pattern was subsequently

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found to coincide exactly with that of a gifted ESP subject when she was engaged in a successful remote viewing session.

Palmer also interviewed 20 of the patients who reported at least a moderate number of S-ESP experiences on INSET. The main purpose of the interviews was to confirm that these patients had the type of S-ESP experiences considered credible by parapsychologists. These analyses were reassuring in that the great majority reported credible experiences. Most of these patients indicated that the ant-convulsant drugs that were helping to relieve their seizures and other neurological symptoms also tended to suppress the frequency of their S-ESP experiences.

For more details about this research, please consult the accompanying full report.

A CONTROLLED ANALYSIS OF SUBJECTIVE PARANORMAL EXPERIENCES IN
TEMPORAL LOBE DYSFUNCTION IN A NEUROPSYCHIATRIC POPULATION

John Palmer

Rhine Research Center, Durham, NC

Vernon Neppe, Heidi Nebel, and Stacie Magill
Pacific Neuropsychiatric Institute, Seattle, WA¹

ABSTRACT: Previous research by Vernon Neppe and Michael Persinger has provided evidence for an association between subjective paranormal experiences (SPEs) (Neppe, 1980a) and the temporal lobes of the brain. These results consist of correlations between scores on questionnaires asking subjects about personal experiences such as ESP, out-of-body experiences (OBEs), mystical experiences and apparitions, and positive responses to questions about symptoms characteristic of temporal lobe dysfunction (TLD). However, this research has been restricted to "normal subjects" whose symptomatology was not of such a degree as to classify them as having temporal lobe disease. The purpose of this project was to see if these results could be replicated with a clinical population. The sample consisted of 100 neuropsychiatric patients of Dr. Neppe. Computerized files were blindly rated by two raters, one of whom rated for TLD diagnosis and one for SPEs. TLD diagnosis was based on four criteria: (1) responses to 16 items from Neppe's short INSET questionnaire that reflect various symptoms characteristic of TLD; (2) etiological predisposing factors including (a) brain insults such as concussions, tumors, and encephalitis and (b) recreational use of certain pleasure drugs, (c) results of waking, sleeping, and ambulatory EEGs, and (d) response to prescribed anti-convulsant (A-C) medications. Scores on these criteria were summed to form a scale with scores potentially ranging from -3 to 11. The patients were also classified separately by their treating physician (Neppe). 60 patients scoring 6 or above were classified as having TLD, 27 with scores of 4 or less were classified as control, and 13 were dropped: 10 for scoring 5, and 3 for disagreements between Neppe and Palmer on final codings. SPE scores were based on 4 item scores from the INSET addressing frequency of ESP experiences, OBEs, and "sense of presence" (apparitions). In both analyses, in support of the hypothesis, the TLD group had a significantly higher mean on the SPE scale than the control group, $p < .05$, one-tailed. However, when gender was introduced as a covariate in an analysis of variance, the TLD hypothesis was no longer supported. A multiple regression analysis predicting SPEs from the four individual TLD criteria plus gender and using all 100 patients indicated significant, independent contributions to the prediction of SPEs by only gender ($p = .004$) and INSET ($p < .001$). Thus, this confirms that the TLD hypothesis is due entirely to the contribution of the INSET component, a result which confirms earlier research by Neppe with a non-clinical population. A second analysis using revised SPE scores reflecting a variety of different kinds of ESP experiences and weighted more heavily by the ESP item gave almost identical results to those with the original SPE scores. The 4 TLD components did not correlate significantly

¹ This research was supported by a grant from the Bial Foundation in Portugal.

among themselves, suggesting they were not measuring the same thing. Reasons were discussed as to why support of the TLD hypothesis was not stronger, and plans were noted for new exploratory analyses to address these possibilities. Nonetheless, the strong relationship in temporal lobe dysfunction patients of temporal lobe symptomatology with SPEs is a major finding.

Exploratory logistic regression analyses were conducted to uncover relationships between specific predictor variables and patient claims of subjective ESP experiences (S-ESP) on INSET. S-ESP was chosen as the dependent variable because it more directly implies a paranormal process than do the other SPE variables, although no claims are made for the evidentiality of the experiences. Eliminating those who claimed S-ESP only rarely left an S-ESP group ($N=53$) to be compared with a No-S-ESP, or control, group ($N=40$). The independent variables included gender, age, the 16 INSET items, EEG measures reflecting the location and type of anomalous activity, measures of handedness and brain laterality, specific recreational drugs, and head injury. All variables besides gender were evaluated controlling for gender, and their interactions with gender were also calculated. The only variables that proved to be significant ($p < .10$, two-tailed) in the final model were gender, laterality, and (from INSET) the jamais vu item and the combined visual and auditory hallucinations items. Thus, the ESP group was characterized by right-lateralized females who scored high on the selected INSET items. A significant interaction was found between gender and a measure of EEG anomalies that occurred in the temporal lobes and sometimes extending to adjacent area, but not generalized over the whole scalp. These anomalies were positively related to ESP in females and negatively in males. More refined analyses indicated that the effect for females was contributed entirely by activity other than slowing (mostly spiking, sharp waves, and bursts of fast beta or alpha) that occurred in the left hemisphere, sometimes extending bilaterally to the right temporal, or the frontal lobes. Redefining the temporal EEG variable in this way left the statistical significance of the reverse effect for males unchanged. Significant ($p < .10$) predictors of S-ESP in a regression model for females were the revised temporal EEG measure, laterality, and visual/auditory hallucinations. The number of males in the sample ($N = 27$) was considered too small for a meaningful regression analysis. As far as the brain is concerned, S-ESP appeared to be most prevalent among right-lateralized females with relatively high-frequency EEG anomalies in their dominant (left) hemisphere. It is recognized that all these exploratory findings need to be cross-validated with a new sample before the results can be considered conclusive.

Palmer interviewed 20 patients from the ESP group to get a sense of the credibility of the S-ESP experiences they claimed, and whether they could detect any effect of anti-convulsant (A-C) drugs on the frequency of these experiences. He found that 13 of the 20 had credible experiences, 4 had marginally credible experiences, and 3 had non-credible experiences (2 of these 3 later told Neppe they had under-reported their ESP experiences to Palmer.) Palmer found that 8 of the 14 patients who were taking A-C drugs claimed they suppressed the frequency of S-ESP, 2 claimed

enhanced frequency, and 4 claimed no difference. Neppe's independent assessments, based on his notes and recall of patient interviews, resulted in slightly stronger trends toward credible S-ESP experiences and a dampening effect of A-C drugs than found by Palmer.

PART I. PRIMARY ANALYSES

Our understanding of psi from a physiological point of view would be greatly enhanced if we could pinpoint a section of the brain in which psi mediation occurs, or at least an area that plays a primary role. Such knowledge would provide at least three concrete benefits. First, by considering the functions performed by this part of brain, we could develop more incisive insights about how psi manifests. For instance, if the area plays a crucial role in the activation of memories, credence would be lent to the hypothesis that psi occurs by activating stored memories (Roll, 1966). Second, if momentary brain states could be found to correlate with the accuracy of discrete psi responses, progress could be made in predicting which particular psi responses (e.g., guesses on a card test) will prove to be correct. Third, attempts could be made through biofeedback, drugs, or other means to alter the functioning of this part of the brain to enhance psi performance.

Although only a limited amount of research has been directed toward determining which part of the brain is most important for the mediation of psi, this research points consistently to one area, the temporal lobes. Before reviewing this research, it should be pointed out that it deals exclusively with subjective psi experiences (SPEs) mostly ESP, that occur in the real world rather than the controlled setting of the laboratory. It is widely recognized that SPEs are more amenable to alternative, non-psi explanations than are statistically significant results emerging from properly conducted laboratory experiments. However, the fact that the psi process has been shown to exist in the lab makes it plausible to assume that it occurs sometimes outside the lab as well, that is, that some SPEs involve genuine psi. Certainly, many, perhaps most, have more mundane explanations, but it is also true that most of the "hits" in laboratory experiments are not due to psi, but rather to chance. Moreover, one can logically evaluate SPEs for the likelihood of alternative explanations and thus single out those that seem most likely to be psi-mediated. In both types of research, the problem boils down to one of false positives, which from the statistical point of view function as noise. One implication of this fact is that large samples will be needed to detect real effects, and it is easier to get large samples of SPEs than successful examples of psi in the lab. This is not meant to denigrate lab approaches to the brain/psi interface; in our opinion both lab and spontaneous case approaches have an important role to play, and the best evidence will come from a convergence of results on a common conclusion. Finally, even if it turns out that research on brain processes and SPEs tells us only about SPEs as *experiences*, it is still an important contribution to knowledge (Neppe, 1990a, 1990b).

The earliest example of an exploration of the relation between SPEs and the temporal lobes is an uncontrolled study by Nelson (1970), who found that 10 of 12 trance mediums had evidence of temporal lobe instability in their EEGs. Subsequently, Nelson and Neppe (1980) failed to replicate these EEG findings in a population from the South

African Society for Psychical Research (SASPR) studied by Neppe (see below). Roll (1977) suggested a link between epilepsy and poltergeist activity, based on the fact that 22 of 92 focal persons of such activity that he surveyed were prone to “seizures or dissociative states” (p. 400). In one particularly noteworthy case (Solfvin & Roll, 1976), poltergeist outbursts appeared to alternate with seizures in a grand mal epileptic.

The first controlled study to investigate the relationship between temporal lobe instability and SPEs was by Neppe (1979, 1980b, 1981a, 1982, 1983b), who analyzed the SPEs reported by all members of the SASPR. He found that a core group of six members reported large numbers of SPEs according to pre-stipulated criteria. These persons had significantly more possible temporal lobe symptoms than a group of six control subjects from the same society who reported no SPEs. Data were collected from interviews and detailed questionnaires. The latter included the Neppe Temporal Lobe Questionnaire and several SPE questionnaires, derived in part from a questionnaire developed by Palmer (1979). These instruments were administered to subjects verbally. The findings demonstrated a link of temporal lobe functioning to SPEs at both a state and a trait level, and also suggested a link of seizure type phenomena with these experiences.

Neppe later delineated a particular type of olfactory hallucination associated with SPEs that is pleasant and perfumy but commonly co-exists with temporal lobe type hallucinations (Neppe, 1983c, d). This again supported the trait link of temporal lobe firing with SPEs. Finally, he demonstrated that the temporal lobe epileptic, the subjective paranormal experient, and the normal subject each describe a specific subtype of déjà vu experience (Neppe, 1981a, 1983a).

Persinger has published extensive research and theorizing on the role of the temporal lobes in psi experiences. He attributes SPEs to instability or micro-seizures in the temporal lobes, particularly the hippocampus and amygdala (Persinger, 1989). He tested this hypothesis by giving a 29-item scale of temporal lobe symptoms and a scale measuring seven different types of SPEs (including telepathy, out-of-body, and spiritual experiences) to groups of 108 and 41 college students (Persinger, 1984). These scales were a direct derivation of Neppe’s original Temporal Lobe and SPE questionnaires. (Neppe, 1981a, b, c) In both samples there were significant positive correlations between scores on the temporal lobe scale and number of different kinds of SPEs reported. This research was replicated and extended with a sample of 99 college students, who were given a 16-item scale measuring complex partial epileptic signs (CPES) in addition to the scale used previously (Persinger & Vaillant, 1985). Both scales yielded significant positive correlations with the number of different kinds of SPEs. It is also noteworthy that persons engaged in artistic professions score high on Persinger’s temporal lobe scales (Persinger & Makarec, 1993) and also show exceptionally positive results in ESP Ganzfeld experiments (Palmer & Broughton, 2000).

Persinger’s TL scales have been validated in two ways. First, it was shown that epileptics score substantially higher than controls on the scales (Persinger & Makarec, 1993). Second, amount of EEG alpha activity in the temporal lobe, but not the occipital lobe, was greater among high than low scorers on Persinger’s scales (Makarec and Persinger, 1990). However, the scales do not show particularly good discriminant validity as measures of temporal-lobe epilepsy. Clinical groups suffering from post-traumatic stress disorder, anxiety-depersonalization, and “exotic dissociation” scored

quite high on the scales (although not as high as the epileptics), and the scales correlate extremely highly (.72 to .83) with the Bernstein-Putnam Dissociative Experiences Scale (Persinger & Makarec, 1993).

All the above data involving temporal lobe symptoms and SPEs were collected from normal populations. If the relationship between these variables is truly linear, one would expect temporal lobe epileptics to have even greater amounts of SPEs than normals who happen to score high on the kinds of scales discussed above. However, this remains to be established. Persinger (1989) argues that repeated epileptic motor seizures destroy brain tissue and thus might actually reduce the incidence of SPEs. Also, the only relevant study we could find in the literature involving diagnosed temporal lobe epileptics found no tendency for them to have more mystical experiences than control groups of primary generalized epileptics and patients suffering from migraine (Sensky, Wilson, Petty, Fenwick, & Rose, 1984). However, the samples in each were group were small in this study ($N < 30$) and the measurement of SPEs did not appear to be very thorough. Clearly, more research is needed to settle the issue.

An opportunity to derive more information from clinical populations is provided by Neppe, who sees many temporal lobe epileptics in his clinical practice at the Pacific Neuropsychiatric Institute in Seattle. He collects enormous amounts of data from each individual, including etiology, current symptoms, EEG data, and responses to anti-convulsant (A-C) drugs. His symptom questionnaire, which is administered orally, includes questions on SPEs. This Inventory of Neppe of Symptoms of Epilepsy and the Temporal Lobe (INSET) serves as the basis for detailed analysis of every positive reported symptom of temporal lobe disease, based on criteria developed initially by Neppe (1979, 1981 a, b, c) and later by Neppe and Tucker (1988, 1992).

Although it is Neppe's subjective impression that his TLD patients have an unusual number of SPEs and more than his other patients, time constraints have prevented him from analyzing these data quantitatively. The purpose of the proposed project is to remedy this deficiency through a collaborative effort involving other qualified individuals who have the time and expertise to complete the task.

Method

Selection of Patients

The initial plan was to evaluate 80 patients and to do a preliminary analysis of the hypothesis after the first 40. However, it soon became apparent that a larger proportion of patients were falling into the TLD group than was anticipated, raising the concern that the control group would be too small to properly analyze. It was thus decided to raise the total sample size to 100 patients and forgo the preliminary analysis. Palmer (JP) and Neppe (VN), who at the time were blind to the patients' SPE scores, made this decision.

Neuropsychiatric patients (with neurological as well as psychiatric symptoms) were selected from among those seen within the previous 6 months. In most cases, follow-up sessions with such patients were anticipated. The folders of these current patients were already arranged alphabetically by last name in the files. Patients were entered into the sample sequentially. An initial batch of 75 patient files passing the

original cut was selected by VN. Additional batches totaling 36 files were later supplied to the raters. The latter batches consisted of cases VN had just completed reports on and were selected in the order of the report completion. Thus, they were more recent than the first batch. The total number of patients exceeded 100 because 11 cases were later found to be invalid and had to be replaced. 8 of these were found to have met the exclusion criteria, and 3 were found to be duplicates of first-batch patients that mistakenly crept into the subsequent batches.

All the included patients gave informed consent to have their files used for the research. Only 6 qualifying subjects were excluded prior to processing because of absence of informed consent. Prior to inclusion in the original sample, VN also excluded cases that he knew met the following exclusion criteria: (a) under age 18 as of 1/1/2000, (b) electroconvulsive therapy within the past 6 months, (c) a major psychological disorder (mental retardation, active psychosis, dementia or malingering), and (d) insufficient investigation despite full reports: this usually involved absence of EEG data during the evaluation or in the preceding six months. Approximately 15 to 20 cases were excluded by these criteria.

Preparation of Computer Files

At the outset, to preserve anonymity, the names of patients were converted to their initials plus a digit.² VN's Microsoft Word computer files of his patients are extensive, ranging in size from about 40 to about 120 typed pages. Only some of the information in the files was relevant to the coding criteria. It was decided to have JP code the files for neurological problems and HN for SPEs and to keep each rater blind to the material coded by the other. To facilitate this blindness, the files were edited to remove references to SPEs from the files sent to JP by using the "find" command in Microsoft Word. Likewise, information about TL and seizure symptoms was removed from the files sent to HN.³ All this editing, including the removal of patient names, was done by HN for the 82 patients and by SM for the last 18 patients.⁴ These files were then checked by VN to ensure that the editing was done properly.

It was admittedly less than ideal for HN to edit the initial 82 files for neurological information, as this was the information she was supposed to be blind to. Unfortunately, staff time and number of staff assigned to the project required this arrangement. To mitigate the problem, HN edited by removing whole sections located by the Word "find" command without reading them, deliberately did not read the folder at all thereafter, and edited the whole set such that her own seeking of SPEs was done at a time weeks or

² All names were changed to initials and numbers (first in the series, 1 and if a duplicate initial, 2, then 3, etc.).

³ To create profiles for SPE ranking (for HN), all INSET questions not pertaining to SPE, déjà vu, or presences, plus any temporal lobe terminology, seizure terminology, and diagnosis information, were extracted. For the ranking of temporal lobe symptomatology (for JP), all information about SPEs, ESP, PK, intuition, presences, déjà vu, OBEs, and auras was extracted, leaving all temporal lobe and diagnostic information.

⁴ At the time of the editing, both HN and SM were employees of the Pacific Neuropsychiatric Institute. SM took over the duties from HN when SM was added to the staff. JP was appointed an honorary staff member prior to the start of the study.

usually months later when she regarded herself as blind. In general, both raters (HN and JP) were instructed to look only at the particular sections of the files containing the information relevant to their respective rating tasks.

Diagnostic Information

There were 4 specific classes of diagnostic information that JP used to assess whether patients were to be assigned to the TL group or to the control group. They will be described briefly below.

Short INSET. The Short form of the Inventory of Neppe of Symptoms of Epilepsy and the Temporal Lobe (INSET) is designed primarily to assess behavioral and subjective symptoms characteristic of TLD and seizures. The part of the INSET relevant for present purposes is a series of 53 multiple-choice questions, each representing a symptom or class of symptoms characteristic of neurological disorders of various kinds. Each question has 7 response choices representing frequency of occurrence. They are: (0) “never”; (1) “less than once per year”; (2) “yearly or more”; (3) “monthly or more”; (4) “weekly or more”; (5) “daily”; and (6) “more than daily”. In some of the results found in the computer files, a different set of response alternatives is reported that translates to the preceding set according to the following transformations: (1-2) “rare”; (3) “occasional”; (4-5) “frequent”, and (6) “very frequent”. All questions are asked both for “recent experience” and for “past experience”.

Of the 53 questions, only 16 refer to symptoms specifically characteristic of TLD. These questions, listed at the top of the Appendix, were the only ones used for TLD classification. In addition, at the end of the INSET are 4 questions referring to SPEs that were coded for this project. Specifically, they address ESP experiences, out-of-body experiences (OBEs), and apparitions. These questions, which provided the SPE scores, are listed at the bottom of the Appendix.

Patients completed the INSET during their first or second visit to PNI. After completing it, VN interviewed them about their responses to each question they answered affirmatively or were unclear about, to elicit further information and to be sure the question was understood as intended.

Absent from the short INSET used in this research are items referring to the particular kinds of déjà vu experiences (Neppe 1981a, 1983a) and olfactory hallucinations (Neppe, 1983c, d) found by Neppe to be closely associated with SPEs. The reason for this decision is that some parts of these experiences themselves either are, or could quickly evolve into, SPEs. Such experiences then cannot be considered independent of SPEs. The INSET is being construed in this research as a trait predictor rather than a state predictor.

Etiology. During the initial interview, summarized in the file under a section labeled “History of Main Complaint”, and as part of the INSET, patients were asked if they ever had a head injury or a brain disease such as encephalitis or meningitis, brain surgery, or brain tumor. They were also asked about the past and current frequency of recreational drug use. The specific drugs inquired about are marijuana, LSD, mescaline,

amphetamines, cocaine, phencyclidine (PCP), heroin, narcotics, alcohol, caffeine, tobacco, and the ever-present “other”. These results are summarized in a table in the files labeled “Abuse History” and summarized briefly in the report of the INSET.

EEG. Patients at PNI are very commonly evaluated electro-encephalographically. Depending on clinical indications, they receive either or both of:

- a. 18 channel wake and sleep EEGs including activation procedures of hyperventilation and photic stimulation .
- b. 2 to 3 days of 16 to 18 channel computerized home ambulatory EEG monitoring with an ongoing record of the patient’s brain waves during waking activities as well as sleep. The technology used is the most sophisticated in the world, involving Sleep-Wake DigiTrace apparatus. Patients or families press a pushbutton when they feel a seizure, spell, event or other anomalous sensation coming on, and these records can be examined in relation to the simultaneous brain-wave activity.

All but 7 of the 100 patients in our sample were given the ambulatory EEG. In 6 of these cases the exclusion was because the sleeping and waking EEGs were both normal and there were no other reasons to think the ambulatory EEG might yield a different verdict. In one case, the patient resisted the ambulatory EEG. The EEG results are given in a section of the file specifically devoted to them and summarized in a subsection under the heading “Diagnosis”.

In a few cases, waking and sleep EEGs were not conducted at PNI because they had recently been done elsewhere or it was felt that going directly to ambulatory EEG was appropriate. In these cases, the reports of these earlier EEGs were considered for the coding. VN read the sleep-wake EEGs. In addition to VN, a second expert, Dr. Donald Schomer, Chief of Electroencephalography at Harvard University and an acknowledged world expert on the DigiTrace technology, interpreted the ambulatory EEG records.

Response to Anticonvulsant Medications. If the previously obtained diagnostic information indicates that the patient is experiencing seizures and or is likely to have TLD, they are generally given one of a variety of anti-convulsant medications. Whether such a medication is to be prescribed and the specific medication(s) of choice are listed in the files under a section called “Specific Recommendations”. Response to these medications is documented in the reports of follow-up visits the patients make to PNI. The number of such visits reported in the files varies greatly from patient to patient, depending in large part on the recency of the initial visit. It is common for the dosage level of the A-C medications to be varied over time, and sometimes patients are changed from one A-C to another. These changes are usually dictated by the effectiveness of the current regimen in ameliorating the symptoms and also the presence of side effects. It is common for patients to be prescribed other drugs, such as anti-depressants or anti-anxiety agents, in addition to the A-Cs.

Diagnostic Coding

At the outset of the project, VN and JP agreed on a preliminary set of coding criteria that were followed for the first patients evaluated. However, it eventually became apparent that some of these initial criteria were producing weird and unrealistic distributions of scores. For instance, the initial scoring scheme for TLD symptoms on the INSET produced an overwhelming proportion of patients given the highest score (3). It was thus decided to modify the criterion for scoring these INSET questions, most notably by downgrading symptoms that appeared infrequently. All such decisions were made by VN and JP based primarily on characteristics of the overall distributions of scores, not scores of individual patients, and, most importantly, without knowledge of SPE scores.

TLD Symptoms (0 to 3). The frequency scores on the 16 INSET items devoted to TLD were transformed as follows:

- 0 = 0 (never)
- 0.25 = 1-2 (rare, yearly or less, etc.)
- 0.50 = 3 (occasional, more than yearly up to monthly, etc.)
- 1 = 4-6 (frequent, more than monthly, etc.)

Each item was scored only once; when the scores for “recent” and “past” differed, the highest was chosen. The item scores were then summed to yield a total raw score, with a possible range of 0 to 16. The raw scores were then transformed to the final classification scores as follows:

- 0 = 0 to 0.75
- 1 = 1 to 3.75
- 2 = 4 to 5.75
- 3 = 6 or higher

Etiology (0 to 2). 1 point was scored if the patient had suffered a head injury, brain tumor, or brain disease (e.g., encephalitis). Head injuries only counted if the patient experienced loss of consciousness, concussion, and/or amnesia.

1 point was scored if the patient had a history of using certain recreational drugs. If the frequency of use of any of the following drugs met the defined standard, the point was scored:

- Marijuana:* Over many years or very frequently over shorter periods of time
- Hallucinogens* (LSD + mescaline + PCP): 3 or more times
- Amphetamines:* 6 months or more, or at least 30 times (unless prescribed)
- Opiates:* 6 months or more (unless prescribed and patient not an addict)

EEG (0 to 3). The EEG reports generally contained information about the number of spikes and paroxysms in the EEG records, their laterality and focality. In most cases, the file also contained evaluative statements of the EEG record regarding its indication of TLD. In these cases, the following formula was used to arrive at a classification score for EEG:

- 0 = “normal”, “do not support” TLD
- 1 = “mildly abnormal”, “weakly support” TLD
- 2 = “abnormal”, “support” or “moderately support” TLD
- 3 = “severely abnormal”, “strongly support” TLD

There were 16 files that did not include evaluative EEG statements. JP had guidelines giving some indication of what specific kinds of abnormalities deserved what scores, but he found this difficult to apply to specific cases. So he decided to extract excerpts of the EEG abnormalities recorded in each file, eliminated any phrases that might identify the patient (e.g., name of physician who had conducted previous EEG examination), removed the patient initials that identified the file, randomized the order of the cases, and emailed them to VN. VN then supplied scores for the 16 cases. He reported that he was unable to identify any of the patients from the material sent.⁵

A-C Response (-3 to 3). The response of patients’ symptoms to A-C medications was coded on a 7-point scale from “much worse” (-3) to “much better” (3). Where applicable, separate judgments were made for seizures and psychological symptoms, and the most positive result was chosen for coding. When there were different results for different psychological symptoms, the average was taken. Side effects, insofar as they could be clearly identified as such, were ignored for purposes of coding. Primary consideration was given to the most recent evaluations and evaluations explicitly linked to an A-C drug.

Patients for whom no A-C drug was recommended nor given by VN were coded 0. Cases in which an A-C drug was recommended but not given (e.g., because of a fear of untoward effects like side-effects or patient resistance) were coded 1.

TLD Classification

Objective Classifications. The classification scores were summed over the 4 diagnostic categories (symptoms, etiology, EEG, A-C response) to yield a TLD score with a possible range of -3 to 11. These were converted to classifications as follows:

- 6 to 11: TLD Group
- 3 to 4: Control Group
- 5: Indeterminate

The indeterminate group was decided upon as a buffer to help protect against misclassification. Patients in this group were eliminated from the comparison testing the main hypothesis.

Clinical Classifications. As the name implies, the purpose of the above coding scheme was to give an objective, quantified basis for patient diagnoses. Blanket diagnostic statements included in the files were not considered in these codings. (Such statements were of limited use anyway, because they predated and thus did not include A-C response). Of course, VN made blanket diagnoses, either explicitly or implicitly,

⁵ JP attempted his own codings of these cases before mailing them to VN. His codings matched VN’s in 10 of the 16 cases. In 5 of these misses, JP was 1 number too low; in the 6th, he was 1 number too high. The final TLD classifications of these 6 patients would not be changed had JP’s codings been used.

during the course of treatment. It is quite possible, if not likely, that classification by the 2 methods could be different for a minority of difficult to classify patients. Thus, it was decided, as a secondary measure, to have VN classify each of the 100 patients as TLD, control, or indeterminate -- mirroring the objective classifications. For this purpose, JP sent VN a final list of the 100 patient file names (initials) along with date of birth and gender. These clinical evaluations were based on VN's recollections, which were clearer for some patients than others. He specifically did not consult the EEG, INSET, SPE, anticonvulsant responsiveness or etiology data, but did review his overall diagnostic assessment.⁶

Final Classifications. After VN completed his clinical classifications, JP sent his codes to VN. VN examined these, noting discrepancies with his clinical classifications. When there were disagreements, he consulted the files and in most cases agreed with JP. However, there were 8 instances where he questioned the validity of JP's codings, 2 on EEG and 6 on A-C response. He sent JP relevant excerpts from these files and JP coded these excerpts blind. Then VN sent JP the file IDs and JP went back to reassess the basis for his codings. He did not automatically agree with his coding of the excerpts, because his original codings had been influenced by statements in the files other than those selected by VN. He decided to change his codings on 5 of the 6 A-C responses, but not on the other A-C response and on 1 of the EEGs. In the 5 A-C response cases, he found that he had simply missed statements in the files that, had he been aware of them, would have affected his original codings. For the remaining EEG case, VN discovered that there was an error in the file: the word "not" was erroneously introduced into a statement that should have read "support" TLD. (This is credible, because a companion statement immediately below the one in question said that the EEG results support prescription of anti-convulsant drugs.)

The net result was a reclassification of 5 patients from JP's original codes: 2 went from control to TLD, 1 went from TLD to control, 1 went from indeterminate to control, and 1 went from control to indeterminate. At this point, JP and VN agreed on the classification of 97 of the 100 patients. 2 remaining patients, who were not among the 7 questioned by VN earlier, had both been coded as control by JP; 1 was coded indeterminate and the other TLD by VN. The third (who had been shifted to the control group by JP) was a difficult patient to code because of A-C drugs being used for bipolar illness and possible temporal lobe symptomatology. It was agreed by JP and VN to treat him, like the other 2, as indeterminate for the test of the hypothesis.

SPE Scores

Generation of the SPE scores involved coding the frequency of the 4 INSET questions for SPEs, using essentially the same coding as for the INSET questions for TLD:

- 0 = 0 (never)
- 1 = 1-2 (rare, yearly or less, etc.)
- 2 = 3 (occasional, more than yearly up to monthly, etc.)

⁶ VN spontaneously recalled more than 90 of the patients in detail. Those that he did not were generally patients who had not consulted him for a year or more and whose case histories had not required review during that time.

3 = 4-6 (frequent, more than monthly, etc.)

We decided to combine the two ESP questions because a few of the sub-items overlapped and we wanted to avoid double scoring any experiences. Each of the three remaining items was scored only once, using the higher of the responses in either the “recent” or “past” column. This gave a possible range of 0 – 9 for the raw SPE scores.

Per pre-defined protocol, HN had reviewed the whole chart for SPEs. In three instances, she had already recorded file notes of SPEs for ESP and presences, described by patients after taking the INSET.

Revised SPE Scores. The combined ESP question covered various possible SPEs yet still received only three possible points, so it may have reflected too low an ESP score. Because of this, it was decided, in a second analysis, to add 1 point for each positive response to (i) premonitions, (ii) psychic, (iii) intuitive, (iv) paranormal experiences, (v) knowing the future, (vi) having dreams which came true, (vii) strange feelings which came true, (viii) felt to have seen events that happened at a great distance before or while they were happening, and (ix) been in touch with someone when they were far away or dead. Consequently, subjects could score up to 11 on the ESP item (3 for frequency, 9 items less the first, which is incorporated as a minimum.) The out-of-body experience question and presences item were single items.

It is possible that patients could have identified the same experiences through more than one category, which could lead to scores that are too high. However, there is no way to tell when that does and does not apply, and it was hoped that any bias would be random and cancel itself out across subjects. The absolute values of the scores are inconsequential for the project; they are only meaningful relative to each other.

There were 13 patients for whom HN needed additional information or guidance to render a proper coding for this further analysis. 1 related to the absence of the formal INSET items in the file; this was solved by consulting the original source material (reviewed by SM and VN). 2 were for blank data, which was correct because no SPEs were reported for the relevant items. 3 were for information that was incompletely pasted; VN located those and re-pasted with the items required by the protocol. The questions for the remaining 7 patients related to frequency data involving a total of 9 items between them (5 patients with 1; 2 with 2). All these were satisfactorily resolved by VN referring back to the notes. In 1 case, the patient had reported phases of very frequent OBEs so it was scored a 3; in 3 others, there were data on subject frequency elsewhere in the file. In the remaining 5 instances, no indication of frequency could be found, so a score of 2 was assigned as a default, 2 being the mean, median, and mode of the possible frequency scores of 1, 2 and 3. VN then checked HN’s codings broadly so as to ensure the interpretation was correct.

Results⁷*Original SPE Scores*

SPEs. The mean SPE score was 2.54 ($SD = 2.46$). The distribution shows a pronounced positive skew. 61% of the sample claimed at least 1 ESP experience, 29% claimed at least 1 OBE, and 35% claimed at least 1 encounter with a presence. The scores of the 3 questions were moderately intercorrelated, with Spearman correlations ranging from .345 to .536.

TLD Components. The distribution of classification scores for the 4 components of TLD are presented in Table 1, and the relationships among them are presented in Table 2.⁸ Although these relationships are generally positive, only the one between EEG and A-C response is substantial and significant. The size of this effect is due to the fact that patients not prescribed an A-C medication were coded 0 on A-C Response, and the decision not to prescribe was heavily influenced by a normal EEG. When these patients are excluded (see last column of Table 2), the relationship vanishes.

Table 1
Frequencies of Diagnostic Codes

	0	1	2	3
INSET	9	32	18	41
ETIOLOGY	47	44	9	
EEG	28	14	24	34
A-C RESPONSE	23 ^a	16	36	25

^a Includes 1 score of -1.

Table 2
Relationships Among Predictors Expressed as
Spearman Correlation Coefficients

	INSET	ETIO.	EEG	A-C RSP.	A-C RSP (2) ^a
INSET		.080	.120	.110	-.083
ETIOLOGY			.081	-.011	-.057
EEG				.361 ***	.000
SEX	.285**	-.198*	.252**	.148	-.050

^a Only patients who were prescribed an A-C medication, $N = 80$.

*** $p < .001$; ** $p < .01$; * $p < .05$

⁷ All p values are two-tailed unless noted otherwise.

⁸ Prior to analysis, subgroups of less than 10 were combined with an adjacent group.

TLD Hypothesis. The 60 patients assigned to the TLD group had a mean SPE score of 3.05 ($SD = 2.69$). The 27 patients in the control group had a mean SPE score of 1.93 ($SD = 1.90$). The difference is marginally significant by the non-parametric U -Test ($U = 1021, p = .049$, one-tailed). Thus, the TLD hypothesis is confirmed. (Recall that the remaining 13 patients were classified as indeterminate.)

Gender. JP noticed during his coding of symptoms that the patients he was assigning to the TLD group tended to be female and those he was assigning to the control group tended to be male. This observation was confirmed in the formal analyses, where it was found that 47 of the 60 females (78.3%) were assigned to the TLD group and 14 of the 27 males (51.9%) were assigned to the control group, $\chi^2(1, N = 87) = 7.93, p = .005$. JP also suspected that females would report more SPEs than males, and this was confirmed by the data as well. The 60 females had an SPE mean of 3.35, compared to 1.26 for the 27 males ($U = 1054, p = .005$). This, of course, suggests that gender has a strong potential to confound the TLD-SPE relationship. To assess this, an analysis of covariance was performed between TLD category and SPE, with gender as a covariate. As expected, the TLD-SPE relationship became nonsignificant, $F(1, 84) = 0.91, p = .344$. Thus, with gender controlled for, the TLD hypothesis is not confirmed.

Analysis of Component Predictors. To assess the independent contributions of the components of TLD to the TLD-SPE relationship, these components (INSET, Etiology, EEG, and A-C Response), plus gender, were treated as predictors of SPEs in a multiple regression analysis.⁹ The analysis was performed on all 100 cases. Only the independent contributions of gender, $t(94) = 2.98, p = .004$, and INSET, $t(94) = 4.74, p < .001$, were significant. The multiple r was .568. The standardized regression coefficients (β), in descending order, were: INSET (.425), gender (.280), etiology (.005), EEG (-.008), and A-C response (-.153).

The INSET clearly has the strongest relationship with SPEs among the four TLD components and it is significant independent of gender. It means that the positive TLD-SPE relationship is completely attributable to the INSET. Gender continues to be an independently significant predictor of SPEs.

Revised SPE Scores

The same analyses were performed using the new SPE score derived from scoring sub-items of ESP, as described above. The results are almost identical to those using the original SPE scores.

The mean SPE score was 4.70 ($SD = 4.79$). The TLD group had a mean SPE score of 5.62 ($SD = 5.08$) and the control group had a mean of 3.52 ($SD = 4.18$), $U = 994.5, p = .043$, one-tailed. The relationship was again destroyed when sex was introduced into the covariance analysis, $F(1, 84) = 0.77, p = .383$. Looking at the multiple regression analysis, once again only INSET, $t(94) = 4.14, p = .00007$, and gender, $t(94) = 3.00, p = .003$, were significant predictors of SPEs. The multiple r was

⁹ The regression analysis was performed on a Spearman correlation matrix.

.528. The β values, in descending order, were as follows: INSET (.383), gender (.291), etiology (.011), EEG (-.091), and A-C response (-.063).

Discussion

The most important finding to emerge from the data analyses completed so far is the significant positive relationship between SPE scores and scores on those INSET items scored for TLD. This outcome confirms the earlier results of Neppe (1983b), who found that members of the South African SPR who reported SPEs scored higher on an earlier version of the INSET than those not reporting SPEs. The only difference is that in Neppe's (1983b) study, SPEs were treated as the independent variable and in the present study the INSET TLD scores were the independent variable. Thus, findings from a non-clinical population have been confirmed using a clinical population, namely, that there is a correlation between temporal lobe symptomatology and subjective paranormal experience.

The finding with INSET supersedes and to an extent redefines the relationship between TLD and SPEs that constituted the main hypothesis. It is noteworthy in this connection that INSET scores did not correlate significantly with any of the other contributors to the TLD classification. This pattern of results suggests that INSET is measuring something not reflected in patients' EEGs, for example. It is also possible that the relationship between INSET scores and SPEs reflects a personality trait associated with an increased tendency to identify or report SPEs rather than to actually have them.

Gender revealed itself to be a confounding factor in the TLD-SPE relationship. It could be that females have attributes that make them more susceptible than males to seek out treatment for TLD related symptoms. In any event, our findings seem to indicate that VN more frequently diagnoses his female patients as having TLD than his male patients. We plan to examine the research literature to see if others have reported TLD as more prominent among females than males. It is noteworthy that the gender difference applies to the EEG and etiology codings, not just the INSET (see bottom of Table 2). Thus, more is at play here than a possible female bias to report more TLD symptoms or more frequent TLD symptoms than males.

Even if these findings reflect a genuine non-artifactual difference of temporal lobe symptoms in a group known to have TLD correlating with a higher incidence of SPEs, the possibility still exists that the findings reflect a personality predisposition, or an attitudinal or behavioral response pattern in this population. This would imply that some people are more likely to take note of experiences in their lives which they then interpret as anomalous and that these same people may report experiential symptoms of temporal lobe anomalies. However, this explanation is unlikely to explain the results fully, as the specific cluster of symptoms that the patients had are very uncommon in the general population (Neppe, 1981a, c; Neppe and Tucker, 1992). Moreover, the population of patients had diagnosed temporal lobe disease by a recognized expert in the area (VN) and were being successfully medicated for this. Nevertheless, the possibility exists that certain TLD patients may be more predisposed to endorsing symptoms, and this may explain why they may have endorsed more SPEs as well as temporal lobe symptoms on the INSET. On the other hand, these patients separately had detailed personality testing on the Minnesota Multiphasic Personality Inventory and other neuropsychological tests,

as well as detailed clinical assessments and neuropsychiatric evaluations over many sessions. These have not been analyzed as part of the research, but it is VN's strong clinical impression that there was no demonstrable personality type or attitudinal predisposition. Even if either of these explanations exist, this does not rule out clinically diagnosable TLD.

There are also limitations to the current statistical analysis, all of which may have weakened the obtained results:

1) In addition to the Short INSET being used here, VN interviewed the patients in significant detail about their symptoms, meaning that the TLD side of symptom analysis was more detailed than the short INSET itself. However, the same cannot be said about the SPE analysis. JP plans to interview a cohort of these patients to ensure greater detail as to frequency and kind of SPEs.

2) In Neppe's (1983b) original work, extensive and detailed information was obtained about subjects' SPEs, and this was used to select an initial experimental group of "subjective paranormal experiencers" that reflected an extreme level of ESP experiences. This extreme group was compared to a control group with no SPEs. We plan comparable exploratory analyses of the present data, comparing patients reporting no SPEs versus those reporting large numbers, based on our revised SPE scores. We will then look for specific diagnostic factors that distinguish the two groups.

3) Based on clinical impression, VN feels many of the "SPEs" found in the control group may not fit a more stringent definition of SPE. This is another reason for a more detailed evaluation of these SPEs.

4) A late methodological decision was made to allocate a score of 2 for a handful of subjects in which frequency of SPEs was not mentioned in the file. Although this was the proper decision from a research methodology point of view, based on VN's clinical experience a score of 1 may have been more appropriate for these patients. We plan an exploratory re-analysis with this in mind. We also plan to redefine the SPE scores once again to maximize differences in frequencies of SPEs.

5) The EEG scores were based on surface recordings that did not necessarily register possible deep-seated pathological temporolimbic activity. This may explain why some patients with high scores in the other TLD diagnostic criteria revealed normal EEGs, thus producing a low correlation between EEG and these other criteria and adversely affecting the predictability of the SPE scores. Moreover, in the original Neppe (1980b, 1983b) work on the SASPR population, despite the subjects having numerous SPEs correlating with possible temporal lobe symptoms, the EEGs were normal. (Nelson and Neppe, 1980).

6) Responsiveness to medication can be an excellent state-related index of the condition underlying pharmacological toleration and responsiveness (Neppe, 1990c), as well as tracing underlying biochemical and electrical abnormality including ostensible SPEs and geomagnetic variations (Neppe, 1999). The correlation between SPE frequency and anticonvulsant responsiveness may be better measured by tracking the differences in frequency of SPEs after administration of anticonvulsants, just as the possible temporal lobe symptoms should also be so tracked. Our present study instead used a generic score

for anticonvulsant response. We plan to follow up to obtain more longitudinal correlation data.

7) In contrast with the original Neppe (1980b, 1983b) SASPR study, this study did not distinguish state and trait temporal lobe phenomena: The study looked at symptoms broadly occurring over time, without directly correlating state related events.

PART II. EXPLORATORY ANALYSES

LOGISTIC REGRESSION ANALYSES

The purpose of the logistic regression analyses was to determine a set of discrete predictors, derived from the global predictors discussed previously, which would distinguish those patients who had a significant number of subjective ESP (S-ESP) experiences from those who did not. S-ESP experiences were chosen as the criterion variable rather than SPEs in general because only S-ESP experiences, if “real”, necessarily involve a paranormal process. This is not the case for apparitions and out-of-body experiences, which could simply be hallucinations (unless they involve a psi component, in which case they would most likely also count as S-ESP experiences). It should be noted, however, that the various SPE categories were highly intercorrelated.

Recall that S-ESP experiences were coded on a 3-point scale, with 3 = frequent, 2 = occasional, 1 = rare, and 0 = never. By dropping the “1” category, we were able to obtain two groups of approximately equal size. The “ESP” group (2+3) had $N = 53$ and the “No ESP” group (0) had $N = 40$. Thus, 93 of the 100 patients were included in the logistic regression analyses.

As was the case with the original SPE variable, females had many more S-ESP experiences than males, $\chi^2(1, N = 93) = 16.73, p < .001$.

The initial regression analyses used the “logit model” in SYSTAT 6.0 (Wilkinson, Blank, & Gruber, 1996). However, it was found that this software could not handle models that contained more than two of our predictors, or interactions. Thus, the more complex analyses were performed using a maximum likelihood estimation procedure from SAS 8.0. The SAS software gave virtually identical results to the SYSTAT software in test comparisons involving two or fewer predictors.

We decided to treat separately at the outset each of 3 broad categories of predictors used for the global analyses -- INSET, EEG, and etiological factors -- plus a new one, brain hemisphere dominance, attempting to find a set of significant predictors for each. These predictors were subsequently combined to yield the final model. Response to A-C drugs was not included because this variable could not be effectively broken down into more discrete categories and did not predict SPEs in the global analyses.

Because gender played such a prominent role in the global analyses, we decided to include it in the regression analyses from the outset. The first analyses were performed with gender, one of the discrete variables of primary interest, and the interaction between them as predictors. These analyses were performed solely to check for the possibility of

interactions. If the interaction term was not significant, the analysis was repeated with the interaction term removed. These later analyses were used to determine if the discrete predictor was significant, controlling for gender.

A p -value of .10 (two-tailed) was used as the criterion for inclusion in the models. Because the N s were identical or nearly identical in most of the models being compared, we generally considered the p -value to be a more meaningful criterion than an effect size measure.

INSET

The 16 INSET items were treated individually for the regression analyses. Five of them were statistically significant controlling for gender. They are listed at the top of Table 3.

Table 3
Significant ($p < .10$) Predictors of S-ESP Experiences,
Controlling for Gender

Item	Description	p -value
INSET:		
48	Nightmares	.003
15	Auditory Hallucinations	.025
13	Visual Hallucinations	.031
19	Jamais Vu	.037
7	Memory Disturbances	.091
EEG:		
LC	Left-Central	.071
Hemisphere Dominance:		
	Laterality	.008
	Handedness	.011

The next step was to include the 5 INSET variables from Table 3 along with gender in a more complex analysis. Although none of the INSET predictors were significant in this model, 4 of them -- nightmares, auditory hallucinations, visual hallucinations, and jamais vu -- clearly outperformed the others. It was noted that 2 of these (visual hallucinations and auditory hallucinations) had comparable p -values (.191 and .161), were conceptually related, and were moderately correlated with each other, $r(97) = .348$. Therefore, it was decided to create a new variable by adding the scores on visual and auditory hallucinations and put this new variable in the model. Further analyses were then performed with the remaining 4 variables plus gender, eliminating the weakest performer each time until all the remaining variables were significant. After nightmares dropped out, we had a final model with 3 significant predictors: Gender, χ^2

(1, $N = 92$) = 11.33, $p = .0008$; visual/auditory hallucinations, $\chi^2(1, N = 91) = 6.52, p = .011$, and jamais vu, $\chi^2(1, N = 91) = 4.48, p = .034$. Both INSET effects were positive, meaning that a high score on the item characterized the ESP group.

EEG

The patient files included details about the nature of EEG abnormalities and their locations as indicated by the surface electrodes. These detailed data were not available for 1 patient, a male. Locations could be specified by hemisphere (right, left, bilateral, or general) and lobe (temporal, frontal, central, parietal, or occipital). Two specific types of activity were also earmarked: spiking and slowing (unusual delta or theta). The remaining activity consisted of such wave patterns as bursts of fast beta or alpha. Because of the nature of Neppe's patient population, anomalies were more frequent in the temporal lobes than in other areas, and more patients had left temporal anomalies than right temporal ones (45 vs 18). Many possible variables could not be included in the analyses because their frequencies were too small. The variables with frequencies of 5 or greater are listed in Table 4.

Table 4
Analyzed EEG Codes

		Right	Left	Bilateral	Total
Temporal	Spike	RTX	LTX	BTX	TX
	Slow	-	-	-	TS
	All	RT	LT	BT	TEM
Frontal	Spike	-	-	-	-
	Slow	-	-	-	FS
	All	RF	LF	BF	FRO
Central	Spike	-	-	-	-
	Slow	-	-	-	-
	All	-	LC	BC	CEN
Parietal	Spike	-	-	-	-
	Slow	-	-	-	-
	All	-	-	-	-
Occipital	Spike	-	-	-	-
	Slow	-	-	-	-
	All	-	-	-	-
General	Spike	-	-	-	X
	Slow	-	-	-	SLO
	All	RG	LG	BG	GEN
Total	Spike	-	-	-	-
	Slow	-	-	-	-
	All	RGT	LFT	-	-

Only one of the EEG variables was a significant predictor of S-ESP with gender controlled. It was left central (LC), $t(91) = -1.81, p = .074$. Note that the relationship is negative; LC is associated with an absence of S-ESP experiences.

Etiology

Recall that the etiology variable consisted of certain kinds of recreational drug use plus head traumas of various sorts (e.g., concussion, tumor, encephalitis). For the regression analyses separate codes were created for the following recreational drugs that had more than 5 patients using them to a significant extent: marijuana, hallucinogens (LSD, psilocybin, mescaline), amphetamines, and cocaine. Significant use of any of the above was coded as a separate variable labeled “drugs”. Because the overwhelming majority of head traumas were concussions, these traumas were combined in a single category labeled “head”. None of these variables predicted S-ESP experiences with gender controlled. Thus, etiology made no contribution to the final model.

Brain Hemisphere Dominance

Because of its possible relevance to the EEG, we decided to include brain hemisphere dominance as a category for the regression analyses. The patients’ files had two indirect measures of this variable: “handedness” and “laterality”. For handedness, patients were simply asked on the INSET screen if they were left- or right-handed. To measure laterality, patients were asked the following three questions: (1) “Which hand do you write with?” (2) “Which side do you bat or throw with?” and (3) “Which side do you kick with?” These questions were intended to establish at a basic level whether or not the patients exhibited mixed functions for controlling basic dominant characteristics, reflecting possible higher brain functions that may be purely on one side or cross into both hemispheres. Laterality and handedness were amplified during the neurological examination by observing which hand was used in certain tests (writing, cerebellar diadokokinesia- a finger nose test) as well as by asking clinically relevant questions.

Both handedness and laterality were recorded as “left”, “right” and “both” (also called “either”). The “both” option was assigned for laterality when the patient gave inconsistent responses to questions or indicated that they used either hand for some or all of the tasks.

Because over 75% of the patients in the regression sample were right handed, and the same percentage right lateral, it was decided for purposes of the regression analyses to combine the “left” and “both” categories. Handedness and laterality were highly correlated in the total sample, $r(97) = .859$ and the regression sample, $r(91) = .866$.

Both variables significantly predicted S-ESP experiences controlling for gender: handedness, $z = 2.58, p = .010$; laterality, $z = 2.78, p = .005$. Because of the high correlation between the two predictors, we decided to select only laterality for the final model. The direction of the effect indicated that right-laterality (left-hemisphere dominance) was associated with the presence of S-ESP experiences.

The Final Model

Five variables have been selected as candidates for the final model: gender, visual/auditory hallucinations, jamais vu, left-central EEG, and laterality.

An initial regression analysis was performed including all 5 variables. The weakest variable was left-central EEG ($p = .224$), and this variable was removed for the next analysis. In this new analysis, all 4 predictors met the inclusion criterion of $p < .10$, and so it was accepted as the final model. Details of this analysis are presented in Table 5. Note that gender is by far the strongest predictor, with the other variables making roughly equal contributions.

Table 5
Analysis of Maximum Likelihood Estimates for Final Model

Variable	DF	Parameter Estimate	Standard Error	Wald χ^2	Prob. of χ^2	Odds Ratio	Confidence Limits (95%)
Intercept	1	-3.71	0.92	16.40	<.0001		
Gender	1	2.12	0.60	12.52	.0004	8.32	2.57/26.93
V/A Hallu.	1	0.33	0.13	6.49	.011	1.39	1.08/ 1.79
Laterality	1	1.70	0.69	6.13	.013	5.49	1.43/21.15
Jamais Vu	1	0.78	0.35	5.07	.024	2.19	1.11/ 4.33

Interactions with Gender

It was mentioned earlier that we began by analyzing all variables for possible interactions with gender. All those discussed so far, including the ones in Table 5, did not interact significantly with gender. Two others, both EEG variables, did.

Temporal Lobe. The first of these variables is labeled TEM, $\chi^2(1, N = 91) = 6.50$, $p = .011$. Patients were coded positive on this variable if their EEGs indicated abnormal activity of any kind in either the right temporal, left temporal, or both. Patients with generalized abnormal activity were not coded positively for TEM, although the generalized activity may have included the temporal lobes. A further examination of this interaction revealed that for females, temporal-lobe abnormalities were significantly associated with the presence of S-ESP experiences, Yates-corrected $\chi^2(1, N = 65) = 3.88$, $p = .049$, whereas for males temporal lobe abnormalities were associated with an absence of S-ESP experiences, $p = .091$ by Fisher's exact test. These relationships are illustrated in Table 6.

The previously reported finding that females had higher average code scores for temporal EEG disorder than did males is confirmed with TEM as the criterion variable, $\chi^2(1, N = 99) = 4.57$, $p = .033$. 61.8% of females had temporal lobe anomalies, compared to 38.7% of males. The relationship is illustrated in Table 7.

Hemisphere Differences. The significant laterality effect prompted us to create a new EEG variable that reflected hemispheric localization of anomalous activity. The variable, labeled LRD, had three levels. Those with left hemisphere activity only were coded 2, those with no activity or activity in both hemispheres were coded 1, and those with activity in the right hemisphere only were coded 0.

Table 6
S-ESP Experiences as a Function of Temporal Lobe
Abnormalities, Separately by Gender

Females:				Males:					
		S-ESP				S-ESP			
		Yes	No	Total			Yes	No	Total
Temp. EEG Abn.	Yes	33	8	41	Temp. EEG Abn.	Yes	1	11	12
	No	13	11	24		No	6	9	15
	Total	46	19	65		Total	7	20	27

Table 7
Temporal Lobe EEG Abnormalities
as a Function of Gender

		Gender		Total
		Female	Male	
Temp. EEG Abn.	Yes	42	12	54
	No	26	19	45
	Total	68	31	

LRD proved to be the second variable to interact significantly with gender, $\chi^2(1, N=92) = 4.73, p = .030$. This arrangement is illustrated in Table 8.

Moreover, although gender did not interact significantly with laterality as a predictor of S-ESP, the relationship between laterality and S-ESP was significant for females, $p = .014$, but entirely absent for males, $p = 1.00$, both by the Fisher exact test. However, the N s for left/mixed are once again quite low, especially for males.

Table 8
S-ESP Experiences as a Function of Hemisphere Differences
in Abnormalities, Separately by Gender

Females:				Males:				
	S-ESP		Total		S-ESP		Total	
	Yes	No			Yes	No		
Hem.	Right	3	2	5	Right	0	1	1
	Equal	24	13	37	Equal	5	15	20
	Left	19	4	13	Left	2	4	6
	Total	46	19	65	Total	7	20	27

Refinements of TEM

Temporal-lobe abnormalities had been further classified in terms of type (spike, slowing, and other) and location (left hemisphere, bilateral, right hemisphere), as illustrated in Table 4 above. A set of univariate regression analyses were performed to explore whether these more refined variables made a difference for females. Regarding type, the positive relationships between the abnormality and S-ESP were stronger for spikes, $z = 0.91$, and other, $z = 1.81$, than for slowing, $z = 0.15$. For spikes and other combined, the relationship was significant, $z = 2.31$, $p = .021$. Removing patients from the TEM group whose anomalies were restricted to slowing increases the relationship in Table 6 slightly, corrected $\chi^2(1, N=65) = 4.71$, $p = .030$. As for location, the regressions were positive for left-temporal, $z = 1.37$, and bilateral, $z = 0.97$, but negative for right temporal, $z = -0.35$. For left temporal and bilateral combined, the relationship was significant, $z = 2.31$, $p = .021$. Restricting the TEM group to those with only left temporal and/or bitemporal anomalies slightly strengthened the Table 6 relationship further for females, corrected $\chi^2(1, N=65) = 5.65$, $p = .017$.

A third refinement was called for by virtue of the significant negative relationship between S-ESP and anomalous firing in the left central lobe. To take this apparent suppressor into account, patients who survived the preceding cuts were removed from the temporal group if the anomalies extended to the central lobes. This final pruning of the TEM group increased the effect further still, $\chi^2(1, N=65) = 9.80$, $p = .002$.

The TEM group has now been redefined as consisting of patients with abnormal EEG activity other than slowing either in the left temporal lobe, sometimes extended *bilaterally* to the right temporal lobe, or to the frontal lobes¹⁰, only. Compared to the initial results depicted in Table 6, the size of the TEM group for females has been reduced from 41 to 28. The new group is labeled TEMR, for "TEM revised".

¹⁰ As the central lobes are eliminated and there were in fact no extensions to the parietal or occipital lobes in the TEMR group, for which there were very few anomalies recorded, the frontal lobes are the only remaining alternative.

Comparable analyses could not be performed for males because there was only one male positive for TEM who was also positive for S-ESP. The only EEG abnormalities in this patient were spiking in the right temporal lobe. However, the TEM group for males could be redefined as it was for females. When this is done, the reversal of the effect for females remains at about the same level of significance as previously, $p = .068$ by Fisher's exact test. The total number of males in the TEM group was reduced by 4. The TEMR classifications for both sexes are illustrated in Table 9.

Table 9
S-ESP Experiences as a Function of Temporal Lobe
Abnormalities (TEMR), Separately by Gender

Females:				Males:					
		S-ESP				S-ESP			
		Yes	No	Total			Yes	No	Total
Tem.	Yes	26	2	28	Tem.	Yes	0	8	8
EEG					EEG				
Abn.	No	20	17	37	Abn.	No	7	12	19
Total		46	19	65	Total		7	20	27

A Regression Model for Females

It was decided to restrict the model to the variables that were significant for the total sample (see Table 5) and for variables that interacted significantly with gender and also significant for females separately. This meant that the variables that entered the model initially were: jamais vu, visual/auditory hallucinations, laterality, and TEMR.

All variables met the $p < .10$ criterion except jamais vu ($p = .294$). The remaining variables then defined the final model for females, which is illustrated in Table 10.

Table 10
Analysis of Maximum Likelihood Estimates for Final Model,
Females Only

Variable	DF	Parameter Estimate	Standard Error	Wald χ^2	Prob. of χ^2	Odds Ratio	Confidence Limits (95%)
Intercept	1	-2.08	0.86	5.90	.015		
V/A Hallu.	1	0.49	0.19	6.85	.009	1.63	1.13 / 2.35
TEMR	1	2.66	0.91	8.53	.004	14.22	2.39/84.50
Laterality	1	1.31	0.79	2.78	.095	3.73	0.80/17.46

The weakness of the contribution by laterality likely results from the fact that for some reason right-lateralized patients were more likely to be in the TEMR group than left/mixed lateralized patients, corrected $\chi^2(1, N=65) = 3.77, p = .052$. Correlated predictors reduce individual contributions to regression equations.

Discussion

The regression analyses succeeded in their objective of highlighting discrete variables that are significantly associated with S-ESP experiences. However, it should be kept in mind that this outcome resulted from a great deal of “data-snooping” and that some of the significant relationships are likely to be type-one errors. A related problem is that the number of patients in some of the cells is quite low, due to a combination of low *Ns* overall and extreme splits on some variables. One implication of this is that the odds ratios for regression variables often have wide confidence intervals. None of the results from the regression analyses can be considered conclusive until they are cross-validated in an independent sample.

The only predictor variables we examined but did not discuss above were age and two sets of four INSET subscales derived from a factor analysis of INSET. Age did not relate to S-ESP at all. Among the INSET subscales, the only independent significant predictors of S-ESP were ones that reflected “sensory” items and were essentially equivalent to the visual/auditory hallucination variable. We felt that the individual item correlations were more informative and that is why they were chosen to report.

Among the individual predictors of S-ESP, gender was clearly the strongest. This, of course, was also evident from the primary analysis, where it was a strong predictor of SPEs. This finding should not be surprising to parapsychologists. Schouten (1979, 1981a, 1981b) found that three major collections of spontaneous cases each included more females than males as percipients. This pattern did not show up as clearly in Palmer’s (1979) Virginia survey using random sampling techniques, although females were more likely to report waking ESP experiences than males to a suggestive degree ($p = .052$).

It is possible that the gender differences regarding S-ESP experiences in the present study, as well as in Schouten’s, could be reporting artifacts. In other words, women might simply be more prone to report S-ESP experiences than men. Schouten discounted this reporting artifact in his collections from Britain (Schouten, 1979) and Germany (Schouten, 1981a), because he found that females were not more likely than males to report cases in which they were not involved as percipient or target person. On the other hand, females did predominate among these outside reporters in the American collection (obtained by Louisa Rhine), so the reporting artifact was considered to be a viable interpretation for this sample (Schouten, 1981b). Palmer’s (1979) survey differed methodologically from the Rhine collection in that Palmer’s solicitations were targeted to specific individuals randomly selected from the population of an American city. Rhine’s cases came from responses to published appeals and from persons who had heard of the Duke University Parapsychology Laboratory and wanted to share their experiences. As more initiative was required from those who responded to Rhine’s survey than to Palmer’s, the latter is less likely to have been influenced by reporting artifacts, and the

preponderance of female percipients was indeed much less pronounced in Palmer's survey. The present study is more similar methodologically to Palmer's survey than to Rhine's, as VN solicited his accounts of S-ESP experiences individually from his "captive audience" of patients. On the other hand, the results from the present sample are more similar to those obtained by Rhine. One way to reconcile these contradictory findings, which represents a reporting artifact interpretation, is to suggest that VN's male patients were more reluctant to mention S-ESP experiences to VN face-to-face than were the respondents to Palmer's mail questionnaire, who also were promised anonymity. Alternatively, the male patients as a group may suppress their S-ESP experiences per se more than females, even when they have the same temporal lobe condition. At any rate, the reporting artifact interpretation needs to be taken seriously in the present study, although it cannot be considered confirmed. Finally, it should be noted that reporting artifacts cannot account for the gender differences in EEG variables found in the present study, which in turn were shown to relate to S-ESP.

Total INSET scores were found to be a strong predictor of SPEs in the main analysis. In the regression analyses, two items (or item clusters) were found to independently predict S-ESP: visual and auditory hallucinations, and jamais vu.

It should be noted that only certain kinds of visual and auditory hallucinations are considered by VN to possibly be associated with TLD. For visual, these are movements and distortions in shape or size; for auditory, they are buzzing, ringing, hissing, and tapping sounds. The auditory and visual hallucination items were combined to form a single item, which admittedly gave them a built-in advantage in entering the final regression model. However, the combination made conceptual sense and the items in isolation were among the four strongest independent predictors of S-ESP, controlling for gender. Visual/auditory hallucinations also makes sense as a predictor of S-ESP for the simple reason that most S-ESP experiences are themselves visual or auditory hallucinations, albeit ostensibly veridical ones.

Although the hallucinations coded for TLD are much more primitive than the content of most S-ESP experiences, the relationship between visual/auditory hallucinations and S-ESP suggests that there are important commonalities in how the two types of experiences are processed in the brain. This relationship also reminds us that ESP per se and the hallucinatory experiences that often carry it are intertwined and cannot be easily teased apart. Thus, when we find correlates of S-ESP we might be finding correlates of hallucinatory activity rather than the ESP process. Resolving the ambiguity will require comparing the correlates of S-ESP experiences with those of other hallucinatory experiences that we can safely assume lack an ESP component.

The item reflecting jamais vu on INSET had the following wording: "How often have you been in a familiar place and had the impression that you have never been in that place before? (the opposite of déjà vu called jamais vu - not recognized at all, totally unfamiliar)." Although VN has found that patients at times interpret jamais vu incorrectly, including the mis-classification of derealization experiences and odd déjà vu experiences as jamais vu experiences, the patients in this research were routinely screened about their positive INSET responses, including jamais vu, so that this error would have been picked up. Nevertheless, the descriptions obtained clinically were

sometimes questionable in nature and difficult to compartmentalize into a *jamais vu* category.

Neppe, who developed the INSET, considers *jamais vu* to be the best single INSET item for the purpose of screening TLD. This conclusion was borne out by his extensive research on *déjà vu*, in which the wording of the *jamais vu* item was identical to that used in the current study (Neppe, 1983a). However, very little research has been conducted on *jamais vu* per se, and more needs to be done. Finally, recall that certain kinds of *déjà vu* experiences, as well as certain types of olfactory hallucinations, have in the past been found by Neppe (1981a, 1983a,c,d) to be closely associated with SPEs but were not studied in this research for reasons outlined in the Method section of Part I.

The strongest INSET predictor, controlling for gender, was actually the nightmare item. It did not enter the model because of its relatively high correlations with the other INSET items in the mix, particularly *jamais vu*, $r(97) = .440$. It was not combined with *jamais vu* to form a single item, as was done with visual and auditory hallucinations, because nightmares and *jamais vu* do not bear an obvious conceptual relationship to each other.

The most surprising correlate of S-ESP experiences to the authors was laterality, which was intended as a measure of hemispheric dominance. However, our operationalization of laterality was incomplete, as it did not measure other of its facets (for example, right or left eye dominance, right or left ear lateralization, right or left foot used to pick up a thumb tack.) Additionally, it did not take into account the major marker of hemispheric dominance, namely speech. Speech dominance is not easily measured except by techniques such as the Wada test (Wada & Rasmussen, 1960) of injecting sodium amytal into the carotid arteries, but even this test has its limitations in interpretation. Laterality measures without speech do not assure completely accurate assessment of which hemisphere is dominant. Nonetheless, pure right laterality as we defined it for the present study almost certainly implies left hemisphere dominance (99% or above), and mixed laterality and left laterality imply likely right hemisphere dominance (80% or above). Still, these are clinical estimates.

The rationale that underlies our research received support from the EEG analyses in the sense that the one area of the brain that seemed to be associated with S-ESP was the temporal lobes (TEM). This singularity may be partly due to the fact that there were much fewer cases of anomalies in other parts of the brain than in the temporal lobes, and there were too few examples of parietal and occipital abnormalities to even analyze.

The effect of EEG abnormalities in the temporal lobes was also found to depend on gender. For females, the relationship was positive, as we predicted at the outset. However, for males it was negative, albeit at a marginal level of significance ($p = .091$). We have no explanation for this reversal for males. The reversal might have been less pronounced, and perhaps nonsignificant, were we able to include data from one male patient with strong S-ESP experiences. Although enough information was available on this patient to classify him for the original analyses as having EEG abnormalities indicative of TLD, the available EEG report (from another clinic) was not precise enough to allow the more refined coding needed for the logistic regression analyses. Thus, this patient was coded as missing for EEG variables in these latter analyses.

If the overall sex difference in reported S-ESP experiences is due to under-reporting of these experiences by males, then the failure of the TEM hypothesis to hold for males can be brought into question. However, if the critical factor is indeed response bias, one would expect no relationship between TEM and S-ESP, not a reversal (unless one entertains the unparsimonious assumption that the response bias is particularly uncharacteristic of males with anomalous temporal EEG activity). However, it should again be emphasized that the reversal is weak statistically and the relevant male sample size small.

We attempted to further refine the nature of the temporal lobe abnormalities predictive of S-ESP by specifying the type of abnormality and its localization by hemisphere, creating a new variable, TEMR. The examination of which temporal lobe (right or left) was most closely associated with S-ESP seems particularly reasonable in light of the interaction between gender and left-side vs. right-side anomalies over the entire scalp. Females showed a greater left-focus than males in this analysis. The emergence of laterality as a key variable also might cause one to expect laterality of the EEG anomalies as well. The effect seems to be that for females the anomalies are most likely to affect S-ESP if they are focused in the dominant (left) hemisphere (or bilaterally, which, of course, includes the left hemisphere).

There has been some exploration of brain hemisphere laterality in the experimental ESP literature, but the results have been inconsistent. Broughton (1978) reported results from three studies that collectively suggested subjects scored best on a forced-choice type ESP task when they performed the test with the left hand (right hemisphere dominance) simultaneously with a left-hemisphere distraction task. The effect was demonstrated only for males. On the other hand, Maher and Schmeidler (1977) found significant scoring, also restricted to males, only when the forced-choice ESP task was taken with the right hand while the left hand was occupied with a pattern-tracing task designed to activate the right hemisphere. However, this finding could not be replicated (Maher, Peratsakis, & Schmeidler, 1979). Finally, Alexander and Broughton (2001) found that left-dominant subjects, as measured by the Cognitive Laterality Battery (Gordon, 1986), scored somewhat better in a free-response ESP ganzfeld experiment than did right dominant subjects, but the performance of the left-dominant subjects only approached significance ($z = 1.60$). No reports of gender effects were included.

Rationales notwithstanding, the results of the refinements of TEM have less statistical foundation than those discussed previously, as they appeal to non-significant trends in the data. Removal of cases where the abnormality consisted of EEG slowing left a slightly stronger relationship between temporal-lobe abnormalities and S-ESP for females, the improvement was not significant. Likewise, right-temporal anomalies contributed nothing to the temporal lobe / S-ESP relationship for females, but neither could these right-temporal anomalies be differentiated from the left-hemisphere contributions to a statistically significant degree. This state of affairs is attributable partly to the low number of cases of slowing and right-temporal loci compared to higher frequency anomalies (spikes, paroxysms, sharp waves, etc.) and left-temporal loci. The best that can be said is that effects were only demonstrated for higher frequency abnormalities that occur in the left temporal lobe.

We also excluded from TEMR cases where the anomalies extended to the central lobes, because of the significant negative relationship between left central EEG anomalies (controlling for gender) and S-ESP. This simplified the model further by restricting extension of the temporal lobe abnormalities to the frontal lobes. Moreover, the left-central finding could conceivably indicate that anomalies outside the temporal lobes might be S-ESP-inhibitory. Generalized anomalies observed over the whole scalp, controlling for gender, also related negatively to S-ESP experiences, although not significantly so, $z = -1.56, p = .119$.

Indirect empirical support for the TEMR model as defined above comes from a recent experiment by Alexander (2000), who found that a reputedly psychically gifted right-handed female showed excess fast EEG activity in the left temporal and frontal lobes when engaged in four marginally successful ($p = .056$) remote viewing trials as compared to matched control periods. The participant also scored high on the Complex Partial Epileptic Signs (CPES) scale (Persinger & Makarec, 1993).¹¹

An examination of Table 9 reveals that prediction of S-ESP was better for those who had temporal lobe EEG anomalies than for those who did not. This discrepancy could be explained by noting that even with the important advantage of ambulatory EEG we only had EEG data from patients for relatively brief periods of time. It is possible that if more EEG data could have been collected, some members of the non-TEMR group with S-ESP experiences might have revealed anomalies that would have placed them in the TEMR group. Additionally, as far as VN is aware, and certainly based on the written reports of patients' experiences during ambulatory EEG, no patient in this sample had any kind of SPE during this period. Consequently, these EEG measures are trait, not state variables. In Neppe's original research linking temporal lobe symptomatology with SPEs, he reported that there was both a state and a trait correlation of SPEs with temporal lobe symptomatology in an ostensibly normally functioning population (Neppe, 1979; 1983b).

Finally, we would like to stress a more general point. The finding that persons with TLD symptoms have more S-ESP experiences than those with the other neurological disorders represented in our sample in no way implies that S-ESP experiences are the product of a diseased brain. Clearly, many people who have S-ESP experiences are in good neurological health, as was borne out by Neppe's original sample of members of the South African Society for Psychical Research (Neppe, 1979; 1983b). What we sought to find out in this study was what parts of the brain might be involved in SPEs. Our guess is that activity in the temporal lobes may indeed be relevant to SPEs, but this activity need not reach the extremes evidenced by some of the patients in our sample. Persinger (1983), for example, has suggested that mystical experiences, including S-ESP, might be associated with micro-seizures in the deep structures of the temporal lobes. In most cases, these micro-seizures would not be considered in any way pathological. A useful adjunct to the present study would be to explore the proportion of "normal" participants who would be classified as S-ESP-positive using the same S-ESP questions employed in the present study, and, furthermore, to see if the INSET items reflecting TLD are as

¹¹ Although Palmer had heard Alexander's paper reported at a conference over a year ago, he had not remembered the specific results at the time he was conducting the regression analyses. His memory was refreshed by Alexander when he shared our results with her after the analyses had been completed.

predictive of S-ESP experiences in this “normal” population as they are in the patient population.

PHONE INTERVIEWS

S-ESP Experiences

Because the information about S-ESP experiences obtained from the INSET items was sketchy, it was decided that JP would attempt to interview by phone as many patients as possible who had reported S-ESP experiences on INSET. Neppe and Magill attempted to reach these patients. If they succeeded, the nature of the phone interview was described to them and informed consent obtained. JP was given only the first names of the patients. Because these were not the same as the patient initials he had been given for the purpose of coding clinical symptoms, he was effectively blind to how each patient had been scored on these variables, at least during discussion of the ESP experiences. The interviews were tape recorded, with the patients’ consent, and transcribed. None of the patients expressed any reservations about the taping.

Interviews were obtained from 20 of the 53 patients (37.7%) who were in the S-ESP-positive group for the regression analyses. Eighteen of them were female. The numbers interviewed were limited because Neppe felt that it was important to not in any way compromise his patients. Thus, he arbitrarily chose only those patients who were:

1. regularly following up with him, so that he could discuss the interview procedure with them face to face and he or Magill could ensure that the detailed new consent form used for this aspect of the research was properly completed. There were 23 patients who met this criterion.
2. not fragile, so that there was no risk of them being adversely affected by the interview. Two patients were eliminated by this criterion, reducing the total available to 21.

One of the remaining patients declined to be interviewed, bringing the total number of interviewees to 20.

Some of the interviews were conducted a considerable period of time after the original obtaining of the source data, in some instances several years. During this time, patients had relocated, were no longer directly under the care of the PNI, may have been potentially in conflict (e.g., unpaid bills potentially compromising further research relationships), or had not been seen in the clinic for a period of six months or greater. Although the percentage of interviewees was less than ideal, it is VN’s impression that they are representative of the larger sample in regard to S-ESP experiences. VN himself had additional insight into the nature of the S-ESP experiences of many of his patients, as a result of conversations during appointments at his clinic.

During the interviews, JP asked the patient to describe their most impressive ESP experience, in the sense of which they would choose if they were trying to convince a skeptical friend that ESP is real. If the chosen experience did not appear to be genuine, JP asked for another example.

Following the interviews, JP rated on a 3-point scale the quality of the best ESP experience described by the patient: (a) a “2” meant that the experience was credible, in the sense that, if it occurred as described, it was unlikely to be a mere coincidence and there was no prior information available from which the event could be logically inferred. An example of an experience in this category is one where the patient had an impression of a traffic accident which he witnessed 7-minutes later and included an accurate image of a child going through the car window; (b) a “1” meant the experience was marginally credible. An example here is a general statement that the patient often “knew” that a particular person was about to phone them and that person in fact promptly called; (c) a “0” meant that the experience was not credible, or the patient denied having any ESP experiences. An example here would be a general statement that the patient had insights into what she had done wrong in her marriage.

Of the 20 patients interviewed, 13 (65%) received a credibility of score of 2, 5 (22.7%) received a score of 1, and 4 (18.2%) received a score of 0. 2 of the 4 patients who scored 0 contacted VN privately to the effect that because they didn’t know JP they did not share with him their best experiences.

In addition, VN made his own ratings based on his notes and recall about conversations with his patients about their SPEs. His ratings reflected SPEs in general more than JP’s, who asked specifically about S-ESP experiences. His initial ratings were somewhat more positive than JP’s. He gave a rating of 2 to 16 patients (80%), a rating of 1 to 3 patients (15%), and for the remaining 1 he felt he had too little information to make a rating (5%). On 2 cases where his codings differed from JP’s, he modified his original codings after consultation with JP. This made his final ratings as follows: 2: 14 patients (70%); 1: 5 patients (20%); no rating: 1 patients (5%). The final ratings are illustrated in Table 11.

JP interviewed 2 additional patients who, unbeknownst to him at the time, did not report any S-ESP experiences on the INSET and thus were not among the 53 ESP-positive patients. He gave both these patients a rating of 1, and for 1 of these he had been tempted to make it 0.

Overall, JP was reassured about the general quality of the ESP experiences of the patients he interviewed. He was surprised at how articulate they were, and the great majority seemed to understand what was necessary if an ESP experience is to be considered credible. Recall that patients with serious psychiatric problems were removed from the sample at the outset. However, no claim is being made that any of the experiences can be considered evidence for ESP.

Anti-Convulsant Drugs

A second objective of the interviews was to get patients’ impressions of whether they noticed any effect of the A-C medications they had been taking on the frequency of their ESP experiences. Five patients had not taken A-C drugs and could not give meaningful responses to the question, and 1 additional patient did not comply with the request to take the prescribed A-C drugs. Of the remaining 14, 8 (57.1%) claimed to JP that at least one of the A-C drugs inhibited their ESP experiences, 2 (14.3%) claimed at least one had a facilitating effect, and 4 (28.6%) claimed no effect either way. There

were no reliable indications that any specific drug or drugs had a particular kind of effect or no effect.

Table 11
Final Ratings on Phone Interview Questions

QUALITY OF S-ESP			EFFECT OF DRUGS		
	JP	VN		JP	VN
Score	<i>N</i>	<i>N</i>	Effect	<i>N</i>	<i>N</i>
2	13	14	Neg.	8	12
1	4	5	None	4	2
0	3 ^a	0	Pos.	2	0
N/R ^b	0	1	N/R ^b	0	0
Total	20	20	Total	14	14

^aIncludes 2 patients who told VN they under-reported S-ESP experiences to JP

^bN/R means not rated.

VN's initial codings reflected the trend toward suppression of S-ESP experiences by drugs a bit more strongly than JP's. He concluded that A-C drugs had an inhibiting effect on 13 of the 14 relevant patients (92.9%), and no effect on 1 patient (7.1%). Following consultation with JP on the two cases in which JP coded for a positive effect of the drug on S-ESP experiences, he modified one of his ratings. Thus, the final tally for VN was: inhibiting effect, 12 of 14 (85.7%), no effect, 2 of 14 (14.3%).

It is clear from these data that if an A-C drug has a perceived effect on S-ESP experiences, the effect is most often to reduce their frequency. As it was found from the original analyses that at least some of these drugs almost universally helped alleviate patients' clinical symptoms, this inhibitory effect on S-ESP suggests that to some degree S-ESP experiences and the clinical symptoms have a common root.

PART III. CONCLUSIONS

This is the first study demonstrating that subjects with clinical temporal lobe dysfunction demonstrate a strong relationship between their clinical temporal lobe symptomatology and SPEs. Given the extremely detailed notes that were reviewed and the care in accurate scoring, it appears to be a real, non-artifactual statistical result. Because of the previously demonstrated link in normal subjects, it is a major finding.

Exploratory findings revealed that jamais vu experiences and visual/auditory hallucinations were the specific symptoms that most clearly predicted S-ESP experiences, a subcategory of SPEs. High-frequency EEG anomalies restricted to the temporal lobes and sometimes extending to the frontal lobes were positively related to S-ESP for females and negatively for males. S-ESP was most prevalent in patients who appeared to be right lateralized, indicating left hemisphere dominance. All these exploratory findings need to be confirmed in an independent sample, including a larger group of male patients, and also compared to results from a reference group not exhibiting neuropathology.

Phone interviews of 20 patients who reported S-ESP experiences on INSET indicated that the great majority had experiences that were credible and that the experiences tended to be inhibited by A-C drugs.

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APPENDIX

TLD Items on INSET

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- 1) How often do you have () fits, () seizures or () "peculiar spells"?
- 2) How often have you had a () blackout or () lost consciousness for a short period for no reason?
- 3) How often have you had () grand mal or () petit mal or () myoclonic or () psychomotor seizures?
- 4) How often do you have or are you told that you at times lose contact with () staring spells or () absences or () episodes where you have a blank look on your face () for seconds or () minutes not hours?
- 6) How often have you for a very short time like seconds or minutes been completely unaware that you did or been told that you did any of the following: () odd behaviors like () buttoning / unbuttoning; () chewing / mouth movements or () other unusual movements or () doing very strange things or () saying strange things or () finding yourself in places you don't remember going to or () jerking the arms ?
- 7) How often do you () have clear cut gaps in your memory during which you totally cannot remember anything for 5 minutes or more; () miss major sections of TV shows you have been watching; () find yourself driving without remembering how you got there or where you are going; () do strange things automatically? Include only if you think these are not only because of difficulty you have concentrating .
- 8) How often do your () moods, () feelings or () thoughts fluctuate within minutes for no reason [like moods which are one moment () very happy then very sad] ?
- 11) How often do you have odd sensations in part of your body like () floating, () turning or () moving when you were doing none of those?
- 12) How often have you come across a smell when there is nothing to cause it? If so, what kind (check applicable)? () medicine; () steak; () perfume; () flowers; () burning; () rotting; () synthetic; () vomit; () incense; () musty; () grass; () bitter; () sweet; () cake; () mustard; () other [ONLY "BURNING", "ROTTING" SCORED]
- 13) How often have you seen any of the following when there is no-one or nothing to cause it? () dots; () lights; () patterns; () shapes; () wrong size; () movements; () distortions; () things moving; () stars; () bugs; () threads; () insects; () none; () other [ONLY "MOVEMENTS", "DISTORTIONS", "WRONG SIZE" SCORED]

15) How often do you hear any of the following, when there is no-one or nothing to cause it? () buzz; () ring; () sizz; () hiss; () tap; () songs; () whistling; () music; () single word; () arguing; () names; () voices; () jumble; () message; () instructing; () radio / TV; () phone; () nothing; () other [ONLY "BUZZ", "RING", "HISS", "TAP" SCORED]

19) How often have you been in a familiar place and had the impression that you have never been in that place before? (the opposite of déjà vu called jamais vu - not recognized at all, totally unfamiliar)

23) How often have you found that, for no apparent reason, you are actually reliving things in the past (as if the past flows like a movie screen before you)?

28) How often do you have sudden, unexplained and uncontrollable attacks of intense fear?

34) How often do you hear what is being said, yet you cannot understand or make sense of it?

48) How often do you have frightening nightmares?

SPE Items on INSET

49) How often have you had () premonitions or () "psychic", () intuitive or () paranormal experiences such as () knowing the future , () sensing correctly unknown past knowledge , () having dreams which came true, or () strange feelings which came true?

50) How often have you felt you've () seen events that happened at a great distance before or while they were happening or () been in touch with someone when they were far away from you or dead?

51) How often have you felt you have () left your body or () had an out of body experience?

52) How often have you been aware of a presence of someone whom you could not see?
