

Anxiety and avoidance in psychogenic nonepileptic seizures: The role of implicit and explicit anxiety



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ABSTRACT

This study examined implicit and explicit anxiety in individuals with epilepsy and psychogenic nonepileptic seizures (PNEs) and explored whether these constructs were related to experiential avoidance and seizure frequency. Based on recent psychological models of PNEs, it was hypothesized that nonepileptic seizures would be associated with implicit and explicit anxiety and experiential avoidance. Explicit anxiety was measured by the State-Trait Anxiety Inventory; implicit anxiety was measured by an Implicit Relational Assessment Procedure; and experiential avoidance was measured with the Multidimensional Experiential Avoidance Questionnaire. Although both groups with epilepsy and PNEs scored similarly on implicit measures of anxiety, significant implicit–explicit anxiety discrepancies were only identified in patients with PNEs ($p < .001$). In the group with PNEs (but not in the group with epilepsy), explicit anxiety correlated with experiential avoidance ($r = .63, p < .01$) and frequency of seizures ($r = .67, p < .01$); implicit anxiety correlated with frequency of seizures only ($r = .56, p < .01$). Our findings demonstrate the role of implicit anxiety in PNEs and provide additional support for the contribution of explicit anxiety and experiential avoidance to this disorder.

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1. Introduction

1.1. Anxiety and avoidance in psychogenic nonepileptic seizures

Psychogenic nonepileptic seizures (PNEs) bear a superficial resemblance to epileptic seizures. However, whereas the experiences and behaviors associated with epileptic seizures are caused by abnormal electrical activity in the brain, most PNEs are considered to be a psychological dissociative reaction to threatening situations, sensations, emotions, thoughts, or memories [1,2]. Indeed, while psychodynamic, cognitive, behavioral, and systemic psychological theories offer different accounts of PNEs [3], all recognize the patient's response to anxiety as a significant contributing factor and suggest that PNEs may reflect an inability, failure, or unwillingness to actively engage with anxiety. This recognition is supported by evidence that patients with PNEs generally report a greater preference for avoidant coping strategies and are more likely to somatize their distress compared with those with epilepsy [4–10]. Nevertheless, relatively little research

has specifically addressed avoidance in PNEs despite its key role in many psychological theories about the etiology of PNEs.

Within the broader psychological literature, avoidance of anxiety or avoidance of other introspective experiences, termed 'experiential avoidance', is frequently associated with psychopathology [11–13]. Experiential avoidance is not merely the avoidance of certain situations but rather the avoidance of one's own thoughts, sensations, and emotions, particularly anxiety-provoking ones [14]. Such avoidance can be voluntary or involuntary, with the involuntary aspect arguably most likely to precipitate clinical syndromes such as PNEs [15].

Anxiety itself is a complex physiological and behavioral experience with both 'explicit' and 'implicit' cognitive components [16,17]. As detailed below, 'explicit cognition' refers to thoughts or experiences in one's subjective awareness, as typically captured via self-report measures; 'implicit cognition' refers to attitudes, beliefs, preferences, learning processes, emotional experiences, or other knowledge or cognitive processes (e.g., attitudes about oneself or others) that occur outside of conscious awareness and that are captured using indirect measures [18,19]. Implicit and explicit measures are typically unrelated or only modestly related [20]. Discrepancies between the two would arguably be more pronounced among patients with limited self-awareness and have, for instance, been demonstrated in patients with borderline personality disorder [21]. Given the types of personality pathology associated with PNEs, including abnormalities of the borderline type

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[22–24], it may be reasonable to expect similar discrepancies between implicit and explicit measures in patients with nonepileptic seizures.

Studies comparing anxiety in individuals with PNEs and epilepsy have failed to identify clear and consistent differences, although the prevalence rates of anxiety disorders have been found to be approximately twice as high in both groups as in the general population [25,26]. Some studies showed similar mean levels of self-reported anxiety in patients with epilepsy or PNEs [27,28], whereas others found significant [29] or trend-level differences [30]. Such inconsistencies may be explained, in part, by the use of explicit measures, which not only are susceptible to social desirability biases but also assume a level of insight and awareness and an ability to accurately report on internal states – skills that may be diminished in individuals with neurological disorders or individuals who tend to avoid interoceptive experiences. Self-report measures such as the MMPI, which attempt to circumvent these problems, have been more likely to find group differences [29,31], although findings have not been consistently replicated and have been questioned in terms of sensitivity and specificity for the differential diagnosis of epilepsy and PNEs [32] (also discussed in [33]). What is more, while the MMPI has been used extensively, it does not separate clearly between psychopathology and normal findings, does not specifically describe different types of avoidance behaviors, and cannot measure implicit cognition.

1.2. Implicit cognition and measurement

'Implicit cognition' is a term widely used by psychologists to refer to hypothetical psychological attributes (e.g., beliefs about self or others, as noted earlier) that are outside of conscious awareness and, therefore, introspectively inaccessible [34]. Importantly, these cognitions can have a strong impact on physiological responses [35] and behavior [36]. Measures of implicit cognition aim to provide an index of an attitude or cognition without requiring a participant's awareness or conscious access to the attribute under investigation [37,38]. This is achieved through tasks where participants respond in an "automatic manner" (p. 347 [39]), with little or no opportunity for attentional controllability or self-monitoring [19,40,41].

Implicit measures often employ a response latency (reaction time) paradigm, underpinned by an assumption that implicit cognitive biases can be detected by examining efficiency of cognitive processing [19,40]. This can be done through the aggregation of many overt responses (e.g., key presses on computerized tasks), frequently under time pressure, and across various types of stimuli (e.g., words or pictures related to a targeted attribute) [42,43]. Studies using implicit measures have offered evidence for their convergent and discriminant validity in different scenarios and groups [44,45], with research to date finding that implicit indices appear to be better than self-report or clinical judgement at predicting important clinical behaviors such as suicide attempts [46], substance misuse [47], and sexual offending [48].

Very few previous studies have used measures of implicit cognition in patients with PNEs. One prior study compared covert attitudes toward sickness in patients with PNEs, patients with epilepsy, and controls using an Implicit Association Test that examined responses to pairings of sickness-related words and pleasant words [49]; however, there were no significant group differences in implicit attitudes toward sickness despite differences in reports of clinical symptoms (e.g., greater somatic complaints in those with PNEs versus those with epilepsy). Other studies found that individuals with PNEs do have implicit biases compared with healthy controls in that they show greater emotional arousal to neutral stimuli [50] and direct greater preconscious attention toward threat cues (angry faces; [51]). Therefore, it is possible that individuals with PNEs have a greater underlying – or implicit – sense of anxiety.

One contemporary measure of implicit cognition is the Implicit Relational Assessment Procedure (IRAP; [43]). The IRAP involves presenting (frequent word) stimuli with specific 'relational terms' (e.g., true, false, same, and opposite) so that the relationships between the presented

stimuli (termed *verbal relations*) can be assessed. For example, participants may be shown a statement such as 'I am – anxious' or 'Others are – anxious' and asked to confirm or deny this relationship (in this example by choosing the term 'true' or 'false'). Importantly, participants are asked to respond quickly and accurately to these statements in ways that, depending on the trial type, are consistent or inconsistent with their beliefs. In the present study, for example, participants were asked to deny being anxious during consistent trials (e.g., selecting 'false' to the stimuli 'I am – anxious') and to endorse the opposite during inconsistent trials (e.g., selecting 'true' to the stimuli 'I am – anxious'). The methodology is predicated on the assumption that the strength of specific implicit verbal relations is reflected in the participant's response times; more simply, the basic IRAP principle is that average response latencies are relatively shorter across trials consistent with the participant's "true" (implicit) beliefs (e.g., those statements that cohere with the participant's implicit verbal relations) compared with trials inconsistent with their beliefs.

A wealth of studies have demonstrated the IRAP effect, providing support for its utility and reliability as an implicit measure (see [52] for an overview). Furthermore, research has indicated that the IRAP compares favorably with other implicit measures of individual differences [53], is perhaps less susceptible to 'faking' or overt manipulation [54], and can target clinically relevant phenomena [48,55].

1.3. Aims and hypotheses

The research outlined above suggests that anxiety and experiential avoidance may play a key part in PNEs. Specifically, this study aimed to (1) compare individuals with PNEs, individuals with epilepsy, and nonclinical controls on implicit and explicit measures of anxiety; (2) examine discrepancies between implicit and explicit anxiety within these groups; (3) examine correlations between anxiety and avoidance in PNEs; and (4) establish whether these measures of anxiety or avoidance have predictive utility in differentiating diagnostic groups. It was hypothesized that patients with PNEs would report higher levels of (explicit) anxiety and experiential avoidance compared with those with epilepsy or controls. However, previous studies have also highlighted that patients with PNEs are more likely than those with epilepsy to deny the relevance of psychological factors for their seizures [56], and, therefore, we predicted that those with PNEs would show greater implicit anxiety and show greater discrepancies between implicit and explicit anxiety (i.e., greater implicit relative to explicit anxiety) compared with those with epilepsy or controls.

2. Method

2.1. Participants

Thirty adults with PNEs and 25 adults with epilepsy (13 with focal epilepsy, 5 with idiopathic generalized epilepsy, and 7 with unclassifiable epilepsy) were recruited from outpatient seizure clinics at the Sheffield Teaching Hospital NHS Foundation Trust between February and September 2012. All diagnoses were made by neurologists specializing in the treatment of seizures, and only those whose diagnoses were supported by a previous video-EEG recording of a typical seizure were included. Patients with mixed seizure disorders (epilepsy and PNEs) were excluded. Thirty-one adults with no reported history of seizures were recruited through an advertisement and served as a nonclinical control group. All participants were at least 18 years old. Individuals unable to complete self-report questionnaires unaided, not fluent in English, and physically unable to use a computer were excluded.

2.2. Ethical approval

The research was approved by both the Leeds Research and Ethics Committee (REC) and the Research Office of the Sheffield Teaching

Hospitals NHS Foundation Trust. All participants provided written informed consent in accordance with REC guidance and Helsinki Good Clinical Practice.

2.3. Procedure

This was a prospective study; participants were informed that the study was looking at differences in unconscious thinking prior to consenting and initially completed a brief demographic questionnaire before proceeding to the self-report measures outlined below. The order of the questionnaires was randomized using an online research randomizer (available from <http://www.randomizer.org>). Following the completion of these measures, participants completed an IRAP procedure designed for the present study (detailed further below). Assessors were not blinded to diagnosis; however, participants completed the questionnaires independently and separate from assessors.

2.3.1. Demographic and medical history

Basic demographic information (age, gender, and level of education), seizure diagnosis, and seizure frequency were self-reported. Participants were also asked to specify in an open-ended fashion whether they had any current or previous mental health problems.

2.3.2. Spielberger State-Trait Anxiety Inventory (STAI)

The STAI is an explicit self-report measure of state and trait anxiety [57]. It is composed of 40 questions with response options ranging from 1 (not at all/almost never) to 4 (very much so/almost always) on a Likert-type scale. This produces two subscale raw scores ranging from 20 to 80, with higher scores reflecting higher levels of either state or trait anxiety. The STAI was chosen because of its ability to examine both state and trait constructs with test–retest reliability of .40 and .86, respectively. It also has concurrent validity with other measures of anxiety having correlations around .80 [58]. The Cronbach alpha scores for the state and trait measures in this study were .93 and .95, respectively. The state measure of the STAI has also been used as a screening tool for mental disorders in general, with an optimal cutoff score of 54/55 for an accuracy of .87 [59].

2.3.3. Patient Health Questionnaire (PHQ-15)

The PHQ-15 was used as a screening tool for somatization and somatic symptoms [60]. The measure comprises 15 somatic symptoms; each scored either 0 (“not bothered at all”), 1 (“bothered a little”), or 2 (“bothered a lot”). Total scores range from 0 to 30 and are classified as reflecting minimum (0–4), mild (5–9), moderate (10–14), or severe (15+) somatization. The measure was not developed as a standalone diagnostic tool but used to supplement other clinical information. The PHQ-15 has good internal consistency (Cronbach's alpha of .80) and moderate associations between items [60]. The test–retest reliability is moderate with a κ coefficient of .60 [61].

2.3.4. Multidimensional Experiential Avoidance Questionnaire (MEAQ)

Experiential avoidance was measured with the MEAQ [62]. This self-report questionnaire asks participants to indicate the extent to which they agree or disagree with 62 statements (e.g., “When negative thoughts come up, I try to fill my head with something else”) on a 6-point Likert scale from 1 (strongly disagree) to 6 (strongly agree). Total scores range from 62 to 372, with a higher score equating to higher endorsement of avoidance-related statements. Aspects of experiential avoidance measured by the MEAQ include the following: behavioral avoidance, distress aversion, procrastination, distraction and suppression, repression and denial, and distress endurance. The alpha for the total MEAQ score is excellent (.91–.92) with average interitem correlation in the low to moderate range (.15) reflecting the multidimensional nature of the questionnaire and indicating its assessment of a broader range of content compared with other measures of experiential avoidance. In this study, the Cronbach alpha was .91 for the overall scale.

2.3.5. Implicit Relational Assessment Procedure (IRAP)

An IRAP which aimed to specifically target implicit anxiety was developed by the authors (IRAP_{ANX}). The stimulus set for the IRAP_{ANX} was designed to reflect the dimensions of the STAI (Table 1), with stimuli and response options presented and recorded by the IRAP software (available from irapresearch.org). One of two category labels (“I am” or “Others are”) was presented on each trial, with a single target stimulus taken from two sets of stimuli: one set of target stimuli contained anxious terms (e.g., anxious) and the other their semantically opposite terms (e.g., calm). Two response options (“true” or “false”) were also presented on each trial. During consistent trials, participants were required to confirm that they were calm and to deny being anxious; during inconsistent trials, these response requirements were reversed.

The IRAP task was presented on a portable laptop computer. Participants read through instructions presented visually with the experimenter (see appendix A). These instructions explained the IRAP procedure and how to complete the task and highlighted that accuracy and speed in responding were a prerequisite to progress to the test phase. Participants were specifically informed that it would sometimes be necessary to respond to the stimuli in a manner consistent with their beliefs and sometimes in ways that may be inconsistent with their beliefs. Participants were instructed to derive the correct response style for each block of trials but were not told which trials were considered to be consistent or inconsistent. To ensure understanding of the task and minimize random responding, each participant was administered at least two practice blocks until they achieved an average response time of less than 3 s and an accuracy rating above 80% (in line with previous research [48]).

Each trial comprised a category label (“I am” or “Others are”) appearing at the top of the screen, with one of 12 target words in the center (e.g., “anxious”, “worried”, and “calm”) and the two response options “true” and “false” in the bottom corners. All of the stimuli (label, target, and response options) were presented simultaneously (Fig. 1) and remained on the screen until the participant selected one of the relational terms by pressing the ‘D’ key for ‘true’ or the ‘K’ key for ‘false’. Choosing the relational term deemed “correct” for a particular trial removed all stimuli from the screen for 400 ms before the next trial was presented. Choosing the relational term that was deemed “incorrect” for that particular trial produced a red “X” in the center of the screen. To remove the X and proceed to the 400-millisecond inter-trial interval, participants were required to select the correct response option.

An accurate response was dependent on whether a consistent or inconsistent trial was administered. During consistent blocks of the IRAP_{ANX}, participants were required to categorize themselves as calm (e.g., I am – Calm – True and I am – Anxious – False) and others as anxious (e.g., Others are – Anxious – True and Others are – Calm – False). During inconsistent blocks, the response contingencies were reversed. Fig. 1 illustrates the two category labels with their respective consistent and inconsistent stimuli.

During the IRAP, participants were exposed to six test blocks, alternating between consistent and inconsistent blocks, each with 24 trials.

Table 1
The stimulus arrangements for the IRAP_{ANX}.

Sample 1: I am	Sample 2: Others are
Response option 1: true	Response option 2: false
Target stimuli consistent with sample 1	Target stimuli consistent with sample 2
Calm	Tense
Relaxed	Nervous
Rested	Anxious
Comfortable	Scared
Secure	Afraid
Laid-back	Worried

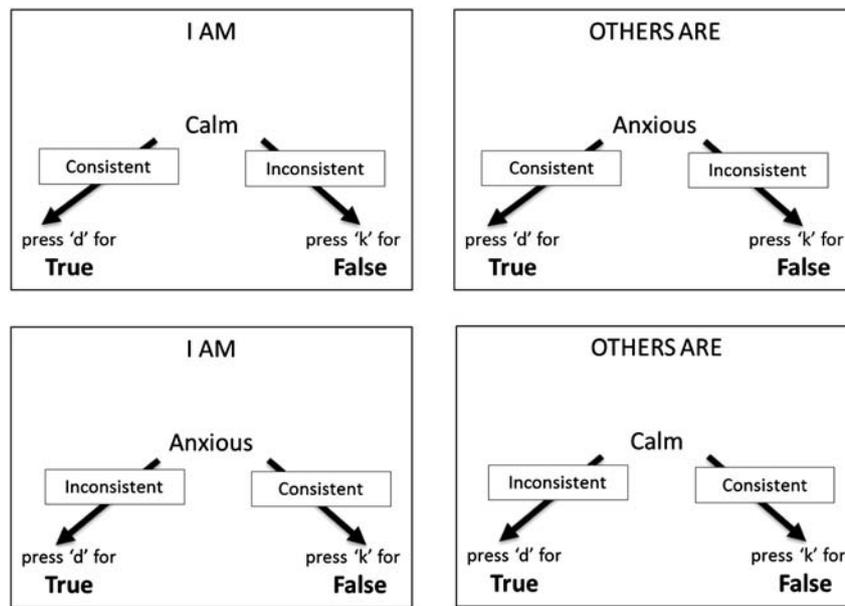


Fig. 1. Examples of the four trial types in the IRAP_{ANK}. Arrows with text boxes showing responses consistent/inconsistent did not appear onscreen and are shown for illustrative purposes only.

The category label and target stimuli within each block were randomized with the constraint that stimuli were not presented more than three times with each sample. Visual instructions after each test block indicated that the next block would involve reversing the previously correct and incorrect responses. Once the final block was completed, participants were thanked and debriefed.

2.4. IRAP data preparation

Raw latency data from the IRAP (time in milliseconds from trial onset to participant response) were converted into a *D* measure (*D*-IRAP), consistent with current implicit measure research outlined by Barnes-Holmes and colleagues [63]. The *D* transformation serves to minimize the impact of individual variability relating to extraneous variables such as age, cognitive ability, and/or motor skills offering a cleaner response latency measurement [64]. *D* scores are relative to response latency differences with larger scores indicating greater differences in response latencies between consistent and inconsistent trials. Implicit Relational Assessment Procedure raw scores were transformed into five *D*-IRAP scores: one for each of the four trial types and an overall *D*-IRAP effect score (mean of the four trial-type scores). Positive scores reflect responding in line with preexperimentally determined consistent items (in the current study: self as calm and others as anxious) and negative scores reflect the reverse (i.e., self as anxious and others as calm). Table 2 details the conversion procedure of the raw latency data. To facilitate interpretation of the results and comparability with

explicit measures, the computed self-trial *D*-IRAP scores were reverse-scored prior to statistical analysis. Consequently, in analyses reported below, positive scores are indicative of anxiety (response tendency toward self as anxious), and negative scores reflect the reverse (self as calm). Implicit anxiety scores are, thus, tuned in the same direction as explicit anxiety scores: i.e., higher positive scores indicative of greater anxiety.

2.5. Statistical analysis

Statistical analysis was completed with IBM SPSS for Windows version 20.0. The explicit measurement data (i.e., self-report measures of state anxiety, trait anxiety, somatic symptoms, and experiential avoidance) were analyzed using a multivariate analysis of variance (MANOVA). Specific predictions were tested using analysis of variance (ANOVA). *Welch's* adjusted *F* is reported where the assumption of homogeneity of variance was not met. Where significant differences were found, Tukey's HSD tests were used to determine where the differences were and correct for multiple comparisons.

For the purpose of computing implicit–explicit discrepancy scores, all indices of self-referent anxiety (explicit trait, explicit state, and implicit self-trials) were first transformed into z-scores (enabling direct comparability) using the appropriate whole sample mean and *SD*. For example, individual trait anxiety z-scores were computed as: $z\text{-trait} = (\text{observed STAI trait score} - \text{Grand Mean STAI trait}) / \text{Grand SD}$. Computed z-scores were then used to compute discrepancy scores by subtracting the

Table 2

The method for converting raw latency scores to the *D*-Implicit Relational Assessment Procedure (*D*-IRAP) scores.

Step	
1	Only use test block data.
2	Eliminate latencies above 10,000 ms from the data set.
3	Remove all data for a participant if 10% of the test block response latencies are less than 300 ms.
4	Calculate 12 standard deviations for the four trial types: 4 from the response latencies from test blocks 1 and 2, 4 from test blocks 3 and 4, and a further 4 from test blocks 5 and 6.
5	Calculate 24 mean latencies for the four trial types in each test block.
6	Calculate difference scores for each of the four trial types, for each pair of test blocks, by subtracting the mean latency of the consistent test block from the mean latency of the corresponding inconsistent test block.
7	Divide each difference score by its corresponding standard deviation from step 4, yielding 12 <i>D</i> -IRAP scores, 1 score for each trial type for each pair of test blocks. Calculate the four overall trial-type <i>D</i> -IRAP scores by averaging the three scores.
8	For each trial type across the three pairs of test blocks. Calculate an overall relative <i>D</i> -IRAP score by averaging all 12 trial-type <i>D</i> -IRAP.
9	Scores from step 8.

implicit z-score (z-transformed $D-IRAP_{ANX}$ self-trials) from the relevant explicit z-score (z-trait for trait discrepancy; z-state for state discrepancy). In this way, higher positive discrepancy scores were indicative of greater explicit relative to implicit anxiety. Transformed z-scores were only used in computation of the anxiety discrepancy scores; untransformed scores were used in analyses of the variables from which these discrepancy scores were derived (preserving original scaling).

3. Results

3.1. Demographics

Groups were closely matched on the variables of gender, age, and education ($ps > .05$), as well as on self-reported seizure frequency ($p > .05$) but differed significantly in relation to self-reported mental health problems ($p = .021$, Fisher's exact test; see Table 3). Participants who self-reported having a mental health problem all stated that they experienced depression, an anxiety disorder, or both. The groups with PNESs and epilepsy did not differ significantly in terms of the proportion of patients above the STAI psychopathology cutoff.

3.2. IRAP results

Eight participants (3 with PNESs, 3 with epilepsy, and 2 controls) were unable to complete the IRAP tasks within the set criterion (median $< 3s, > 80\%$ accuracy). Data from all other participants were retained following the transformation of raw latencies into the $D-IRAP$ scores. The self and other mean $D-IRAP_{ANX}$ scores for the three groups ($N = 78$) are presented in Fig. 2. The data show that all groups demonstrated a general bias toward self and others as calm (illustrated by negative scores).

A 3×4 mixed repeated analysis of variance (ANOVA) was conducted on the $D-IRAP_{ANX}$ scores, with diagnosis as the between-participants variable and trial type as the within-participant variable. There was a substantial effect for trial type, $F(3, 75) = 30.85, p < .001, \eta_p^2 = .01$, with faster responding on the self-trials versus the other-trials. The analysis revealed no significant interaction between diagnosis and trial type, $F(6, 225) = .47, p = .87, \eta_p^2 = .02$, with all groups demonstrating similar responses $F(2, 75) = .59, p = .56, \eta_p^2 = .02$. Four one-way between-participants ANOVAs were also used to conduct planned comparisons for each trial type. No significant effects were found

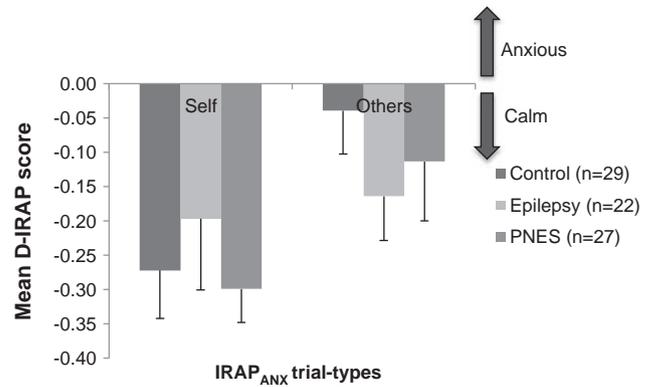


Fig. 2. Mean self and other $D-IRAP_{ANX}$ scores.

(p values $\geq .47$). Contrary to our expectations, this suggests no differences in implicit anxiety between the diagnostic categories.

3.3. Explicit measures

A one-way multivariate analysis of variance (MANOVA) was conducted with group as an independent variable and the four explicit measures (trait anxiety, state anxiety, somatization, and experiential avoidance) as dependent variables. There was a significant multivariate effect of group, Wilks' Lambda = .49, $F(8, 160) = 8.73, p < .001, \eta_p^2 = .30$. To determine which variable(s) differed between groups, a series of four one-way between-groups ANOVAs was carried out. To conservatively protect against multiple testing errors, the alpha criterion for these follow-up ANOVAs was adjusted using sequential Holm–Bonferroni correction (from smallest to largest observed p value, the threshold for significance of omnibus F statistics, thus, ranged from $p < .0125$ to $p < .05$).

There was a significant effect of group on trait anxiety, *Welch's* $F(2, 54.5) = 6.17, p = .004, \eta_p^2 = .15$. Tukey's HSD test indicated that the group with PNESs ($M = 79.00, SD = 50.10$) scored significantly higher compared with the control group ($M = 61.00, SD = 42.84$). The group with epilepsy ($M = 64.00, SD = 38.23$) did not differ significantly from either the control or the group with PNESs. Group differences did not reach significance for state anxiety, as measured by Spielberger's State-Trait Anxiety Inventory, $F(2, 83) = 3.08, p = .051, \eta_p^2 = .07$.

Table 3
Demographic and clinical data of the three groups.

	Controls (n = 31)	Epilepsy (n = 25)	PNESs (n = 30)	<i>p</i>
Gender (%)				
Females	21 (67.7)	16 (64.0)	22 (73.3)	
Males	10 (32.3)	9 (36.0)	8 (26.7)	.75
Mean age (<i>SD</i>)	42.97 (13.93)	39.40 (16.49)	40.87 (12.88)	.65
Highest level of education (%)				
Secondary school	6 (19.4)	4 (16.0)	8 (26.7)	
College/sixth-form	10 (32.3)	9 (36.0)	12 (40.0)	
Diploma	7 (22.6)	5 (20.0)	5 (16.7)	
UG degree	7 (22.6)	2 (8.0)	4 (13.3)	
PG qualification	1 (3.2)	5 (20.0)	1 (3.3)	.43
Number reporting mental health problems (%)				
None	23 (74.2)	17 (68.0)	14 (46.7)	
Past	6 (19.4)	5 (20.0)	4 (13.3)	
Present	2 (6.5)	3 (12.0)	12 (40.0)	.02
Number above STAI psychopathology cutoff (%)	1 (3.2)	2 (8.0)	7 (23.3)	.16
Number of seizures reported per month				
Mean (<i>SD</i>)	–	4.38 (7.48)	7.36 (7.45)	
Median (<i>IQR</i>)	–	1.0 (0.0–7.3)	6.0 (2.0–12.0)	.09

Note: *SD* = standard deviation; *IQR* = interquartile range; UG = Undergraduate; PG = Post-graduate; group differences for highest level of education, number reporting mental health problems, and STAI psychopathology cutoff were tested using Fisher's exact test to account for small cell sizes; gender was tested using a chi-squared test; seizure frequency was based on self-report estimates and tested using the Kruskal–Wallis test.

There was a significant difference between the three groups on reported somatic symptoms, as measured by the PHQ15; *Welch's F* (2, 52.49) = 29.21, $p < .001$, $\eta_p^2 = .49$. Tukey's HSD test revealed that the group with PNESs ($M = 14.80$, $SD = 6.19$) scored significantly higher compared with the control group ($M = 5.00$, $SD = 3.33$) and the group with epilepsy ($M = 6.60$, $SD = 3.46$). The group with epilepsy and the control group did not significantly differ from each other.

Finally, there was a significant difference between the three groups on experiential avoidance (MEAQ total score); *Welch's F* (2, 54.07) = 8.89, $p < .001$, $\eta_p^2 = .21$. Tukey's HSD test indicated that the group with PNESs ($M = 235.50$, $SD = 48.86$) scored significantly higher compared with the control group ($M = 190.03$, $SD = 34.73$) and the group with epilepsy ($M = 198.68$, $SD = 33.37$). The group with epilepsy and the control group did not differ significantly from each other.

Overall, consistent with expectations, the group with PNESs scored significantly higher compared with the healthy control group and the group with epilepsy on somatization and experiential avoidance; the group with PNESs also scored significantly higher on trait anxiety compared with the control group (but not the group with epilepsy). Fig. 3 summarizes group scoring on the explicit measures and highlights significant differences.

3.4. Implicit–explicit discrepancies

To test the hypothesis that there would be larger discrepancies between the implicit and explicit measures of anxiety in patients with PNESs, a one-way between-groups ANOVA was conducted. There was a statistically significant difference for the three groups in terms of discrepant anxiety, $F(2, 75) = 6.26$, $p = .003$, $\eta_p^2 = .14$. Tukey's HSD test indicated that the group with PNESs had significantly larger discrepancies compared with the control group and the group with epilepsy, who did not differ significantly from each other. These discrepancies are illustrated in Fig. 4.

3.5. Relationships between avoidance and anxiety

Within-group relationships between experiential avoidance and anxiety/somatization were examined using Pearson's correlations (see Table 4). For each set of correlations within each group (i.e., control, epilepsy, and PNESs), significance levels were adjusted for multiple testing using a sequential Holm–Bonferroni procedure. Table 4 highlights both relationships that were only significant before adjusting the .05 alpha criterion for multiple testing (*) and relationships that

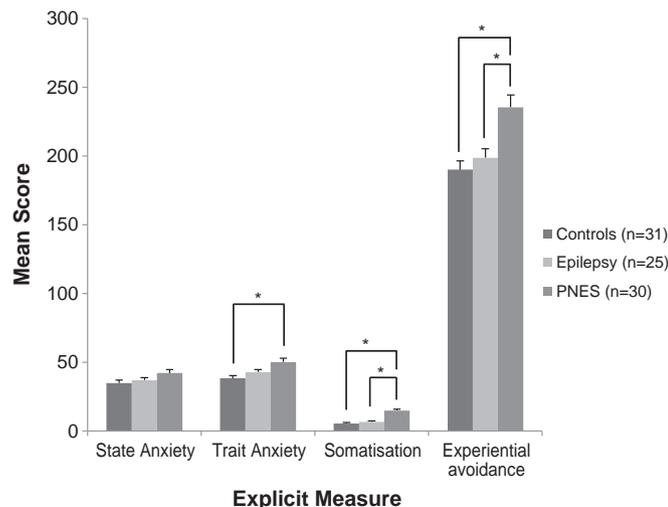


Fig. 3. Mean scores on each of the explicit self-report measures, with standard bars indicating one standard error; * $p < .05$.

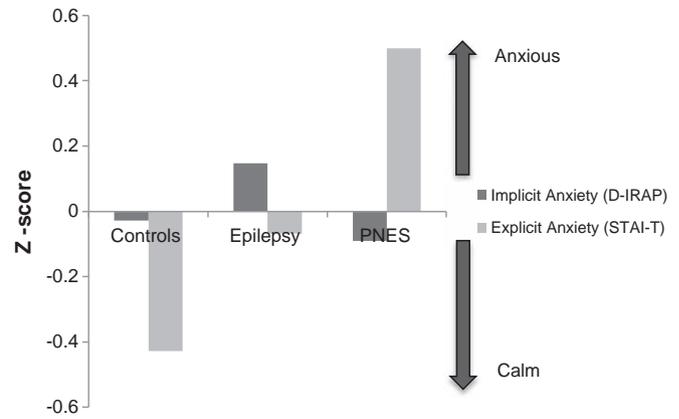


Fig. 4. Discrepancies between implicit and explicit anxiety.

remained significant after adjustment (**). Given the limited power within each group, it can be seen that only relationships with large effect-sizes ($r_s \approx .50$) met adjusted criteria for significance.

After adjustment, avoidance was positively associated with (1) higher explicit trait anxiety levels and (2) greater discrepancy scores between (high) explicit trait anxiety and relatively (low) implicit anxiety in the group with PNESs. No significant relationships were found between avoidance and implicit anxiety scores in the group with PNESs ($ps > .16$), and none of the relationships were significant for the group with epilepsy or the control group.

3.6. Psychological factors and seizure frequency

The relationship between state and trait anxiety, experiential avoidance, and somatization and seizure frequency was investigated using Spearman's rank order correlations (Table 5). For each family of tests (correlations within each group and comparative Fisher Z tests), significance levels were adjusted for multiple testing using a sequential Holm–Bonferroni procedure as before.

In the group with epilepsy, there were no significant correlations between seizure frequency and any of the psychological measures. In the group with PNESs, there were strong positive correlations between seizure frequency and trait anxiety, implicit anxiety, and avoidance.

3.7. Predicting diagnosis

As somatization (PHQ-15) and experiential avoidance (MEAQ) were significantly higher in the group with PNESs compared with the group with epilepsy, these were analyzed by using univariate binary logistic regression to assess how well they predicted diagnoses. The full model containing both predictors was statistically significant, $\chi^2(3, N = 55) = 32.05$, $p < .001$, indicating that the model could

Table 4
Correlations with experiential avoidance.

	Controls	Epilepsy	PNESs
State anxiety			
Explicit	-.05	.02	.41*
Explicit–implicit	-.09	.10	.27
Trait anxiety			
Explicit	.09	.20	.63**
Explicit–implicit	.00	.24	.49**
Somatization	.10	-.13	.18
Implicit anxiety			
Self D-IRAP _{ANX}	.10	-.10	.16

* Indicates r values that are significant at the unadjusted $p < 0.05$ level.

** Indicates r values that remain significant following Holm–Bonferroni correction for multiple testing (all $ps < .01$).

Table 5
Correlations with seizure frequency.

	Group with epilepsy	Group with PNEs	Test of difference: Fisher Z
State anxiety			
Explicit	-.06	.36	-1.52
Explicit-implicit	-.07	-.16	0.32
Trait anxiety			
Explicit	-.03	.67**	-2.93**
Explicit-implicit	-.04	.16	-0.42
Somatization	.34	.38	-0.16
Experiential avoidance	-.02	.55**	-2.22*
Implicit anxiety			
Self <i>D</i> -IRAP _{ANX}	.09	.56**	-1.89

* Indicates *r* and two-tailed Fisher Z values that are significant at the unadjusted $p < 0.05$ level.

** Indicates *r* and two-tailed Fisher Z values that remain significant following Holm-Bonferroni correction for multiple testing (all $ps < .01$).

predict individuals with either PNEs or epilepsy. The model was able to explain between 44.2% (Cox and Snell *R* square) and 59.1% (Nagelkerke *R* square) of the variance in diagnosis and correctly classified 83.6% of the cases (84.0% sensitivity and 83.3% specificity). As shown in Table 6, both somatic symptoms and avoidance made a unique statistically significant contribution to the model. The addition of implicit anxiety scores did not improve the model significantly.

4. Discussion

The current study aimed to examine implicit and explicit anxiety in people with PNEs, explore the relationship with experiential avoidance and PNEs frequency, and determine whether they could be useful in discriminating between people with PNEs and epilepsy.

In line with previous findings, individuals diagnosed with PNEs or epilepsy self-reported significantly higher levels of anxiety compared with nonclinical controls [28]. However, no significant differences were found between the two clinical groups themselves. The group with PNEs endorsed significantly more somatic complaints compared with both the group with epilepsy and the healthy control group, as well as reported significantly higher levels of experiential avoidance consistent with previous findings [2,65]. Frequency of PNEs was strongly correlated with explicit anxiety scores and experiential avoidance; however, consistent with some previous reports [66] but in contrast with others [67], psychological factors as measured in the present study were unrelated to the frequency of epileptic seizures within the group with epilepsy.

Uniquely, this study also examined implicit anxiety in people with PNEs. In contrast to our expectations, we found no clear differences between patients with PNEs and those with epilepsy or healthy controls in terms of implicit anxiety. Importantly, however, we did detect significantly larger discrepancies in implicit and explicit anxiety scores in the group with PNEs compared with the two comparison groups. What is more, there was a strong positive correlation between implicit anxiety scores and PNEs (but not epileptic seizure) frequency. These novel findings are discussed in more detail below.

Table 6
Logistic regression predicting diagnosis.

	<i>B</i>	<i>SE</i>	Wald	Odds ratio	95% CI for odds ratio	
					Lower	Upper
Somatization	.32	.09	11.89**	1.34	1.15	1.65
Experiential avoidance	.02	.01	8.77**	1.02	1.00	1.04

Note: CI = confidence interval.

** $p < .01$.

4.1. Anxiety

The current findings suggest that individuals with PNEs may not hold automatic or unconscious perceptions of themselves as anxious, despite reporting more anxiety than control participants on explicit measures. One interpretation of these results is based on the dual-attitude model formulated by Wilson and colleagues that suggests that implicit measures reflect older, habitual cognition [68]. A profile of low-implicit high-explicit anxiety could, therefore, be reflective of individuals who have become anxious later in life; in populations with PNEs, this may relate to explicit anxiety developing following the onset of the seizures themselves. However, a more plausible explanation for this pattern might be that the dissociation associated with PNEs themselves (combined with wider avoidance tendencies in a patient's life in between seizures) effectively stop patients from holding implicit anxious cognitive biases which they might have developed in the absence of PNEs. Consequently, those with PNEs may explicitly report anxiety while failing to "internalize" anxiety as part of their self-concept. Such a "protective" function of PNEs could also help explain the observation that patients with PNEs report more negative life events compared with those with epilepsy but fail to make a link between these life events and their seizures [4] or that a large subgroup of patients with PNEs are limited in their emotional and psychological awareness [69].

Despite the fact that the group with PNEs was characterized by a discrepancy between low-implicit anxiety and high-explicit anxiety, we found a strong correlation between greater implicit anxiety and higher PNEs frequency. It is possible that this finding reflects the psychopathological heterogeneity of PNEs: previous studies have identified at least two major groups characterized by low and high levels of emotional dysregulation [70]. Psychogenic nonepileptic seizures may be linked to implicit anxiety in some and explicit anxiety in other patients.

A previous study in a nonpatient population demonstrated that implicit anxiety predicted cardiovascular responses (i.e., heart rate and blood pressure) to threat above and beyond explicit measures [71]. Importantly, the authors of this study highlighted that implicit anxiety only predicted cardiovascular responses to acutely stressful events rather than cardiovascular responding more generally. Nevertheless, given that it predicted responses measured at later points in time, they suggest that implicit anxiety has a "trait"-like influence on behavior. Previous studies have demonstrated that PNEs are associated with similar heart rate variability (HRV) changes to those seen in acute stress [72]. Future studies could explore to what extent implicit anxiety and seizure frequency relate to such physiological responses in this patient group.

This study is the first to show a relationship between self-reported trait anxiety and PNEs frequency. While the strong positive correlation does not allow us to draw definite conclusions about the direction of the relationship, the fact that trait rather than state anxiety was correlated with PNEs frequency supports previous suggestions that anxiety plays an important etiological role in PNEs [65,73]. A variety of psychological theories can be applied to account for the proposed relationship between PNEs and anxiety; psychodynamic theories, for example, conceptualize anxiety as the by-product of an intrapsychic conflict and propose that PNEs can be a symptom of that conflict [74]. Behavioral models of human functioning (e.g., [75]) can also be adapted to explain the observed relationship between anxiety and PNEs in terms of conditioned responses and reinforcement history; such theories postulate that anxiety is a conditioned response to a threat or trigger (e.g., a flashback or a familial conflict) and that PNEs consequently function as a negatively reinforcing response to threat and anxiety, perpetuating their occurrence in threat-inducing situations [76].

4.2. Experiential avoidance

As expected, as well as in line with previous research, individuals with PNEs reported higher levels of avoidance compared with those

with epilepsy [7,9,65,77]. The results of this study extend prior research by highlighting the idea that it is especially emotional experience that people with PNEs work to avoid, including greater avoidance of painful and uncomfortable feelings, emotional disconnection, and believing that negative emotions are damaging.

In the current sample, experiential avoidance did not correlate with somatic symptoms. However, avoidance was strongly correlated with self-reported seizure frequency in the group with PNEs. The present study, therefore, is consistent with the idea that experiential avoidance (perhaps as an “overlearned” or practiced response style) may be a risk factor for the development of PNEs. Similarly, Myers and colleagues [69] found that reports of alexithymia, which refers to a lack of emotional awareness and expression, did not differ between PNEs and epilepsy but that within the group with PNEs, alexithymia was associated with anxious arousal and avoidance.

Finally, we observed a strong positive correlation between discrepant implicit–explicit anxiety scores and experiential avoidance. Recent studies on implicit cognition have conceptualized such discrepancies from within a cognitive dissonance theory perspective [78], suggesting that aversive dissonance-related discomfort increases when implicit and explicit beliefs diverge [79]. The application of cognitive dissonance theory to PNEs may, therefore, suggest that nonepileptic seizures function to reduce cognitive dissonance, and targeting this dissonance (e.g., using strategies from dialectical behavior therapy (DBT; [80])), is an avenue for future research and treatment approaches in populations with PNEs.

4.3. Implications

Recent developments in screening measures aimed at facilitating the differential diagnosis of epilepsy and PNEs are promising [81]. The results presented here suggest that the inclusion of avoidance scales may enhance the predictive utility of such tools. The information provided by patients on such measures may also aid health professionals in developing formulations and intervention plans and evaluating outcomes.

Cognitive behavioral therapy (CBT) and psychodynamic therapy are the leading published psychological interventions effective for PNEs [82–86]. Our findings support the idea that increasing tolerance of unpleasant emotions and reducing maladaptive avoidant behavior patterns might represent mechanisms of change in these approaches. Therapies which directly target experiential avoidance, such as acceptance and commitment therapy (ACT), or DBT (which also addresses cognitive dissonance, as noted above [80]), may be useful in augmenting treatment for patients with PNEs [87].

4.4. Limitations

There are a number of limitations within the current study that require acknowledgment. Patients were only recruited to the study if they had a firm diagnosis, but the amount of time for which they had been experiencing seizures, any formal psychiatric diagnosis, or whether they were prescribed any psychotropic medication or anti-epileptic drugs were not recorded. The fact that many patients had a chronic seizure disorder means that it is more difficult to draw conclusions about the direction of the relationship between the psychological variables and PNEs. In addition, only the relationship between psychological variables and seizure frequency was explored; one previous study showed that seizure severity was a predictor of psychological variables in epilepsy [66], and, therefore, future studies may want to consider the role of both severity and frequency. Moreover, this study was conducted with patients with seizures receiving current outpatient neurology care; it is, therefore, uncertain to what extent the results can be generalized to other patient groups.

In terms of methodology, the IRAP stimuli were developed specifically to reflect dimensions of the explicit anxiety scale used in the study. The term ‘others are’ was used to avoid double negatives (e.g., I am

not anxious — false), which can be problematic in IRAP research, but this rewording may have not been as effective in capturing people’s beliefs about themselves in relation to others. Although there was no indication that our measure was ineffective in this population, it is nevertheless possible that there are differences in implicit cognition in people with PNEs that the IRAP did not successfully detect. The results of implicit assessments depend on the specific stimuli presented. It is important that the stimuli used are salient to the individual completing the measure and relate to the phenomena of interest. For example, nonword stimuli, or words based on other conceptualizations of anxiety, may have yielded different results.

Finally, this study did not use blinded assessors or implement any scales of effort or social desirability, and while it seems unlikely that differences in explicit anxiety were due to exaggerated responses, it is possible that the results were due to a response bias [88].

5. Conclusion

In conclusion, this study found significant differences between people with PNEs, those with epilepsy, or those without a history of seizures in terms of experiential avoidance and explicit (self-reported) anxiety, as well as significant relationships between PNE frequency with implicit anxiety, explicit anxiety, and experiential avoidance. While there were greater implicit versus explicit anxiety discrepancies in the group with PNEs, implicit anxiety levels did not differ between the three groups. These findings support various psychological models of PNEs and offer a rationale for psychological treatments targeting avoidant behavior patterns or cognitive dissonance.

Conflict of interest

The authors report no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.yebeh.2014.02.016>.

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