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Development and Cross-Validation of Personality Assessment Inventory Decision Rules for the Identification of Psychogenic Nonepileptic Seizures

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DEVELOPMENT AND CROSS-VALIDATION OF
PERSONALITY ASSESSMENT INVENTORY DECISION RULES
FOR THE IDENTIFICATION OF PSYCHOGENIC NONEPILEPTIC SEIZURES

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Arts and Sciences
at the University of Kentucky

By
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2021

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ABSTRACT OF DISSERTATION

DEVELOPMENT AND CROSS-VALIDATION OF PERSONALITY ASSESSMENT INVENTORY DECISION RULES FOR THE IDENTIFICATION OF PSYCHOGENIC NONEPILEPTIC SEIZURES

The published literature on the Personality Assessment Inventory (PAI) for psychogenic nonepileptic seizure (PNES) diagnosis includes a variety of interpretation methods to distinguish PNES from epileptic seizures (ES) and offers mixed findings. The purpose of this study was to use a cross-validation approach to create and derive new decision rules for the PAI to best differentiate PNES from ES. Data from 773 patients (PNES $n = 328$, ES $n = 445$) who underwent long-term video EEG (vEEG) monitoring and completed a PAI were examined. Individuals with invalid PAI profiles were removed, and patients were randomly assigned to the “development” group (DEV) or the “application” group (APP). Receiver operating characteristic (ROC) curves with DEV demonstrated the best cut score for each scale of interest. ROC curves were repeated with APP. Additional analyses examined the utility of sequential decision rules incorporating multiple scales. Of the individual scales, SOM-C demonstrated the best diagnostic accuracy (sensitivity [SN] = 60.7%, specificity [SP] = 81.3%) at a cut score of $T \geq 75$. Cross-validation with APP confirmed this cut score outperformed other cut scores (positive predictive value [PPV] = 67.2%, negative predictive value [NPV] = 76.1%), as well as other decision rules presented in the literature. Additional analyses examining sequential decision rules with SOM-C ≥ 75 or SOM-C = 70-74 with SOM-S ≥ 65 demonstrated the highest predictive power (PPV = 73.2%, NPV = 79.1%). The results of this study demonstrate a new and effective method for using the PAI as a screener to distinguish PNES from ES. Utilization of these decision rules can assist clinicians in determining appropriateness of and immediate need for vEEG monitoring for diagnostic clarification.

KEYWORDS: Psychogenic Nonepileptic Seizures, Epilepsy, Personality Assessment, Diagnostic Classification

Chelsea Marie Bosch

10/26/2021

Date

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DEDICATION

To my husband Matthew, who has been a constant source of strength and encouragement during the challenges of graduate school and life. Words cannot express my gratitude for your unwavering support. And for my son August, who inspires me every day to continue chasing my dreams. This work is also dedicated to my parents, Jeff and Debbie Bouquet, who have always loved me unconditionally and whose good examples have taught me to work hard for the goals that I aspire to achieve.

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CHAPTER 1. INTRODUCTION

1.1 Psychogenic Nonepileptic Seizures (PNES)

Psychogenic nonepileptic seizures (PNES) are considered to be a form of conversion disorder characterized by involuntary sensations or behaviors that may resemble an epileptic seizure but are not associated with abnormal electrical activity in the brain (Asadi-Pooyah & Sperling, 2015). Instead, PNES are typically a result of significant psychological dysfunction that manifests in a physical manner. Between 2 and 33 people per 100,000 experience PNES, making the condition relatively common (Alsaadi & Marquez, 2005). Given their paroxysmal nature and associations with convulsions and alterations in consciousness, PNES is oftentimes confused with epileptic seizures (ES; Devinsky et al., 2011). Further complicating the diagnosis of this condition, both PNES and ES can occur concurrently, at prevalence rates ranging from 10% to over 50% (Benbadis et al., 2001).

The co-occurrence of these conditions paired with their similar presentations oftentimes leads to a delay in diagnosis for individuals suffering from PNES. On average, individuals with PNES experience a mean delay of 7.2 years before diagnosis (Reuber et al., 2002). The time to diagnosis can be impacted by several demographic and clinical factors including age of seizure onset, use of antiepileptic medication, and occurrence of ictal injury (Bahrami et al., 2019). Misdiagnosis may occur because individuals with PNES often first appear to general practitioners instead of epileptologists (Reuber & Elger, 2003). Inaccurate diagnosis of PNES is not only an obstacle to obtaining appropriate treatment and relief from symptoms; the effects of mistakenly being diagnosed with ES can be detrimental to those with PNES. In addition to exorbitant healthcare costs,

individuals with PNES are at risk for iatrogenic hazards resulting from unnecessary use of antiepileptic drugs, poor psychosocial outcomes, overall poor quality of life, and even early death (Martin et al., 1998; Ahmedani et al., 2013; Reuber et al., 2004). Following an inpatient video-electroencephalography (vEEG) monitoring stay resulting in a definitive diagnosis of PNES, patients saw a dramatic reduction in healthcare costs over a 12-month period associated with fewer inpatient stays and neurology visits (Ahmedani, et al., 2013). This finding highlights the need for better tools to identify possible PNES, so patients can receive an accurate diagnosis, the burden of the disorder can be minimized, and treatment can be tailored effectively.

1.2 Diagnosing PNES

Approaches to diagnosing PNES are numerous, and some methods are more successful than others. To address this issue, the International League Against Epilepsy (ILAE) has outlined a staged approach to diagnosing PNES, providing four levels of diagnostic certainty including possible, probable, clinically established, and documented (LaFrance et al., 2013). These labels are based on combinations of clinical history, witnessed events, and EEG data. Presently, the gold standard for distinguishing PNES from ES is long-term vEEG, routinely conducted in an inpatient epilepsy monitoring unit (Popkirov et al., 2017). This technique has the advantage of capturing both behavioral and potential ictal EEG data simultaneously over a prolonged period making an accurate diagnosis more likely.

Unfortunately, vEEG monitoring is not a widely available resource in most medical centers, and it is very complex and costly (Wagner et al., 2005). For this reason, clinicians often use short-term interictal EEG monitoring. This diagnostic approach requires only a

short outpatient visit, and the service is more widely available. The absence of interictal epileptiform discharges on routine EEG is thought to differentiate PNES from ES; however, this method comes with a high false negative rate for detecting ES due to the limited duration of activity recorded (Cragar et al., 2002). According to the ILAE guidelines, even a routine EEG showing no epileptiform activity during a typical event (for which semiology would suggest expectable epileptiform activity), a clinician cannot obtain the highest level of diagnostic certainty. To achieve the highest level of diagnostic certainty (documented), a vEEG must be obtained.

In addition to the traditional use of EEG to differentiate PNES from ES, clinicians often employ several other variables to inform their decision. Demographic and medical history is relatively easy to obtain through clinical interview and review of the electronic health record. Researchers have reported that women experience PNES at approximately three times the rate of men (Myers et al., 2018). Individuals with PNES are also more likely to have a history of psychiatric treatment, history of abuse (physical and sexual), and a later age of seizure onset than individuals with epilepsy (Cragar et al., 2002). Semiology of the events can also provide useful information distinguishing PNES from ES, with ES often evidencing stereotyped movement, shorter spells, events during sleep, and postictal confusion (Cragar et al., 2002).

Additionally, some new techniques are gaining interest among clinicians and researchers in the diagnosis of PNES including the use of biomarkers and neuroimaging. Researchers have suggested that examination of four plasma protein concentrations (TRAIL, ICAM-1, MCP-2, and TNF-R1) can effectively differentiate PNES from ES (Gledhill, et al., 2021); however, this research is still in the early stages and additional

research is needed to determine the clinical utility of these findings. Regarding neuroimaging, 18F-fluorodeoxyglucose positron emission tomography (FDG-PET) has been useful in identifying areas of hypometabolism in the brain that have assisted in surgical decision-making for individuals with intractable epilepsy (Rathore et al., 2014). When compared to healthy controls, examination of PET imaging with a PNES sample demonstrated hypometabolism in the right inferior parietal/central brain region and bilateral anterior cingulate (Arthuis et al., 2015). Additional research comparing PNES and ES samples is needed to determine the utility of PET imaging in diagnostic clarification. Another neuroimaging technique of interest is single-photon emission computed tomography (SPECT). With this approach, cerebral blood flow changes between ictal and interictal scans can identify a seizure focus. In instances where no cerebral blood flow abnormalities are found, PNES may be suspected (Cragar et al, 2002; Neiman et al., 2009). This diagnostic method is also very costly and not widely available; however, it may present an additional tool for clinicians with complex cases when results from more traditional methods are unclear.

1.3 Psychological Assessment for PNES

In addition to the aforementioned procedures, researchers have examined the utility of psychological assessment measures to distinguish between PNES and ES. Measures of personality and psychopathology are useful in providing a comprehensive and person-centered evaluation, taking into consideration the potential function PNES serves in a person's life. Additionally, they are generally less expensive and less time-consuming than medical approaches. A summary of published decision rules with psychological assessments can be found in Table 1. Historically, the Minnesota Multiphasic Personality

Inventory (MMPI; Hathaway & McKinley, 1942) and its subsequent editions (MMPI-2, MMPI-2-RF; Butcher et al. 1989; Ben-Porath & Tellegen, 2008) have been the most consistently studied measures (Barrash et al., 1989; Del Bene et al., 2017; Derry & McLachlan, 1996; Henrichs et al., 1988).

Wilkus and colleagues (1984) were among the first to create decision rules for the MMPI to differentiate PNES from ES. According to their algorithm, a person was classified as having PNES if scales 1 (Hypochondriasis) or 3 (Hysteria) have a T score of 70 or higher and were one of the two highest scales, disregarding scales 5 (Masculinity-femininity) and 0 (Social introversion). Additional criteria involve scales 1 or 3 with T scores of 80 or higher (regardless of other scale elevations) or scales 1 and 3 both at a T score of 60 or higher, and they are at least 10 points higher than scale 2, mirroring the conversion “V” (Wilkus, et al., 1984). Using these rules, they demonstrated 87% sensitivity and 81% specificity to PNES, though with a limited sample size (N = 20). A review presented by Cragar and colleagues (2003) further examined the accuracy of these decision rules across ten studies and determined an average sensitivity of 62% and an average specificity of 75% when compared to vEEG diagnoses.

Once the MMPI was restandardized and the MMPI-2 was released, Derry and McLachlan (1996) established a new set of decision rules with the goal of increasing classification accuracy between PNES and ES. These new rules were designed to be simpler than those for the MMPI and indicate a diagnosis of PNES if the following criteria are met: (a) a T score of 65 or greater on scale 1 (Hypochondriasis) and/or scale 3 (Hysteria), (b) scale 1 or 3 appearing in the 2-point code type, and (c) if scale 1 or 3 was not the highest elevation, it must be less than or equal to 6 points below the highest

elevation (Derry & McLachlan, 1996). These rules demonstrated 92% sensitivity for PNES and 94% specificity within their sample. Cragar and colleagues (2003) attempted to validate these decision rules and found sensitivity of only 48% and specificity of only 58%. Using the same sample, Cragar and colleagues (2003) attempted to validate a set of modified Wilkus et al. (1984) decision rules that reflected scale changes from the MMPI to the MMPI-2 and obtained 68% sensitivity and 55% specificity. Ultimately, the high levels of diagnostic accuracy reported for the MMPI and MMPI-2 have not been replicated, suggesting it is limited at differentiating between PNES and ES.

With the release of the MMPI-2 Restructured Form (MMPI-2-RF) came significant changes to the clinical scales. The purpose of these changes was to increase scale homogeneity and reduce intercorrelations between scales (Ben-Porath, 2012). Locke and Thomas (2011) developed two complementary scales assessing physical complaints (PNES-pc) and attitudes (PNES-a) based on MMPI-2-RF items to classify patients as having PNES or ES. Diagnostic accuracy for these scales is maximized with a PNES-pc total score greater than or equal to 3 and PNES-a total score greater than or equal to 3, with 73% sensitivity and 73% specificity (Locke & Thomas, 2011). At the scale level, significant differences between PNES and ES patients have been demonstrated on RC1 (Somatic Complaints), RC3 (Cynicism), MLS (Malaise), GIC (Gastrointestinal Complaints), HPC (Head Pain Complaints), NUC (Neurological Complaints), SUI (Suicidal/Death Ideation), NFC (Inefficacy), and SUB (Substance Abuse; Del Bene, et al., 2017; Duncan et al., 2018; Locke et al., 2010). Due to the variability in findings, further research is needed to determine specific scales and clinical cutoffs to best differentiate PNES from ES with the MMPI-2-RF.

In response to the variability of the diagnostic accuracy of the MMPI, the Personality Assessment Inventory (PAI; Morey, 1991) has gained popularity as a replacement in many clinical settings. Compared to the MMPI/MMPI-2, the PAI offers many advantages. It requires a lower reading level (4th grade vs. 8th grade), consists of fewer items (344 items vs. 567 items), is composed of non-overlapping items between scales, and provides broader response options (4-point scale vs. true-false; Mason et al., 2000). Wagner and colleagues (2005) created the first decision rule for the PAI, referred to as the NES Indicator score. They hypothesized that there would be a difference in the Somatic Complaints subscales Conversion (SOM-C) and Health Concerns (SOM-H), with individuals with PNES showing higher elevations on Conversion than Health Concerns (Wagner et al., 2005). This belief was based on the idea of *la belle indifférence*, which suggests that individuals with conversion disorder often appear unconcerned about their unusual physical symptoms. Their NES Indicator is derived by subtracting the T score of the Health Concerns subscale from the T score of the Conversion subscale such that positive scores indicate PNES, whereas negative scores indicate ES (Wagner et al., 2005). This methodology demonstrated 84% sensitivity for diagnosing PNES and 73% specificity (Wagner et al., 2005). An attempt to replicate these findings demonstrated 59% sensitivity and 85% specificity (Thompson et al., 2010).

Researchers have also examined the utility of single scale and subscale elevations in distinguishing PNES from ES with notable differences reported in most areas, including somatic complaints, anxiety, anxiety-related disorders, depression, mania, schizophrenia, borderline features, antisocial features, alcohol problems, suicidal ideation, stress, treatment rejection, dominance, and warmth (Mason et al., 2000; Pritchard et al., 2002;

Testa et al., 2011; Thompson et al., 2010; Wagner et al., 2005). These scale distinctions between PNES and ES were ambiguous across studies and are suggestive of a need for a more comprehensive examination of PAI scale elevations to improve diagnostic accuracy. Few studies have directly compared the diagnostic accuracy of the MMPI and its subsequent editions to the PAI. In a conference abstract, Stroup and colleagues (2006) demonstrated 75% classification accuracy with the PAI using a cut score of 70T on the Somatic Complaints-Conversion (SOM-C) subscale or the Depression-Physiological (DEP-P) subscale; they found 69% classification accuracy with the MMPI using the Wilkus et al. (1984) decision rules. Locke et al. (2011) completed a randomized prospective comparison of the PAI and MMPI-2/MMPI-2-RF to differentiate seizure type in an epilepsy monitoring unit. They found that the PAI indicators (SOM \geq 70T, SOM-C \geq 70T, Stroup criteria, Wagner NES indicator) outperformed the MMPI-2 indicators. Correct classification rates for the PAI indicators ranged from 71-79%, whereas correct classification rates for the MMPI-2 ranged from 63-68% (Locke et al., 2011). After exploring the diagnostic validity of the PAI and the MMPI-2-RF at multiple cut scores, the PAI also outperformed the MMPI-2-RF with likelihood ratios as high as 9.86 and 9.61 at scores \geq 80T on the SOM and SOM-C scales compared to likelihood ratios ranging from 1.72 to 5.16 on the MMPI-2-RF RC1 or Locke and Thomas (2011) indicator. Notably, Gale and Hill (2012) were the first to administer both the PAI and the MMPI-2 to the same sample, and they reported the best classification accuracy (85%) with a combination of both tools, including elevations on the PAI's SOM-C and SOM-H and the MMPI-2 Hy scales.

1.4 Meta-Analytic Findings

The MMPI and its subsequent editions have a long history of diagnostic utility in differentiating PNES from ES. However, the PAI offers many advantages over the MMPI/MMPI-2 including nonoverlapping scales, lower reading level, and fewer items, making it a desirable choice for integration into diagnostic consultations for seizures. The PAI is a comparatively new tool for the purpose of seizure classification, and researchers have reported mixed findings regarding optimal use to obtain the highest level of diagnostic accuracy.

A recent meta-analysis (Bosch et al., 2020) sought to synthesize these findings and investigate the utility of the PAI to differentiate between PNES and ES groups. This analysis examined 12 studies ($N = 1,838$) and focused on the effectiveness of the SOM, ANX, and DEP scales and subscales given their conceptual and empirically supported relevance to PNES. Findings of this analysis suggested that there are marked differences in the PAI profiles of PNES and ES groups. At the clinical scale level, there was a large effect observed for SOM ($g = 0.82$) and small effects observed for ANX ($g = 0.32$) and DEP ($g = 0.45$). At the subscale level, large effects were seen for SOM-C ($g = 1.02$) and SOM-S ($g = 0.92$), and medium effects were seen for ANX-P ($g = 0.56$) and DEP-P ($g = 0.66$). Weighted mean T score data demonstrated clinical elevations ($T \geq 70$) for the PNES group on SOM ($M = 74.0, SD = 1.6$), SOM-C ($M = 75.8, SD = 1.2$), and SOM-H ($M = 70.6, SD = 1.4$) and moderate elevations ($T \geq 60$) on DEP ($M = 63.0, SD = 2.1$), SOM-S ($M = 68.2, SD = 1.0$), ANX-P ($M = 61.3, SD = 0.9$), and DEP-P ($M = 66.1, SD = 2.1$). The ES group did not demonstrate clinical elevations on any scales, but they had moderate

elevations on SOM ($M = 63.8$, $SD = 1.3$), SOM-C ($M = 60.9$, $SD = 1.4$), and SOM-H ($M = 67.2$, $SD = 1.3$).

Based on these findings, it appears that clinically elevated scores on SOM and SOM-C could be predictive of PNES, and these scores paired with moderate elevations on SOM-S, ANX-P, and DEP-P would offer secondary support for a diagnosis of PNES. This meta-analysis was helpful in determining patterns of scores that may best distinguish PNES from ES, but additional research is needed to determine appropriate cut scores that offer the highest classification accuracy.

1.5 The Present Study

The purpose of the present study is to build on the meta-analytic findings by using a cross-validation approach to create and derive new decision rules for the PAI to differentiate PNES from ES. Informed data interpretation from an objective psychological inventory will improve clinical insight when making diagnostic determinations. The results can serve as an effective screener in distinguishing PNES from ES and assist in determining which patients would be best suited to undergo vEEG monitoring for diagnostic certainty. This study will utilize data from all eleven clinical scales and their subscales, as well as the treatment consideration scales and interpersonal scales to consider comprehensive PAI profiles for patients with PNES and those with ES. Observed score differences between the groups and calculated values of classification accuracy will inform the decision rules.

Table 1 Previously Published Decision Rules for Psychological Assessments

Source	Assessment	Rule
Wilkus et al., (1984)	MMPI	Scales 1 (Hs) or 3 (Hy) $\geq 70T$ and one of the two highest scales (disregarding scales 5 and 0, Mf and Si) OR Scales 1 or 3 $\geq 80T$ regardless of other scale elevations OR Scales 1 and 3 $\geq 60T$ and 10 points higher than scale 2 (D)
Derry and McLachlan (1996)	MMPI-2	Score of $\geq 65T$ on scale 1 (Hs) and/or scale 3 (Hy) OR Scales 1 or 3 appearing in the 2-point code type OR If scale 1 or 3 not the highest elevation, it is ≤ 6 points below the highest elevation
Locke and Thomas (2011)	MMPI-2-RF	PNES-pc ≥ 3 and PNES-a ≥ 3
Wagner et al., (2005)	PAI	NES Indicator
Stroup et al., (2006)	PAI	SOM-C $T \geq 70$ or DEP-P $T \geq 70$
Thompson et al., (2010)	PAI	SOM-C $T \geq 70$
Locke et al., (2011)	PAI	SOM $T \geq 70$
Testa et al., (2011)	PAI	SOM-C $T \geq 67$

Note. MMPI = Minnesota Multiphasic Personality Inventory; Hs = hypochondriasis; Hy = hysteria; Mf = masculinity/femininity; Si = social introversion; D = depression; MMPI-2 RF = MMPI-2 Restructured Form; PNES-pc = psychogenic nonepileptic seizure – physical complaints; PNES-a = psychogenic nonepileptic seizure – attitudes; PAI = Personality Assessment Inventory.

CHAPTER 2. METHODS

2.1 Participants

Data from 1230 patients who underwent a comprehensive evaluation for seizure diagnosis at the Mayo Clinic Arizona were retrospectively analyzed. This sample includes some participants presented in Locke et al., 2011 and Purdom et al., 2012. This study was deemed exempt from IRB review due to using deidentified pre-existing data, and a data sharing agreement was executed between the Mayo Clinic Arizona and the University of Kentucky to share select deidentified demographic and clinical variables and PAI item responses. All individuals in the study were over the age of 18 and completed vEEG monitoring and were administered the PAI during their inpatient stay. The breakdown of diagnoses is as follows: PNES (n = 328), ES (n = 445), PNES+ES (n = 32), Other (n = 142), and Indeterminate (n = 283). Demographic and clinical data collected included gender, age at time of evaluation, level of education, age at seizure onset, number of antiseizure medications (ASMs) at admission, psychiatric history, current psychiatric medications, substance abuse history, sexual abuse history, other abuse history, and other trauma history.

The main decision rule analyses focused on PNES and ES diagnostic groups, as determined by vEEG monitoring. Those with comorbid PNES+ES, other diagnoses (as determined during their seizure workup), or indeterminate diagnoses were excluded. Although the PNES+ES group is certainly of interest when making diagnostic considerations in the EMU, this group was not included in the main analysis due to low power from a small sample size. Individuals with invalid PAI profiles were also excluded from the analyses, with the final sample consisting of PNES (n = 275) and ES (n = 395).

Additional exploratory analyses were conducted with the PNES+ES group (n=32) after exclusion of invalid PAI profiles (n = 28) from the entire sample.

2.2 Measures

The PAI is a 344-item self-report inventory designed to assess personality characteristics and features of psychopathology. Responses are scored on a 4-point scale (False, Slightly True, Mainly True, Very True) to generate 22 non-overlapping scales. These scales include 4 validity scales, 11 clinical scales, 5 treatment consideration scales, and 2 interpersonal scales. The validity scales assess levels of response inconsistency, endorsement of unusual or bizarre statements, a tendency to present oneself in an overly positive way, or a tendency to present oneself in an overly negative way. The clinical scales are based on the diagnostic theory and criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2013), indicating elevated profiles are well-aligned with clinical syndromes such as somatization, anxiety, depression and other mood disorders, schizophrenia and psychotic disorders, substance use disorders, and some personality disorders. Except for the substance use scales, each of the clinical scales is comprised of multiple subscales that offer further detail on a patient's profile. A unique feature of the PAI is the treatment consideration scales that can provide information about additional characteristics that may not be captured in the criteria of a diagnosis but may be relevant when considering interventions. These features include levels of aggression, suicidal ideation, lack of social support, stress, and potential for treatment rejection. The final two scales provide information related to a patient's interaction style with others, specifically in the areas of dominance and warmth.

2.3 Procedure

PAI scale and subscale scores were collected for all PNES and ES patients. Patients who produced invalid profiles related to inconsistency (ICN $T \geq 73$), infrequency (INF $T \geq 75$), positive impression management (PIM $T \geq 68$), or negative impression management (NIM $T \geq 92$) were excluded from further analysis based on standard validity rules (Morey, 1991). One half of the remaining patient profiles in each group were then randomly assigned to the decision rule “development” group (DEV), and the other half were assigned to the decision rule “application” group (APP).

For DEV, mean T scores on each scale were calculated for PNES and ES groups, and independent samples t-tests were used to determine significant differences between scale and subscale scores for each group. For the scales and subscales found to be significantly different with at least one moderate or clinical elevation between the PNES and ES groups, receiver operating characteristic (ROC) curves were created to plot sensitivity and specificity values at each possible cut score. The area under the curve (AUC) served as an interpretation of the scale’s ability to correctly classify patients as having PNES or ES, with higher values suggesting more accurate classification. The ROC curves also indicated the optimal cut scores for each scale to maximize sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). In determining the cut score for each scale that demonstrated the highest diagnostic accuracy, sensitivity and specificity values were prioritized for DEV, as the focus was on assessing the foundations of the PAI as a screening tool for PNES. Predictive power values were later prioritized with APP to assess the clinical utility of the newly developed decision rules. After examining the ability of single scales to differentiate between PNES and ES,

hierarchical regression analyses were used to examine if the combination of multiple scales enhanced diagnostic accuracy by contributing to the explained variance in diagnosis.

After determining decision rules shown to effectively differentiate between PNES and ES groups with DEV, APP served as a novel sample of patients for cross-validation. ROC curve analysis was repeated with APP to examine diagnostic accuracy values at multiple cut scores. Superior diagnostic accuracy was determined by the highest summed PPV and NPV values. Additional analyses were conducted with a subset of patients who appeared to demonstrate mixed results, leading to the development of sequential decision rules.

Finally, exploratory ANOVA analyses were conducted with the PNES+ES patients and the PNES only and ES only patients to examine demographic and clinical differences, as well as to compare PAI profiles among the groups. Factors examined included gender, age at time of evaluation, level of education, age at seizure onset, number of ASMs at admission, psychiatric history, current psychiatric medications, substance abuse history, sexual abuse history, other abuse history, and other trauma history in addition to PAI scale and subscale scores. Given the discrepancy in samples sizes, heterogeneity of variances was assessed for each variable of interest, so the appropriate statistical method could be utilized, with homogenous variances indicating use of the classic ANOVA test and heterogenous variances indicating the use of Welch's ANOVA test.

CHAPTER 3. RESULTS

3.1 Power Analysis

An a priori power analysis was performed to determine the minimum number of participants required to detect a significant difference between groups. A medium effect size of 0.50 (Cohen, 1988) was utilized as the minimum effect of interest for this study, given the objective of determining clinically significant differences in PAI scores between PNES and ES groups (Kraemer et al., 2003). To achieve a power level of .80 ($\alpha = .05$; Cohen's $d = 0.50$, two-sided test), the sample size for each group needed to reach 64, for a total sample of 128 patients per analysis. Thus, it appears this study included a sufficiently large sample for both the decision rule development analysis, as well as the cross-validation analysis.

3.2 PAI Profile Validity

As noted above, PAI profiles were determined to be invalid if they met any of the following criteria: Missing Items $T \geq 18$, INC $T \geq 73$, INF $T \geq 75$, NIM $T \geq 92$, or PIM $T \geq 68$ (Morey, 1991). A total of 103 profiles were found to be invalid (PNES $n = 54$, ES $n = 49$) and were excluded from further analyses. There were no differences in the specific validity scales elevated by individuals with PNES compared to ES, but overall, PNES patients were more likely to generate invalid profiles, $\chi^2(1, N = 773) = 3.96, p < .05$. Overall, individuals with invalid profiles did not differ from individuals with valid profiles on any of the other recorded demographic or clinical variables. The final sample consisted of 670 patients, 275 with PNES and 395 with ES.

3.3 Sample Characteristics

Table 2 provides a summary of the sample by diagnosis. Variables of interest included age at the time of evaluation, level of education, gender, age of seizure onset, total number of ASMs at the time of admission, history of psychiatric diagnosis, current psychiatric medications, history of substance abuse, history of any abuse, history of sexual abuse, and history of other trauma. The PNES group had a significantly higher percentage of females (75.2%) and reported a later age of seizure onset ($M = 36.4$; $SD = 16.0$) than the ES group. The PNES group was also more likely to have a history of psychiatric diagnosis, to be prescribed psychiatric medications, and to have experienced abuse, sexual abuse, and other trauma. There were no differences in reported substance abuse history. On average, the ES group was prescribed more ASMs than the PNES group at the time of admission.

3.4 Randomization of DEV and APP

The total sample of 670 patients was randomly assigned into DEV or APP. The final DEV group consisted of 332 patients, 140 PNES (42.4%) and 192 ES (57.8%), and the final APP group consisted of 338 patients, 135 PNES (39.9%) and 203 ES (60.1%). Comparison of the DEV PNES group to its APP counterpart revealed no significant differences in any demographic or clinical variables, suggesting the equivalence of the two groups. The same was true for both ES groups.

3.5 DEV Performance

Table 3a provides a comparison of PAI scores for DEV across all eleven clinical scales and their subscales, as well as the treatment consideration scales and interpersonal

scales. Given the large number of comparisons, a Bonferroni-adjusted alpha level of .001 was used to determine significance. At this level, significant differences between the PNES and ES groups were observed on the following scales: Somatic Complaints (SOM), Antisocial Features (ANT), Alcohol Problems (ALC), Somatic Complaints – Conversion (SOM-C), Somatic Complaints – Somatization (SOM-S), Anxiety – Physiological (ANX-P), Depression – Physiological (DEP-P), and Antisocial Features – Egocentricity (ANT-E). A summary of the significant results is presented in Table 3b. Although statistically different, scales ANT, ALC, and ANT-E were not considered for further analysis or inclusion in the decision rules because the mean T score differences on these scales did not appear clinically significant. Neither the PNES group nor the ES group exhibited moderate ($T \geq 60$) or clinical ($T \geq 70$) mean elevations on these scales. Notably, the ES group had a higher mean T score on these scales than the PNES group, whereas the PNES group had higher mean T scores on the scales remaining in the analysis.

ROC curves and AUC analysis were used to examine the diagnostic accuracy of the remaining scales showing statistically significant mean group differences. The following criteria were used to interpret the AUC: 0.9-1.0 (excellent), 0.8-0.9 (good), 0.7-0.8 (fair), 0.6-0.7 (poor), 0.5-0.6 (fail; Tape, 2008). SOM-C showed the highest diagnostic accuracy and performed in the fair range (AUC = .77), followed by SOM (AUC = .74) and SOM-S (AUC = .74). DEP-P (AUC = .64) and ANX-P (AUC = .60) performed in the poor range. Performance of each scale at various cut scores is represented in Table 4. Performance of each scale was assessed by summing the sensitivity and specificity values at each cut score, with higher values indicating better performance (Kaivanto, 2008). At this step of the analysis, sensitivity and specificity values were prioritized to evaluate the

foundations of the PAI as a screening tool for PNES relative to the gold standard of vEEG. At the standard clinical cut score of $T \geq 70$, SOM-C outperformed all other scales (SN = 65.0%, SP = 75.5%). In examining all possible cut scores across the scales, the best diagnostic accuracy overall was achieved with SOM-C at $T \geq 75$ (SN = 60.7%, SP = 81.3%).

To analyze the classification accuracy with multiple scales, two separate hierarchical regression analyses were run to avoid issues with multicollinearity. The first analysis consisted of SOM, DEP-P, and ANX-P, and the second consisted of SOM-C, SOM-S, DEP-P, and ANX-P. Each scale was entered into the analysis at a different step, with the order determined by the previously calculated AUC. Results of the regressions are summarized in Table 5. In regression 1, there was no significant increase in predictive power by adding DEP-P or ANX-P to SOM. In regression 2, the addition of SOM-S and ANX-P add significant predictive power over SOM-C alone, although they each only added $< 2\%$ of explained variance. This small change in R^2 is likely due to significant correlations between SOM-C, SOM-S, and ANX-P (SOM-C and SOM-S: $r = .58$, $p < .001$; SOM-C and ANX-P: $r = .49$, $p < .001$; SOM-S and ANX-P: $r = .57$, $p < .001$). Classification accuracy values for combinations of SOM-C, SOM-S, and ANX-P are shown in Table 6. Performance at the standard clinical cut score of $T \geq 70$, as well as the optimal cut scores for each scale determined by the ROC curves are presented. No combination of these scales outperforms SOM-C at a cut score of $T \geq 75$.

3.6 APP Performance

Scale performance with APP was evaluated by summing PPV and NPV values to assess clinical utility. Higher sums indicated better classification accuracy. At this stage of the analysis, PPV and NPV values were prioritized to examine the clinical utility of the PAI in differentiating PNES from ES. Table 7 displays the performance of the statistically significant scales at various cut scores with APP. Results indicated SOM-C demonstrated the highest diagnostic accuracy and performed in the fair range (AUC = .77). SOM (AUC = .73) and SOM-S (AUC = .71) also performed in the fair range. DEP-P (AUC = .68) and ANX-P (AUC = .63) performed in the poor range. Examination of possible cut scores confirms a cut score of $T \geq 75$ on SOM-C outperformed other cut scores (PPV = 67.2%, NPV = 76.1%), as suggested by DEV.

Important to note, however, is within APP, the sensitivity and specificity values of SOM-C at $T \geq 70$ (SN = 68.1%, SP = 75.4%) outperformed those at $T \geq 75$ (SN = 62.2%, SP = 79.8%), suggesting the superior diagnostic accuracy and clinical utility at a firm cut score of $T \geq 75$ could, in part, be a product of the sample. Results indicate that individuals with SOM-C T scores between 70 and 74 may be more difficult to classify with a single scale score than those with scores outside of this range. Additional analyses were conducted with the small subsamples of patients who had SOM-C T scores between 70 and 74 (DEV: n = 17, 6 PNES and 11 ES; APP: n = 17, 8 PNES and 9 ES) to determine if the addition of another scale aided in diagnostic accuracy. Classification accuracy values for DEV and APP with this subsample at multiple cut scores, are presented in Table 8.

For both DEV and APP, SOM-S proved more successful than ANX-P in differentiating this subsample of PNES and ES patients. For DEV, sensitivity and specificity values as

well as predictive power values were maximized at a cut score of $T \geq 75$ on SOM-S. For APP, values indicate that a cut score of $T \geq 65$ on SOM-S performs best in distinguishing PNES from ES in the subsample. Likely contributing to these different results is the substantial difference in PNES base rates between the DEV subsample (35%) and the APP subsample (47%) due to the small number of participants. In determining which cut score to use as a secondary decision rule, efforts were made to balance sensitivity and specificity values to ensure high numbers of both PNES and ES patients will be correctly classified. Additionally, meta-analytic data examining PNES and ES performance on the PAI were considered (Bosch et al., 2020), with findings indicating moderate elevations on SOM-S are suggestive of PNES. For these reasons, a secondary cut score of SOM-S $T \geq 65$ was selected for this subsample. When these sequential decision rules (SOM-C $T \geq 75$ or SOM-C $T = 70-74$ with SOM-S $T \geq 65$) are applied to the entire APP group, diagnostic accuracy and predictive values improve (PPV = 73.2%, NPV = 79.1%) beyond those of using just SOM-C at $T \geq 75$ alone (PPV = 67.2%, NPV = 76.1%).

To again test the performance of these newly developed decision rules, the APP sample was used to calculate classification accuracy values for other decision rules presented in the literature as a source of comparison. The results of these analyses are presented in Table 9. The performance of the sequential decision rules in this study offers superior diagnostic accuracy over other decision rules presented in the literature, with the highest summed sensitivity and specificity, as well as the highest summed predictive power, and highest hit rate.

3.7 Comorbid PNES+ES Diagnoses

Despite the relatively high base rate of comorbid PNES+ES, little research exists on how the results of psychological assessments with this group compare to individuals with PNES only or ES only. Exploratory analyses with a small sample of PNES+ES patients (n = 32) were compared to PNES and ES samples. PAI profile validity of the PNES+ES group was assessed using the same criteria as above, and 4 profiles were removed. The final sample for this group of analyses was as follows: PNES+ES (n = 28), PNES (n = 275), and ES (n = 395).

Regarding demographic and clinical variables, variances for age at the time of evaluation, age of seizure onset, and number of ASMs at the time of admission were heterogenous. Variance for level of education was homogenous. The results of the comparison are presented in Table 10. There was no significant difference in age at time of evaluation among all three groups. Initial results suggested a difference in education level among the groups, but Tukey follow-up did not reveal a significant difference, suggesting a possible trend for PNES+ES patients to have less formal education than PNES or ES patients. Age of seizure onset was significantly older for PNES patients than for PNES+ES patients or ES patients; there was no difference in age of seizure onset between PNES+ES patients and ES patients. PNES+ES patients and ES patients were also similar in their number of ASMs at admission, with PNES patients having less than either group. The PNES+ES group showed no differences in gender, psychiatric history, use of psychiatric medication, history of substance abuse, history of any abuse, history of sexual abuse, or history of other trauma compared to the PNES group. The PNES+ES group had significantly more females (82.1%), were more likely to use psychiatric medication, were

more likely to have a history of any abuse and were more likely to have a history of sexual abuse than ES patients. There were no differences between PNES+ES and ES groups regarding psychiatric history, history of substance abuse, or history of other trauma.

PAI scale mean T scores for the PNES+ES group and comparison to PNES and ES groups are presented in Table 11. A Bonferroni-adjusted alpha of .001 was again used to determine significance, and indicated differences among the groups on SOM, DEP, ANT, ALC, SOM-C, SOM-S, SOM-H, ANX-P, DEP-P, and ANT-E. Post-hoc analyses revealed PNES+ES profiles were not significantly different than PNES profiles on any scales, and PNES+ES patients had significantly higher scores on SOM and SOM-C than ES patients, with scores on SOM-S also nearing statistical significance.

Table 2 Demographic and Clinical Features of PNES and ES Groups

	PNES (n = 275)			ES (n = 395)			χ^2	<i>p</i>
Gender	207 female (75.2%)	68 male (24.7%)		208 female (52.7%)	187 male (47.3%)		35.17	<.001
	<i>M</i>	<i>SD</i>		<i>M</i>	<i>SD</i>		<i>t</i>	<i>p</i>
Age at evaluation	41.8	14.5		42.3	17.3		-0.43	.67
Education	14.3	3.0		14.0	2.4		1.76	.78
Age of onset	36.4	16.0		27.3	20.3		6.47	<.001
ASMs at admission	0.8	1.0		1.7	1.1		-10.62	<.001
	Yes	No	Unk.	Yes	No	Unk.	χ^2	<i>p</i>
Psychiatric history	244	31	0	255	135	5	50.53	<.001
Current psych. meds	159	116	0	133	262	0	38.45	<.001
Substance history	40	235	0	71	324	0	1.38	.24
Any abuse history	143	127	5	83	293	19	70.47	<.001
Sexual abuse history	82	184	9	38	338	19	45.09	<.001
Other trauma	173	102	0	162	232	1	31.48	<.001

Note. PNES = psychogenic nonepileptic seizure; ES = epileptic seizure; χ^2 = chi-square statistic; *p* = probability value; *M* = mean; *SD* = standard deviation; *t* = *t* statistic; ASMs at admission = number of antiseizure medications taken at the time of admission; Unk. = unknown; Current psych. meds = current psychiatric medications.

Table 3a DEV PAI Score Comparison – All Scales

Scale	PNES (n = 140)		ES (n = 192)		<i>t</i>	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
SOM	76.9	12.5	66.4	11.6	7.92	<.001
ANX	60.5	13.0	57.4	12.8	2.10	.04
ARD	57.0	13.5	54.4	12.0	1.87	.06
DEP	63.7	12.4	60.0	13.0	2.59	.01
MAN	49.4	10.3	49.5	9.2	-0.10	.92
PAR	50.4	10.1	49.7	9.4	0.62	.54
SCZ	56.4	11.7	54.4	11.4	1.56	.12
BOR	54.5	10.8	53.1	10.4	1.17	.24
ANT	46.8	7.8	50.2	9.1	-3.50	<.001
ALC	45.5	5.9	48.2	9.2	-3.33	<.001
DRG	48.8	7.2	50.0	9.9	-1.24	.22
AGG	47.0	9.7	48.5	9.6	-1.36	.18
SUI	52.4	12.7	52.0	11.2	0.28	.78
STR	55.0	10.9	53.3	10.0	1.53	.13
NON	49.7	11.7	48.6	9.5	0.93	.36
RXR	48.7	10.3	48.7	9.8	0.00	1.00
DOM	49.6	10.7	49.5	10.1	0.06	.95
WAR	51.0	10.8	50.7	11.3	0.21	.83
SOM-C	78.2	16.5	63.0	13.1	9.04	<.001
SOM-S	69.5	12.2	59.0	12.5	7.69	<.001
SOM-H	73.5	11.7	70.0	11.4	2.72	.01
ANX-C	58.3	12.2	57.0	12.6	1.02	.31
ANX-A	59.0	13.3	56.6	13.8	1.58	.11
ANX-P	61.3	13.3	56.7	11.6	3.40	<.001
ARD-O	53.4	11.0	52.1	11.4	1.10	.27
ARD-P	53.7	11.0	51.7	11.5	1.55	.12
ARD-T	57.7	14.7	55.3	11.8	1.59	.11
DEP-C	58.0	12.6	56.3	13.1	1.13	.26
DEP-A	58.4	13.9	56.8	12.5	1.05	.29
DEP-P	67.2	10.6	61.4	13.0	4.41	<.001
MAN-A	48.7	10.0	47.2	9.7	1.36	.17
MAN-G	50.0	11.0	51.2	11.0	-0.99	.32
MAN-I	50.0	10.5	50.0	10.2	0.00	1.00
PAR-H	51.2	11.2	50.7	10.9	0.41	.68
PAR-P	48.8	10.1	48.6	9.1	0.18	.86
PAR-R	50.6	9.3	49.6	10.0	0.94	.35
SCZ-P	47.7	9.0	46.5	8.0	1.29	.20
SCZ-S	51.7	12.1	51.1	11.2	0.49	.62
SCZ-T	64.8	13.5	61.9	14.9	1.84	.07
BOR-A	54.2	12.3	54.0	11.1	0.15	.88
BOR-I	55.6	10.7	54.1	11.0	1.24	.22
BOR-N	55.1	10.7	52.9	10.6	1.89	.06
BOR-S	48.2	9.2	47.9	9.2	0.36	.72
ANT-A	47.5	8.6	49.7	10.5	-2.10	.04

Table 3a (continued)

ANT-E	45.8	7.0	50.3	8.1	-5.32	<.001
ANT-S	48.5	8.7	50.6	9.9	-1.94	.05
AGG-A	47.1	10.0	48.8	10.7	-1.41	.16
AGG-V	47.0	10.2	48.4	10.1	-1.18	.24
AGG-P	48.4	8.7	49.0	8.1	-0.70	.49

Note. PNES = psychogenic nonepileptic seizure; ES = epileptic seizure; *M* = mean; *SD* = standard deviation; *t* = *t* statistic; *p* = probability value; SOM = somatic complaints; ANX = anxiety; ARD = anxiety-related disorders; DEP = depression; MAN = mania; PAR = paranoia; SCZ = schizophrenia; BOR = borderline features; ANT = antisocial features; ALC = alcohol problems; DRG = drug problems; AGG = aggression; SUI = suicidal ideation; NON = nonsupport; STR = stress; RXR = treatment rejection; DOM = dominance; WRM = warmth; SOM-C = conversion; SOM-S = somatization; SOM-H = health concerns; ANX-C = anxiety cognitive symptoms; ANX-A = anxiety affective symptoms; ANX-P = anxiety physiological symptoms; ARD-O = obsessive-compulsive; ARD-P = phobias; ARD-T = traumatic stress; DEP-C = depression cognitive symptoms; DEP-A = depression affective symptoms; DEP-P = depression physiological symptoms; MAN-A = activity level; MAN-G = grandiosity; MAN-I = irritability; PAR-H = hypervigilance; PAR-P = persecution; PAR-R = resentment; SCZ-P = psychotic experiences; SCZ-S = social detachment; SCZ-T = thought disorder; BOR-A = affective instability; BOR-I = identity problems; BOR-N = negative relationships; BOR-S = self-harm; ANT-A = antisocial behaviors; ANT-E = egocentricity; ANT-S = stimulus seeking; AGG-A = aggressive attitude; AGG-V = verbal aggression; AGG-P = physical aggression.

Table 3b DEV PAI Score Comparison – Significant Scales

Scale	PNES (n = 140)		ES (n = 192)		<i>t</i>	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
SOM	76.9	12.5	66.4	11.6	7.92	<.001
ANT	46.8	7.8	50.2	9.1	-3.50	<.001
ALC	45.5	5.9	48.2	9.2	-3.33	<.001
SOM-C	78.2	16.5	63.0	13.1	9.04	<.001
SOM-S	69.5	12.2	59.0	12.5	7.69	<.001
ANX-P	61.3	13.3	56.7	11.6	3.40	<.001
DEP-P	67.2	10.6	61.4	13.0	4.41	<.001
ANT-E	45.8	7.0	50.3	8.1	-5.32	<.001

Note. PNES = psychogenic nonepileptic seizure; ES = epileptic seizure; *M* = mean; *SD* = standard deviation; *t* = *t* statistic; *p* = probability value; SOM = somatic complaints; ANT = antisocial features; ALC = alcohol problems; SOM-C = conversion; SOM-S = somatization; ANX-P = anxiety physiological symptoms; DEP-P = depression physiological symptoms; ANT-E = egocentricity.

Table 4 DEV Diagnostic Test Performance of Select Scales

Scale	Cut score	AUC	<i>p</i>	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM	60	.74	<.001	.893	.349	1.242	.500	.817	1.317	.578
	65			.821	.474	1.295	.532	.784	1.316	.620
	70*			.757	.630	1.387	.599	.781	1.380	.684
	75			.550	.766	1.316	.631	.700	1.331	.675
	80			.436	.880	1.316	.726	.681	1.407	.693
SOM-C	60	.77	<.001	.857	.438	1.294	.526	.808	1.334	.614
	65			.771	.630	1.401	.603	.791	1.394	.690
	70			.650	.755	1.405	.659	.747	1.406	.711
	75*			.607	.813	1.419	.702	.739	1.441	.726
	80			.457	.885	1.342	.744	.691	1.435	.705
SOM-S	60*	.74	<.001	.736	.609	1.345	.579	.760	1.339	.663
	65			.664	.656	1.320	.585	.728	1.313	.660
	70			.557	.766	1.323	.634	.703	1.337	.678
	75			.393	.885	1.287	.714	.667	1.381	.678
	80			.236	.943	1.179	.750	.628	1.378	.645
ANX-P	60*	.60	.002	.486	.635	1.121	.493	.629	1.122	.572
	65			.357	.755	1.112	.515	.617	1.132	.587
	70			.214	.859	1.073	.526	.600	1.126	.587
	75			.179	.911	1.090	.595	.603	1.198	.602
	80			.107	.964	1.071	.682	.597	1.279	.602
DEP-P	60	.64	<.001	.807	.411	1.218	.500	.745	1.245	.578
	65*			.671	.583	1.254	.540	.709	1.249	.620
	70			.364	.760	1.124	.526	.621	1.147	.593
	75			.236	.849	1.085	.532	.604	1.136	.590
	80			.100	.911	1.011	.452	.581	1.033	.569

Note. AUC = area under the curve; *p* = probability value; SN = sensitivity; SP = specificity; SN+SP = sum of SN and SP values; PPV = positive predictive value; NPV = negative predictive value; PPV+NPV = sum of PPV and NPV values; HR = hit rate; SOM = somatic complaints; SOM-C = conversion; SOM-S = somatization; ANX-P = anxiety physiological symptoms; DEP-P = depression physiological symptoms.

*Optimal cut scores that maximize SN and SP

Table 5 DEV Hierarchical Regressions for Multiple Scales Predicting Diagnosis

Regression 1						
Variable	β	t	R	R^2	ΔR^2	p
Step 1			.40	.16	.16	<.001
SOM	-0.40	-7.91**				
Step 2			.40	.16	.00	.76
SOM	-0.39	-6.48**				
DEP-P	-0.02	-0.31				
Step 3			.41	.17	.01	.13
SOM	-0.44	-6.51**				
DEP-P	-0.05	-0.76				
ANX-P	0.10	1.54				
Regression 2						
Variable	β	t	R	R^2	ΔR^2	p
Step 1			.46	.21	.21	<.001
SOM-C	-0.46	-9.37**				
Step 2			.48	.23	.02	.004
SOM-C	-0.35	-5.74**				
SOM-S	-0.18	-2.87*				
Step 3			.48	.23	.00	.64
SOM-C	-0.35	-5.76**				
SOM-S	-0.19	-2.75*				
DEP-P	0.03	0.48				
Step 4			.50	.25	.02	.009
SOM-C	-0.40	-6.31**				
SOM-S	-0.23	-3.27*				
DEP-P	-0.02	-0.29				
ANX-P	0.17	2.64*				

Note. β = beta coefficient; t = t statistic; R = correlation; R^2 = coefficient of determination; ΔR^2 = change in R^2 ; p = probability value; SOM = somatic complaints; DEP-P = depression physiological symptoms; ANX-P = anxiety physiological symptoms; SOM-C = conversion; SOM-S = somatization.

* $p < .01$, ** $p < .001$

Table 6 DEV Diagnostic Test Performance of Combined Scales

<i>T</i> ≥ 70							
Scale	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM-C	.650	.755	1.405	.659	.747	1.406	.712
SOM-C <i>and</i> SOM-S	.429	.885	1.314	.732	.680	1.412	.693
SOM-C <i>and</i> SOM-S <i>and</i> ANX-P	.171	.932	1.103	.649	.607	1.256	.611
<i>SOM-C T</i> ≥ 75, <i>SOM-S T</i> ≥ 60, <i>ANX-P T</i> ≥ 60							
Scale	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM-C	.607	.812	1.419	.702	.739	1.441	.726
SOM-C <i>and</i> SOM-S	.529	.849	1.378	.718	.712	1.430	.714
SOM-C <i>and</i> SOM-S <i>and</i> ANX-P	.343	.896	1.239	.706	.652	1.358	.663

Note. *T* = *T* score; SN = sensitivity; SP = specificity; SN+SP = sum of SN and SP values; PPV = positive predictive value; NPV = negative predictive value; PPV+NPV = sum of PPV and NPV values, HR = hit rate; SOM-C = conversion; SOM-S = somatization; ANX-P = anxiety physiological symptoms.

Table 7 APP Diagnostic Test Performance of Select Scales

Scale	Cut score	AUC	<i>p</i>	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM	60	.73	<.001	.904	.350	1.254	.480	.845	1.325	.571
	65			.770	.512	1.282	.512	.770	1.282	.615
	70			.644	.709	1.353	.596	.750	1.346	.683
	75			.519	.788	1.307	.619	.711	1.330	.680
	80*			.393	.872	1.265	.671	.683	1.354	.680
SOM-C	60	.77	<.001	.874	.433	1.307	.506	.838	1.344	.609
	65			.778	.635	1.413	.587	.811	1.398	.692
	70			.681	.754	1.435	.648	.781	1.429	.725
	75*			.622	.798	1.420	.672	.761	1.433	.728
	80			.474	.862	1.336	.696	.711	1.407	.707
SOM-S	60	.71	<.001	.681	.626	1.307	.548	.747	1.295	.648
	65			.630	.685	1.315	.570	.735	1.305	.663
	70			.422	.798	1.220	.582	.675	1.257	.648
	75			.304	.867	1.171	.603	.652	1.255	.642
	80*			.193	.946	1.139	.703	.638	1.341	.645
ANX-P	60	.63	<.001	.533	.690	1.223	.533	.690	1.223	.627
	65*			.356	.823	1.179	.571	.657	1.228	.636
	70			.193	.901	1.094	.565	.627	1.192	.618
	75			.163	.926	1.089	.595	.625	1.220	.621
	80			.081	.951	1.032	.524	.609	1.133	.604
DEP-P	60*	.68	<.001	.837	.453	1.290	.504	.807	1.311	.607
	65			.674	.591	1.265	.523	.732	1.255	.624
	70			.407	.778	1.185	.550	.664	1.214	.630
	75			.252	.877	1.129	.576	.638	1.214	.627
	80			.141	.941	1.082	.613	.622	1.235	.621

Note. AUC = area under the curve; *p* = probability value; SN = sensitivity; SP = specificity; SN+SP = sum of SN and SP values; PPV = positive predictive value; NPV = negative predictive value; PPV+NPV = sum of PPV and NPV values; SOM = somatic complaints; SOM-C = conversion; SOM-S = somatization; ANX-P = anxiety physiological symptoms; DEP-P = depression physiological symptoms.

*Optimal cut scores that maximize PPV and NPV

Table 8 SOM-C T = 70-74 Subsample Diagnostic Test Performance of Select Scales

DEV								
Scale	Cut Score	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM-S	60	.667	.545	1.212	.444	.750	1.194	.588
	65	.500	.545	1.045	.375	.667	1.042	.529
	70	.500	.545	1.045	.375	.667	1.042	.529
	75	.333	1.000	1.333	1.000	.733	1.733	.765
	80	.000	1.000	1.000	.000	.647	.647	.647
ANX-P	60	.333	.455	.788	.250	.556	.806	.412
	65	.000	.545	.545	.000	.500	.500	.353
	70	.000	.727	.727	.000	.571	.571	.471
	75	.000	.818	.818	.000	.600	.600	.529
	80	.000	1.000	1.000	.000	.647	.647	.647
APP								
Scale	Cut Score	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
SOM-S	60	.875	.667	1.542	.700	.857	1.557	.764
	65	.750	.889	1.639	.857	.800	1.657	.824
	70	.357	.889	1.246	.750	.615	1.365	.647
	75	.125	.889	1.014	.500	.533	1.033	.529
	80	.125	1.000	1.125	1.000	.563	1.563	.588
ANX-P	60	.625	.556	1.181	.556	.625	1.181	.588
	65	.500	.667	1.167	.571	.600	1.171	.588
	70	.125	.889	1.014	.500	.533	1.033	.529
	75	.125	.889	1.014	.500	.533	1.033	.529
	80	.000	1.000	1.000	.000	.529	0.529	.529

Note. DEV = development group; SN = sensitivity; SP = specificity; SN+SP = sum of SN and SP values; PPV = positive predictive value; NPV = negative predictive value; PPV+NPV = sum of PPV and NPV values, HR = hit rate; SOM-S = somatization; ANX-P = anxiety physiological symptoms; APP = application group.

Table 9 Comparison of PAI Decision Rules Presented in the Literature

Source	Rule	SN	SP	SN+SP	PPV	NPV	PPV+NPV	HR
Current study	SOM-C $T \geq 75$ or SOM-C $T = 70-74$ with SOM-S $T \geq 65$.667	.837	1.504	.732	.791	1.523	.769
Wagner et al., (2005)	NES Indicator	.659	.724	1.383	.613	.761	1.374	.698
Stroup et al., (2006)	SOM-C $T \geq 70$ or DEP-P $T \geq 70$.726	.655	1.381	.583	.782	1.365	.683
Thompson et al., (2010)	SOM-C $T \geq 70$.681	.754	1.435	.648	.781	1.429	.725
Locke et al., (2011)	SOM $T \geq 70$.644	.709	1.353	.596	.750	1.346	.683
Testa et al., (2011)	SOM-C $T \geq 67$.748	.690	1.438	.616	.805	1.421	.713

Note. SN = sensitivity; SP = specificity; SN+SP = sum of SN and SP values; PPV = positive predictive value; NPV = negative predictive value; PPV+NPV = sum of PPV and NPV values, HR = hit rate; SOM-C = conversion; SOM-S = somatization; DEP-P = depression physiological symptoms; SOM = somatic complaints.

Table 10 Demographic and Clinical Features of PNES+ES Group

	PNES+ES (n = 28)		χ^2	<i>p</i>
Gender	23 female (82.1%)	5 male (17.9%)	40.17	<.001
	<i>M</i>	<i>SD</i>	<i>F</i> or Welch ^a	<i>p</i>
Age at evaluation	39.0	14.8	0.65 ^a	.52
Education	13.2	2.7	3.07	.05
Age of onset	20.6	16.2	27.46 ^a	<.001
ASMs at admission	2.1	1.3	62.65 ^a	<.001
	Yes	No	χ^2	<i>p</i>
Psychiatric history	24	4	53.07	<.001
Current psych. meds	16	12	40.35	<.001
Substance history	3	25	2.07	.36
Any abuse history	14	14	73.70	<.001
Sexual abuse history	8	20	47.27	<.001
Other trauma	13	15	31.69	<.001

Note. PNES+ES = comorbid PNES and ES; χ^2 = chi-square statistic; *p* = probability value; *M* = mean; *SD* = standard deviation; *F* = *F* statistic; ASMs at admission = number of antiseizure medications taken at the time of admission; Current psych. meds = current psychiatric medications.

^aWelch statistic used for unequal variance

Table 11 PNES+ES Group PAI Score Comparison

Scale	<i>PNES+ES</i> <i>M (SD)</i>	<i>PNES</i> <i>M (SD)</i>	<i>ES</i> <i>M (SD)</i>	Statistic (F or Welch ^a)	<i>p</i>
SOM	76.7 (12.5)	76.9 (12.5)	66.4 (11.6)	64.50 ^a	<.001
ANX	61.6 (16.1)	60.5 (13.0)	57.4 (12.8)	4.23 ^a	.02
ARD	54.0 (14.5)	57.0 (13.5)	54.4 (12.0)	1.42 ^a	.25
DEP	64.6 (14.8)	63.7 (12.4)	60.0 (13.0)	8.66	<.001
MAN	46.8 (8.8)	49.4 (10.3)	49.5 (9.2)	0.79	.46
PAR	50.2 (12.3)	50.4 (10.1)	49.7 (9.4)	0.06	.94
SCZ	55.7 (12.7)	56.4 (11.7)	54.4 (11.4)	1.21	.30
BOR	54.8 (12.0)	54.5 (10.8)	53.1 (10.4)	0.41	.64
ANT	48.3 (8.0)	46.8 (7.8)	50.2 (9.1)	9.83	<.001
ALC	45.0 (5.3)	45.5 (5.9)	48.2 (9.2)	9.87 ^a	<.001
DRG	48.1 (6.3)	48.8 (7.2)	50.0 (9.9)	2.97	.05
AGG	47.2 (8.5)	47.0 (9.7)	48.5 (9.6)	1.91	.15
SUI	53.9 (11.3)	52.4 (12.7)	52.0 (11.2)	0.51	.60
STR	58.3 (12.4)	55.0 (10.9)	53.3 (10.0)	2.74	.07
NON	50.1 (12.8)	49.7 (11.7)	48.6 (9.5)	0.40	.67
RXR	46.3 (10.4)	48.7 (10.3)	48.7 (9.8)	1.36	.26
DOM	48.1 (9.9)	49.6 (10.7)	49.5 (10.1)	0.79	.46
WAR	54.6 (9.8)	51.0 (10.8)	50.7 (11.3)	1.59	.21
SOM-C	76.0 (18.5)	78.2 (16.5)	63.0 (13.1)	84.91 ^a	<.001
SOM-S	67.3 (12.1)	69.5 (12.2)	58.9 (12.5)	53.21	<.001
SOM-H	76.6 (9.1)	73.5 (11.7)	70.0 (11.4)	11.80	<.001
ANX-C	58.6 (15.6)	58.3 (12.2)	56.9 (12.6)	0.47	.63
ANX-A	60.0 (15.9)	59.0 (13.3)	56.6 (13.8)	2.82	.06
ANX-P	63.3 (14.4)	61.3 (13.3)	56.7 (11.6)	14.82 ^a	<.001
ARD-O	50.7 (13.7)	53.4 (11.0)	52.1 (11.4)	0.40	.67
ARD-P	52.4 (13.7)	53.7 (11.0)	51.7 (11.5)	1.18	.31
ARD-T	55.1 (12.1)	57.7 (14.7)	55.3 (11.8)	1.51 ^a	.23
DEP-C	59.1 (15.2)	58.0 (12.6)	56.3 (13.1)	1.19	.31
DEP-A	59.8 (15.4)	58.4 (13.9)	56.8 (12.5)	1.62 ^a	.21
DEP-P	66.7 (13.6)	67.2 (10.6)	61.4 (13.0)	27.17 ^a	<.001
MAN-A	48.3 (9.4)	48.7 (10.0)	47.2 (9.7)	0.50	.61
MAN-G	46.4 (9.3)	50.0 (11.0)	51.2 (11.0)	2.03	.13
MAN-I	48.3 (10.6)	50.0 (10.5)	50.0 (10.2)	0.39	.68
PAR-H	50.8 (13.3)	51.2 (11.2)	50.7 (10.9)	0.26 ^a	.77
PAR-P	50.6 (10.3)	48.8 (10.1)	48.6 (9.1)	0.49	.61
PAR-R	49.1 (12.7)	50.6 (9.3)	49.6 (10.0)	0.13	.88
SCZ-P	45.1 (9.0)	47.7 (9.0)	46.5 (8.0)	1.02	.36
SCZ-S	49.7 (12.1)	51.7 (12.1)	51.1 (11.2)	0.29	.75
SCZ-T	67.6 (15.0)	64.8 (13.5)	61.9 (14.9)	5.54	.004
BOR-A	54.8 (11.4)	54.2 (12.3)	54.0 (11.1)	0.54	.58
BOR-I	55.9 (13.2)	55.6 (10.7)	54.1 (11.0)	0.93	.40
BOR-N	55.8 (13.1)	55.1 (10.7)	52.9 (10.6)	1.15	.32
BOR-S	47.6 (7.9)	48.2 (9.2)	47.9 (9.2)	0.09	.91
ANT-A	48.4 (7.1)	47.5 (8.6)	49.7 (10.5)	3.24 ^a	.04

Table 11 (continued)

ANT-E	47.9 (9.1)	45.8 (7.0)	50.3 (8.1)	17.93 ^a	<.001
ANT-S	49.6 (8.6)	48.5 (8.7)	50.6 (9.9)	4.20	.02
AGG-A	48.1 (10.9)	47.1 (10.0)	48.8 (10.7)	3.27	.04
AGG-V	46.9 (9.3)	47.0 (10.2)	48.4 (10.1)	0.62	.53
AGG-P	47.8 (7.5)	48.4 (8.7)	49.0 (8.1)	1.00	.37

Note. PNES+ES = comorbid PNES and ES; PNES = psychogenic nonepileptic seizures; ES = epileptic seizures; *M* = mean; *SD* = standard deviation; *p* = probability value; SOM = somatic complaints; ANX = anxiety; ARD = anxiety-related disorders; DEP = depression; MAN = mania; PAR = paranoia; SCZ = schizophrenia; BOR = borderline features; ANT = antisocial features; ALC = alcohol problems; DRG = drug problems; AGG = aggression; SUI = suicidal ideation; NON = nonsupport; STR = stress; RXR = treatment rejection; DOM = dominance; WRM = warmth; SOM-C = conversion; SOM-S = somatization; SOM-H = health concerns; ANX-C = anxiety cognitive symptoms; ANX-A = anxiety affective symptoms; ANX-P = anxiety physiological symptoms; ARD-O = obsessive-compulsive; ARD-P = phobias; ARD-T = traumatic stress; DEP-C = depression cognitive symptoms; DEP-A = depression affective symptoms; DEP-P = depression physiological symptoms; MAN-A = activity level; MAN-G = grandiosity; MAN-I = irritability; PAR-H = hypervigilance; PAR-P = persecution; PAR-R = resentment; SCZ-P = psychotic experiences; SCZ-S = social detachment; SCZ-T = thought disorder; BOR-A = affective instability; BOR-I = identity problems; BOR-N = negative relationships; BOR-S = self-harm; ANT-A = antisocial behaviors; ANT-E = egocentricity; ANT-S = stimulus seeking; AGG-A = aggressive attitude; AGG-V = verbal aggression; AGG-P = physical aggression.

^aWelch statistic used for unequal variance

CHAPTER 4. DISCUSSION

4.1 Decision Rules

A diagnosis of PNES can be very difficult to distinguish from ES given the similarities in clinical presentation and the relatively high prevalence of PNES in the population. Monitoring with vEEG is the gold standard for diagnosis, but this specialized approach is costly and not widely available, leading to significant diagnostic delays for individuals with PNES. For this reason, clinicians could benefit from a screening method to assist in determining which patients may benefit most from vEEG monitoring. The purpose of this cross-validation study was to derive a set of decision rules on the PAI that could best differentiate PNES patients from ES patients.

The results of this study support other published findings demonstrating significant differences in reported aspects of somatic complaints, depression, and anxiety in PNES and ES groups. Specifically, individuals with PNES are more likely to report a higher level of unusual sensory or motor dysfunction (SOM-C), as well as generic physical complaints (SOM-S), whereas an elevated level of health concerns (SOM-H) appears likely for either group. Additionally, individuals with PNES are more likely to report higher levels of physiological symptoms of depression (DEP-P) and anxiety (ANX-P) compared to individuals with ES. When considering the comprehensive PAI profile of 49 clinical, treatment consideration, and interpersonal scales and subscales, SOM-C had the strongest ability to distinguish between the two groups and demonstrated the highest diagnostic accuracy. At a cut score of $T \geq 75$, SOM-C outperformed all other individual scales in its ability to correctly differentiate PNES patients from ES patients. These findings are similar to other previously published decision rules as they highlight the importance of SOM,

ANX, and DEP scales and subscales in differentiating PNES and ES; however, this study demonstrated that a higher cut score on SOM-C resulted in better classification accuracy when compared to other single scale decision rules.

Despite the complex assortment of symptoms often reported by individuals with PNES, decision rules broadly incorporating multiple scales did not surpass the diagnostic accuracy of SOM-C alone. However, the results indicated that individuals with slight clinical elevations on SOM-C ($T = 70-74$) were not reliably categorized as having PNES or ES, suggesting both groups may be likely to report some degree of unusual somatic symptoms. For this subsample of individuals, application of a secondary decision rule (SOM-S $T \geq 65$) improved diagnostic accuracy, indicating that individuals with PNES may be more likely to also report a moderate degree of diffuse physical complaints than individuals with ES. The systematic method with which these rules were derived offers meaningful clinical information about patients with PNES and the symptoms that appear to be most salient to them.

In previous studies, the PAI has been used in numerous ways in an attempt to distinguish PNES from ES. Some decision rules focus on a single scale of interest in making the distinction, while other published decision rules utilize multiple scales or complex formulas to assist in diagnosis. The classification accuracy of many of these rules is variable, with attempts at replication generating inconsistent results. Findings of these studies could be discrepant based on characteristics of the study samples, as well as inconsistent research methodology between studies. The comprehensive nature of the current study allowed for a cross-validation approach consisting of development and replication of the new decision rules, while also allowing for comparison of performance

with the previously established decision rules. This thorough examination of various PAI decision rules demonstrated that the newly formed sequential decision rules outperformed other previously published rules, exhibiting higher combined sensitivity and specificity, higher combined predictive power, and a higher hit rate. Importantly, the new decision rules are also easy to implement clinically and can quickly be applied by looking at the PAI score report.

4.2 Comorbid PNES+ES Diagnoses

Additional exploratory analyses were conducted comparing individuals with PNES+ES to patients with PNES only or ES only, given the high prevalence of comorbidity. Typically, individuals with PNES+ES are excluded from research on PNES to make groups more homogenous, but this rigid research practice limits the generalizability of findings and is responsible for a lack of research in this area. Although some demographic and clinical data from the PNES+ES group align in an expected way with the ES group (earlier age of seizure onset and more ASMs at the time of admission than the PNES group), other variables of more psychological interest do not align clearly with either group. For example, individuals with PNES were more likely to have reported experiencing trauma than individuals with ES; however, there was no significant difference in report of trauma between PNES+ES and either of the other groups. Findings suggest that individuals with PNES+ES display some characteristics typical of both groups, and there is no clear demographic or clinical profile that sets these individuals apart. Examination of PAI profiles with the PNES+ES group showed a slightly clearer alignment of scores, with no significant differences between PNES and PNES+ES on any scales, and the PNES+ES group scoring significantly higher than the ES group on SOM and SOM-C.

Scores between these groups were nearing significance on SOM-S. These scales appear to be an appropriate focus for distinguishing individuals with PNES and individuals with comorbid PNES+ES from those with ES only.

4.3 Strengths and Limitations

One of the main strengths of this study was the exceptionally large clinical sample. Inclusion of so many patients allows for improved confidence in the findings and increased generalizability of the results. The second main strength of this study was the cross-validation methodology to develop and test the decision rules. A significant benefit of cross-validation studies is the avoidance of overfitting data to a particular sample, as well as built-in replication of findings. The variability in results across other studies testing PAI decision rules for the differentiation of PNES and ES suggests the cross-validation model has a significant advantage. A final strength of this study is the clinical applicability of the results. The newly developed decision rules are objective and easy to implement in clinical practice, and they require no more effort than reviewing the PAI score report. This feature is in contrast to decision rules in other areas that may rely on complex regression formulas or ambiguous clinical information to arrive at a decision.

One limitation of the study was the small size of the subsample used to create the secondary decision rule of SOM-C $T = 70-74$ with SOM-S $T \geq 65$. Results of the main analyses with DEV and APP suggested that individuals with slight clinical elevations on SOM-C may be more difficult to categorize with a single scale score, and the inclusion of a secondary scale could enhance clinical utility. Although the varying base rates from the small subsamples led to inconsistent results between DEV and APP at this step of the

analysis, selection of a cut score at $T \geq 65$ for SOM-S was well-supported by meta-analytic results showing a large effect for SOM-S between PNES and ES groups, with PNES patients more likely to demonstrate moderate elevations on this scale (Bosch et al., 2020). The selection of this cut score is further supported by the overall enhanced diagnostic accuracy when applied to APP, and the exceptional performance of the sequential decision rules when compared to other previously published rules.

Another possible limitation of the study was the exclusion of individuals with comorbid PNES+ES from the main analyses. Estimates of comorbidity in this population range significantly (8% - 60%; Kutlubaev et al., 2018), and vEEG-confirmed diagnosis can be especially complicated because it requires multiple types of events to be captured on EEG during the hospital admission. The difficulty of obtaining a confirmed diagnosis can make it challenging to recruit a sufficiently large PNES+ES sample for research. For context, out of the 1230 individuals who underwent vEEG monitoring and were considered for inclusion in this study, only 32 (2.6%) had vEEG confirmed PNES+ES. Due to the lack of research with this population, it is unclear to what extent individuals with dual diagnosis are similar to or different from individuals with PNES or ES only; however, the exploratory analyses with the PNES+ES group in this study offer some meaningful initial insights about demographic and clinical differences, as well as PAI score profile differences. The results suggest a continued focus on the SOM and SOM-C scales may offer the best differentiation between PNES and PNES+ES from ES only. More research is needed to determine the best method for distinguishing PNES+ES from PNES only, so individuals with dual diagnosis are not mistaken for PNES only and continue to receive appropriate treatment for their ES, as well.

Another potential limitation of the study is related to nonequivalent demographic characteristics of the PNES and ES groups. Quality appraisal criteria established for evaluating research on individuals with PNES (Brown & Reuber, 2016) suggested comparison groups should be comparable in terms of age (≤ 5 years different) and gender ($\leq 10\%$ difference in number of females). In this sample, the ages of both groups are equivalent, but the total percentage of females with PNES (75.3%) far surpasses the percentage of females with ES (52.7%). It is possible comparisons between the two groups could be confounded by demographic variables that may influence ratings on the PAI; however, the gender difference exhibited in these samples is representative of gender difference between the PNES and ES populations that have been well-documented in the literature (Asadi-Pooya & Sperling, 2015). As such, the findings of this study may better generalize to the PNES population due to the nonequivalent groups.

A final possible limitation also stems from the aforementioned quality appraisal criteria, which recommended inclusion of a procedure that distinguished possible PNES events from anxiety attacks. Although important to differentiate PNES from other diagnostic confounds, the degree to which PNES can be reliably separated from anxiety is unclear. Some researchers focus on impairment in consciousness during paroxysmal events as a key feature distinguishing PNES from anxiety (Alper et al., 1995; Brown & Reuber, 2016), whereas others consider atypical expressions of anxiety and panic to be a common etiology of PNES (Alsaadi & Shahrour, 2015). The recent meta-analysis for which these quality appraisal criteria were created (Brown & Reuber, 2016) found that only 42 out of 155 (27.1%) studies included procedures to distinguish PNES from an anxiety disorder. These findings suggest it is not a common practice in the field of PNES research

to explicitly differentiate the diagnosis from anxiety, and further research is needed to establish whether anxiety should be considered a possible key feature of PNES or a completely independent diagnosis. In the current study, physiological symptoms of anxiety (ANX-P) were found to be a noteworthy differentiator between PNES and ES, indicating anxiety is frequently co-occurring with PNES and likely should not be used as a rule-out for the diagnosis.

4.4 Conclusions and Future Directions

Overall, the results of this study demonstrate that the PAI can serve as an effective screener to differentiate individuals with PNES from those with ES. The highest diagnostic accuracy was achieved with the following decision rules being indicative of PNES: SOM-C T ≥ 75 or SOM-C T = 70-74 with SOM-S T ≥ 65 . Application of these decision rules also showed superior classification accuracy over other PAI decision rules presented in the literature. Utilization of these rules by clinicians can provide support for a diagnosis of PNES and inform appropriate next steps in the diagnostic and treatment planning process.

Exploratory analyses with comorbid PNES+ES patients did not suggest a demographic or clinical profile that clearly aligned with PNES only or ES only patients; instead, individuals with comorbid PNES+ES are likely to exhibit characteristics typical of both groups. The PAI profile generated by PNES+ES patients was not statistically different than that of PNES only patients; however, they exhibited significant differences from ES only patients on SOM and SOM-C, with performance on SOM-S nearing significance. More research is needed with PNES+ES patients, but consideration of SOM

and SOM-C scales on the PAI appears to be an appropriate focus for differentiation from ES patients.

Future research should consider further replication of these findings with additional samples. Although the cross-validation method is advantageous for including built-in replication with a novel sample, the generalizability of these findings will be enhanced with samples from other regions and settings. Additional research should also include individuals with PNES+ES whenever possible, as they are an underrepresented group in research and more information is needed regarding the best approach for their diagnosis. Finally, future research could also investigate the differences between PNES and ES groups on ANT, ALC, and ANT-E scales. Scores on these scales were found to be significantly different from each other, with the ES patients scoring higher than the PNES patients. The lack of moderate or clinical elevations on these scales for either group put this topic outside the scope of this study, but the score differences suggest there may be underlying differences between the groups related to antisocial features and alcohol use that should be further examined.

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Thesis Title: The Wechsler Memory Scale - Fourth Edition
Flexible Approach: An Exploratory Analysis of Convergent
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- 2010 B.S., Psychology
Minor in Spanish
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PROFESSIONAL POSITIONS HELD

- 01/2019 – Present **Neuropsychology Assessment and Neurocognitive Disorders
Research**, Neurology Department, Kentucky Neuroscience
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- 10/2020 – 06/2021 **Blood And Clot Thrombectomy Registry And Collaboration
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- 09/2018 – 06/2021 **Hyperbaric Oxygen Brain Injury Treatment (HOBIT) Trial**
Glasgow Outcome Scale (GOSE) Assessor
Lexington, KY
- 08/2020 – 06/2021 **Eastern State Hospital**
Graduate Student Therapist
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- 07/2019 – 04/2020 **Kentucky Neuroscience Institute, Department of Neurology**
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- 08/2016 – 08/2019 **Jesse G. Harris Psychological Services Center**
Graduate Student Therapist
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- 08/2018 – 05/2019 **Norton Neuroscience Institute, Department of Neurosurgery**
Graduate Student Neuropsychology Trainee
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- 04/2018, 04/2019 **The Lexington School: Achievement Testing**
Graduate Student Evaluator
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- 08/2015 – 01/2019 **Neuropsychology Assessment and Malingering Research Lab**
Psychology Department, University of Kentucky
Lexington, KY
- 07/2017 – 06/2018 **Cardinal Hill Rehabilitation Hospital**
Graduate Student Therapist
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- 09/2017 – 03/2018 **Coping Skills for Mild Traumatic Brain Injury**
Group Creator/Leader
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- 08/2016 – 05/2017 **Lexington VA Medical Center, Neuropsychology Clinic**
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- 08/2011 – 05/2012 **Brain and Behavior Research Lab**
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- 06/2011 – 12/2011 **Mercy Hospital, Neuropsychology Clinic**
Graduate Student Neuropsychology Trainee
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SCHOLASTIC AND PROFESSIONAL HONORS

- 2019-2020 Research Challenge Trust Fund Fellowship
2016, 2020 Graduate Student Travel Award
2018 International Society of Neurogastronomy Travel Award
2015-2016 Psychology Department Academic Fellowship, University of Kentucky
2011 Second Place in the Educational Psychology Poster Competition,
 Southwestern Psychological Association, San Antonio, TX
2007-2010 Dean's List, Missouri State University

PEER-REVIEWED JOURNAL ARTICLES

- Wallace, E. R., Garcia-Willingham, N. E., Walls, B. D., **Bosch, C. M.**, Balthrop, K. C., & Berry, D. T. R. (2019). A meta-analysis of malingering detection measures for attention-deficit/hyperactivity disorder. *Psychological assessment, 31*(2), 265.

MANUSCRIPTS IN PREPARATION

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BOOK CHAPTERS

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