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Comparison of Psychogenic Movement Disorder Patients with Non-epileptic Seizures and other Hyperkinetic Motor Manifestations: an Integrated Model of Psychosocial and Neuropsychological Functioning

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COMPARISON OF PSYCHOGENIC MOVEMENT DISORDER PATIENTS WITH NON-EPILEPTIC SEIZURES AND OTHER HYPERKINETIC MOTOR MANIFESTATIONS: AN INTEGRATED MODEL OF PSYCHOSOCIAL AND NEUROPSYCHOLOGICAL FUNCTIONING

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Abstract

Psychogenic movement disorders (PMDs) represent a complex and severe form of psychopathology, which even after a century of research remains poorly understood. As previous investigations have neglected to differentiate symptom phenotypes and have approached assessment from the perspective of cognitive dysfunction apparent in PMD patient's "neurological" counterparts, the current study aimed to examine the neurocognitive performance and psychological profiles of PMD patients with hyperkinetic motor manifestations guided by a theoretical lens of frontal lobe pathology and informed by previous neuroimaging studies with this patient population. Subsequent to diagnostic confirmation of their condition by means of video-electroencephalographic monitoring or adherence to Fahn and Williams criteria, 16 patients with psychogenic non-epileptic seizures (PNES) and 16 patients with other hyperkinetic PMDs were administered an abbreviated neuropsychological battery and completed a series of self-report measures assessing psychological functioning. Results of the present study suggest that patients with psychogenic conditions demonstrate specific neurocognitive deficits mediated by frontal lobe structures, and that severity of posttraumatic symptomatology may be predictive of general cognitive impairment in this patient population. When considered in the context of extant neurobiological data, the present findings generally support a cortico-limbic disconnection conceptualization of psychogenic illness. The diagnostic and conceptual implications of these findings are discussed, as well as treatment implications for motor subgroups based on psychological

and neurocognitive discrepancies observed between patients with PNES and other hyperkinetic PMDs. Future investigators are encouraged to adopt a multidisciplinary approach employing recent technological advances and utilizing theoretical models guided by empirically established principles of neurocognitive functioning.

Introduction

Background

The myriad of motor manifestations currently designated as psychogenic movement disorders (PMDs) has traditionally been assumed to represent maladaptive coping mechanisms, which enable traumatically induced psychological and psychosocial distress to be expressed (Alsaadi & Marquez, 2005; Freud, 1910). This interpretation is also supported by research demonstrating affective dysregulation in individuals with PMD (Prigatano & Kirlin, 2009; Scaer, 2001). Although many of these patients also suffer from comorbid affective and neurological conditions that could very well produce abnormal neural activity, some researchers have begun to identify potential neurobiological abnormalities specific to this form of conversion disorder (Friedman & LaFrance, 2010; Nowak & Fink, 2009; Rowe, 2010; Stone et al., 2007). Nevertheless, PMDs continue to be considered medically unexplained symptoms and individuals with these conditions are often referred from the diagnosing physician to clinicians in the psychiatric community for treatment (Barry & Sanborn, 2001; Carton, Thompson, & Duncan, 2003; Strutt, Hill, Scott, Uber-Zak, & Fogel, 2011a).

It is postulated that nearly 3% of all cases evaluated at movement disorder clinics suffer from PMD, and that psychogenic tremor (14-65%), dystonia (24-54%), and myoclonus (0-19%) constitute the greatest frequency of phenotypes encountered (Hallett et al., 2006; Thomas, Vuong, & Jankovic, 2006). However, in addition to PMD manifested by these phenotypes and other abnormal movements, psychogenic patients

may also present with the absence of normal motor function (e.g. paralysis) and/or abnormal sensory phenomenon (Hallett et al., 2006; Rowe, 2010). A variety of other motor manifestations are also frequently encountered in such settings, including psychogenic non-epileptic seizures (PNES), which are paroxysmal episodes resembling epilepsy—most commonly imitating epileptic complex partial episodes—but which occur in the absence of electroencephalographic abnormalities (Hallett et al., 2006). Non-epileptic activity is estimated to occur in 5-20% of outpatients and 10-40% of inpatients referred for epilepsy evaluations (Benbadis & Hauser, 2000; Reuber & Elger, 2003).

Consistent with the traditional Freudian conceptualization of PMDs, the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) classifies these sensorimotor abnormalities as conversion disorders (American Psychiatric Association, 2000). Alternatively, extant experimental data linking dissociative pathology with PMDs has been sufficiently convincing that the International Classification of Diseases, Tenth Revision (ICD-10) now classifies this condition as a dissociative disorder (Brown, Cardena, Nijenhuis, Sar, & Van der Hart, 2007; World Health Organization, 1992). However, despite the lack of consensus regarding the nosology and psychiatric classification of PMDs, both classification systems agree that these are neurological symptoms without a known neurological cause that are not intentionally produced, and which are assumed to have psychological underpinnings (Reuber & Mayor, 2012). While video-electroencephalographic (video-EEG) monitoring remains the "gold standard" for differentiating epileptic seizures (ES) from PNES, such technology is not readily available outside of epilepsy centers and several factors have been noted to limit the utility (e.g., comorbid ES and PNES, failure

to experience an attack during monitoring, some ES patients with EEG activity within normal limits, etc.) and reliability (e.g., moderate inter-rater agreement) of this technology (Reuber & Mayor, 2012). Additionally, despite the establishment of operationalized diagnostic criteria for other PMD subtypes (i.e., Fahn-Williams criteria: Fahn & Williams, 1988; Williams, Ford, & Fahn, 1995; Shill-Gerber criteria: Shill & Gerber, 2006; Gupta and Lang revisions: Gupta & Lang, 2009), the diagnosis of PMD remains a "crisis for neurology" (Hallett, 2006).

Demographic Profile

The age of onset for PMD can range from early childhood to late adulthood with a mean age of approximately forty-four years (Thomas & Jankovic, 2004). PMDs are more common in females than males (Hallett et al., 2006), and some evidence suggests that it may also be more prevalent in certain U.S. ethnic minority groups (3-4%) than the general population (<0.03%; Benbadis & Hauser, 2000). Traumatic experiences have been cited as a potential risk factor for PNES, as such patients tend to report considerable rates of trauma (44-100%) and abuse (23-77%; Arnold & Privitera, 1996; Bowman & Markand, 1996; Fizman, Alves-Leon, Nunes, D'Andrea, & Figueira, 2004; Fleisher et al., 2002; Griffith, Polles, & Griffith, 1998; Synder, Rosenbaum, Rowan, & Strain, 1994). A significantly higher prevalence of posttraumatic stress disorder (PTSD) has also been found in PNES samples than in the general population, along with the associated PTSD symptom of dissociation (Dikel, Fennel, & Gilmore, 2003; Marchetti et al., 2008; Rosenberg, Rosenberg, Williamson, & Wolford, 2000), leading some researchers to suggest that PNES may be a related condition (Fizman et al., 2004).

Although psychiatric comorbidity estimates vary considerably, depression, anxiety and dissociative disorders are concomitant conditions repeatedly associated with PMDs (Araujo Filho & Caboclo, 2007; Fleisher et al., 2002; Hallett et al., 2006; Marchetti et al., 2008). However, a recent study conducted by Van Merode and colleagues (2004) suggests that co-morbid Axis I disorders actually afflict PMD patients prior to the onset of motor manifestations and that chronic anxiety may be a factor conferring risk to the initial development of this condition. Moreover, the dissociative symptoms frequently observed in PMD patients have been shown to correlate significantly with both physical and sexual abuse (Chu, Frey, Ganzel & Matthews, 1999; Draijer & Langeland, 1999), as well as other forms of childhood trauma (e.g., emotional abuse and neglect) frequently reported by PMD patients (Kuterovac-Jagodic, 2003; Roelofs, Keijsers, Hoogduin, Naring, & Moene, 2002; West, Adam, Spreng, & Rose, 2001). Also, consistent with the proposed pattern of dissociation in PMD (i.e. dissociative amnesia), Chu's et al. (1999) research demonstrated that a considerable proportion of participants reporting histories of abuse or trauma had either partial or complete amnesia for those events. However, not all individuals suffering from PMD claim to have experienced abuse or trauma, and while such events may indeed serve an etiological role in the development of this affliction, the relationship appears to be more complex than originally assumed (Edwards & Bhatia, 2012; Stone & Edwards, 2011; Kranick, et al., 2011).

Unfortunately, however, there remains a paucity of research investigating variables that may potentially modify the relationship between trauma histories and dissociative tendencies. While this void may have contributed to the conflicting findings

regarding PMDs, such results could also be the product of inappropriate methodology, the most notable of which includes the heterogeneity of PMD samples utilized in research, along with a tendency for investigators to neglect phenotype distinctions (Rowe, 2010; Stone, Sharpe & Binzer, 2004). The few studies that have compared the features of PMD patients with different motor manifestations have yielded significant results (Abubakr, Kablinger & Caldito, 2003; Crimlisk et al., 1998; Stone et al., 2004). For instance, research conducted by Stone and colleagues (2004) with a sample of consecutive neurological inpatients found that PNES cases tended to occur at a significantly younger age than other motor conversion symptoms and were more likely to be associated with external factors. If, as Stone et al. (2004) suggest, the only feature uniting these disorders is their physical imitation of neurological conditions, then further research with more homogeneous PMD samples and motor phenotype distinctions is warranted.

Psychological & Neuropsychological Features

Given that the diagnosis of PMD remains one of exclusion, differential diagnosis can pose considerable difficulty (Hallett et al., 2006). In a recent survey study of over five hundred members of the Movement Disorder Society conducted by Espay et al. (2009), seventy-one percent of neurologists indicated that they involved psychiatrists or other mental health professionals in the diagnosis of PMD. As such, neuropsychologists are frequently called upon to provide additional data that may be utilized to confirm a psychogenic diagnosis, as well as to assess the functional status of PMD patients. Traditional assessment methods of PMD have included examination of semiological features and personality profiles by neurologists and neuropsychologists, respectively

(Bodde et al., 2011; Hallet et al., 2006). Greater affective distress compared to "organic" neurological conditions has also traditionally been taken to suggest a psychogenic diagnosis (Hallett et al., 2006). However, recent research indicates that depressive symptoms are severe and common in both patients with psychogenic and "organic" neurological conditions, and thus, not effective in differentiating these patient populations (Asmussen, Kirlin, Gale, & Chung, 2009; Hesdorffer, Hauser, Olafsson, Ludvigsson, & Kjartansson, 2006; Strutt, Hill, Scott, Uber-Zak, & Fogel, 2011b).

Alternatively, some evidence suggests that more specific affective symptoms may differentiate PMD patients from their neurological counterparts. For instance, Asmussen et al. (2009) also observed comparable depression severity in PNES and ES patients, yet closer examination of the data revealed significantly higher physiological symptoms of depression in psychogenic patients, especially females. Such findings correspond with the elevated rates of somatization symptoms (Locke et al., 2010; Reuber et al., 2003) and impaired affective perception and expression observed in PMD samples (Prigatano & Kirlin, 2009). Additionally, several studies have identified a greater severity of dissociative symptoms in PMD patients (Brown et al., 2007; Goldstein, Mellers, O'Malley, & Oakley, 2000; Reuber, House, Pukrop, Bauer, & Elger, 2003; Van Merode et al., 2004), as well as chronic anxiety (Strutt et al., 2011b; Van Merode et al., 2004), and general psychopathology (Reuber et al., 2003; Van Merode et al., 2004). Other findings from psychological research with PNES samples suggest that, unlike their ES counterparts, these patients tend to appraise stressful life events as significantly more distressing (Testa et al., 2012), exhibit a greater external control orientation regarding health factors (Goldstein et al., 2000; Strutt et al., 2011b), and utilize passive coping

strategies (e.g., denial, dissociation/mental disengagement, and escape-avoidance) more frequently (Goldstein et al., 2000; Testa et al., 2012). However, the relationship between such symptoms remains a controversy, and some evidence suggests that the psychological disturbances observed in PNES patients may differ in severity from those with other PMDs, as well as vary across semiology-based subtypes of PNES patients (Griffith et al., 2007; Kranick, Ekanayake, Martinez, Ameli, Hallett, & Voon, 2011).

In contrast to the relatively consistent psychological findings in PMD patients, the vast majority of extant data concerning neurocognitive functioning in this patient population is devoid of consensus (Barry & Sanborn, 2001; Carton, Thompson, & Duncan, 2003; Dodrill, 2010), with some studies demonstrating PMD patient cognitive abilities within normal limits (Drane et al., 2006), and others suggesting general neuropsychological impairments in PMD patients equivalent to their neurological counterparts (Binder, Kindermann, Heaton, & Salinsky, 1998; Dodrill, 2008; Drake, 1993; Fargo et al., 2004; Hermann, 1993; McNally et al., 2009; Van Beilen, Griffioen, & Leenders, 2009; Wilkus, Dodrill, & Thompsom, 1984). Some researchers have also identified specific neurocognitive deficits in PMD patients, sometimes in the presence of otherwise intact cognition, including: impairments in fine motor skills (Criswell et al., 2010; Kalogjera-Sackellares & Sackellares, 1999; Sackellares & Sackellares, 2001); bilateral weakness (Sackellares & Sackellares, 2001); poor attention and working memory (Black et al., 2010; Strutt et al., 2011a); executive dysfunction and impaired problem-solving (Black et al., 2010; Kalogjera-Sackellares & Sackellares, 1999); finger agnosia (Binder et al., 1994); impaired lexical versus semantic fluency (Strutt et al.,

2011a); and below average verbal memory and confrontation naming abilities (Prigatano & Kirlin, 2009).

Despite the lack of consensus regarding specific deficits and neurocognitive profiles in PMD patients, altogether the extant data is suggestive of some form of neuropsychological compromise within this patient population. Nevertheless, proposed explanations for the variation observed across studies have included the higher incidence of neurological injury observed in PMD patients (Fiszman et al., 2004; Wilkus & Dodrill, 1989; Wilkus et al., 1984), anxiety (Prigatano & Kirlin, 2009), negative response bias (McNally et al., 2009), pessimistic attributional style (Griffith et al., 2008), and even inadequate effort (Drane et al., 2006). However, while the results of Drane et al.'s (2006) study would seem to challenge the validity of previous neuropsychological findings, a multitude of subsequent studies have been unable to replicate their results, demonstrating instead that PMD patients put forth valid effort on neuropsychological tests (Binder et al., 1998; Criswell et al., 2010; Dodrill, 2008; Preiss, Kramska, & Vojtech, 2012; Strutt et al., 2011a).

Alternatively, some investigators have suggested that the inconsistency in neuropsychological findings for PMD patients across studies may be the product of traditional, yet inappropriate, methodology of indiscriminately grouping all symptom phenotypes together in statistical analyses (Hill & Gale, 2011; Stone, Sharpe & Binzer, 2004). Thus, it may be the case that examination of general profiles in all PMD patients has potentially obscured specific differences between subgroups, which may actually be more homogeneous with respect to neuropsychological functioning as well as underlying psychopathology. As such, the establishment of unique psychosocial and neurocognitive

profiles within subgroups could potentially improve differential diagnostic accuracy as well as subsequent treatment planning for this patient population, and perhaps, facilitate specification of the etiopathogenesis of this debilitating condition (Bodde et al., 2013; Magaudda et al., 2011).

As previously mentioned, the few studies that have attempted to compare PMD subgroups have yielded significant results. For instance, research conducted by Hopp, Anderson, Krumholz, Gruber-Baldini, & Shulan (2012) suggests that greater gender heterogeneity and different clinical manifestations (e.g., altered consciousness, episodic symptoms, and lateralization) may characterize patients with other PMD phenotypes as compared to those with PNES. Additional differences observed have included a younger age of onset, higher rates of reported trauma and environmental stressors, as well as greater borderline personality features and external control orientation associated with PNES patients than those with other PMD phenotypes (Oto, 2008; Stone et al., 2004). Furthermore, emerging evidence suggests that subtype classification of PNES may have relevant clinical and research implications (Griffith, Smith, Schefft, Szaflarski, & Privitera, 2008), and thus various classification schemas have been proposed (Bodde et al., 2012; Cragar, Berry, Schmitt, & Fakhoury, 2005; Drury, 2000; Griffith et al. 2007; Henry & Drury, 1998; Hill & Gale, 2011; Magaudda et al., 2011; Selwa et al., 2000). Unfortunately, however, such studies have yet to be replicated and a consensus has yet to be reached.

Neuroanatomical Correlates

A recent boom in neuroimaging studies with PMD patients has begun to shift our conceptualization of these motor manifestations from a purely psychological theory to a

broader neurobiological model (Hallett, 2010; Lang & Mula, 2013; Mayor, 2012). For instance, a recent study conducted by Labate and colleagues (2012) provides evidence that several structural brain abnormalities are present in patients with PNES, including abnormal cortical atrophy of the motor (i.e., precentral gyrus) and premotor (i.e., superior frontal gyrus and paracentral gyrus) regions in the right hemisphere, as well as the cerebellum bilaterally. Moreover, a significant inverse relationship was observed between the aforementioned premotor regions and the severity of depressive symptomatology (Labate et al., 2012). Such findings are consistent with a number of functional neuroimaging studies revealing hypoactivation of the primary motor cortex contralateral to the affected limb in PMD patients with both hypokinetic and hyperkinetic motor manifestations (Burgmer et al., 2006; Cojan, Waber, Curruzzo, & Vuilleumier, 2009; De Lange, Roelofs, & Toni, 2007; Marshall, Halligan, Fink, Wade, & Frackowiak, 1997; Schrag et al., 2013; Stone et al., 2007). The majority of neuroimaging studies have also identified functional abnormalities in prefrontal cortical areas (e.g., ventromedial prefrontal cortex [VMPFC], dorsolateral prefrontal cortex [DLPFC], orbitofrontal cortex [OFC]), limbic structures (e.g., amygdala, anterior cingulate cortex [ACC]), and other subcortical regions (e.g., basal ganglia, thalamus), as well as specific abnormalities in functional connectivity between such neuroanatomical regions (Van der Kruijs et al., 2012; Voon et al., 2010a, b).

Although the majority of research in this area has focused on psychogenic paralysis, and the variegated sample composition and methodology of each study make it difficult to provide direct comparisons, altogether the available data seem to implicate abnormal cortico-limbic interactions in PMDs. Thus, it may be the case that both

hyperactivation of limbic areas in response to emotionally arousing stimuli and functional disconnection of motor areas from the inhibitory control of the prefrontal cortex are involved in the production of psychogenic motor phenomena (Labate et al., 2012; Mula, 2013). Additionally, the abnormalities observed in the thalamus and basal ganglia may contribute to the production of psychogenic motor manifestations via striato-thalamo-cortical premotor loops (Vuilleumier et al., 2001), as well as alterations in motor intention or attention (Cummings, 1993; Edwards & Bhatia, 2012; Schrag et al., 2013). Moreover, such functional abnormalities along with aberrations in several cortical regions may contribute to cognitive dysfunction in various domains via disruption of cortico-striatal-thalamo-cortical loops, including executive dysfunction (DLPFC), impaired attention (dorsal ACC), emotion dysregulation (ventral ACC), and impulsivity or compulsivity (OFC; Stahl, 2008).

Neurobiological Markers

Considering that PMDs are conceptualized as "functional" or "stress related" movement disorders, the paucity of research investigating stress-related phenomena in this patient population is striking. However, in addition to neuroimaging data, emerging evidence suggests that PMD patients may have distinguishing neurobiological features previously shown to be associated with abnormal responses to stress (Bakvis et al., 2009; Bakvis et al., 2010; La France, Leaver, Stopa, Papandonatos, & Blum, 2010). For instance, a recent study conducted by Bakvis et al. (2009) found decreased levels of basal heart rate variability in PNES patients, who also demonstrated a positive attentional bias for fear-inducing stimuli (suggesting a hypervigilant state), which was significantly related to self-reported trauma. In a later study, Bakvis et al. (2010) demonstrated

significantly greater basal diurnal cortisol levels in PNES patients compared to healthy controls, which was significantly associated with reports of sexual trauma and independent of the acute occurrence of seizures, current depressive symptoms, medications, and smoking. Additionally, research by La France and colleagues (2010) identified significantly lower levels of plasma brain-derived neurotrophic factor (BDNF) in PNES patients compared to healthy controls.

Thus, extant neurobiological data seem to implicate dysregulation of the hypothalamic-pituitary-adrenal axis (HPA-axis), which, along with limbic system structures, are particularly sensitive to stress exposure during early development. Considering the higher incidence of traumatic episodes and chronic anxiety in PMD patient populations, as well as the greater tendency of these patients to perceive such events as subjectively more distressing, it seems likely that stress-related HPA-axis dysregulation may play a role in the etiopathogenesis of this condition. Hence, it may be the case that PMD patients experience overwhelming or chronic stress during development, leading to stress-sensitized neuronal circuits that may potentially be activated even without exposure to new stressors (Stahl, 2008). The decompensation of these vulnerable circuits may lead to the production of an abnormal stress response not unlike that proposed for anxiety disorders, wherein normal stress hormones are all released, but they all remain persistently elevated rather than recovering rapidly as they would during a normal/adaptive stress response. The persistent elevation of glucocorticoids can produce hippocampal atrophy, changes in gene expression, disinhibition of the HPA-axis, and can increase the risk of subsequent affective disturbances—including the development of anxiety and mood disorders (Stahl, 2008).

Such neurobiological abnormalities could also account for the observed structural and functional neural changes observed in PMD patients.

Moreover, excessive secretion of norepinephrine from the locus coeruleus in response to stress can produce alterations in cognitive functioning (via the prefrontal cortex), symptoms of anxiety, panic attacks and hyperarousal (via the amygdala), as well as motor disturbances such as tremor and changes in sympathetic discharge and parasympathetic tone (via the brainstem; Benarroch, 2009; Stahl, 2008). This increase in norepinephrine output can also inhibit serotonin release via presynaptic alpha 2 heteroreceptors located on the nerve terminals of serotonin neurons. A deficiency in serotonin availability would result in diminished activation of signal transduction cascades initiated by this neurotransmitter, and therefore, decreased production of BDNF, which can lead to a loss of synapses as well as entire neurons via apoptosis. Serotonergic projections from brainstem neurotransmitter centers also exert an inhibitory effect on the amygdala, and thus, loss of inhibitory input to this region could contribute to the chronic anxiety observed in PMD patients (Stahl, 2008). Additionally, dysregulation of the amygdala may be related to the functional abnormalities exhibited in prefrontal regions of PMD patients, as the amygdala has reciprocal connections with the ACC, OFC, hypothalamus, thalamus, hippocampus and several brainstem nuclei, which are all involved in the neurobiology of fear. Finally, changes in both norepinephrine and serotonin may be related to the functional abnormalities of the DLPFC documented in PMD patients, as both of these neurotransmitters are involved in the regulation of a cortico-striatal-thalamo-cortical loop originating in this area of the PFC and are believed

to mediate worry-related symptoms in anxiety disorders, including catastrophic thinking, apprehensive expectation, obsession, and anxious misery (Stahl, 2008).

An additional mechanism of action potentially implicated in anxiety disorders involves deficient GABA and serotonergic input to the amygdala, which in turn, could lead to diffuse glutamate excitotoxicity. If as some researchers suggest (Van Merode et al., 2004), chronic anxiety is indeed a condition conferring risk to the initial development of psychogenic movements, then perhaps the etiopathogenesis involves the destruction of cortical pyramidal neurons via such mechanisms. However, even in the absence of neuronal damage such neurochemical abnormalities would, nevertheless, alter the regulatory input to cortical pyramidal neurons causing them to be "out of tune" (Stahl, 2008). Moreover, such alterations could lead to dysregulation of descending cortical projections, impaired functional connectivity between multiple regions, and ultimately the production of cognitive, affective, and perhaps even motor symptoms as well.

Integration of Previous Research

Altogether, the available data seem to suggest a diffuse pattern of functional and perhaps structural neuronal changes associated with psychogenic movement disorders, and while principle causative factors remain elusive, converging evidence seems to suggest that frontal lobe dysfunction may play a critical role. If this is indeed the case, then given our current understanding of frontal lobe function, PMD patients should exhibit a disruption in attention (dorsal ACC), executive functioning and problem-solving (DLPFC), as well as emotional processing (VMPFC) and affective regulation (ventral ACC).

As such, the purpose of the current study is to compare the neuropsychological performance, psychological profiles, and comorbid affective symptomatology of PMD patients with hyperkinetic motor manifestations. Therefore, statistical analyses will be aimed at (1) testing the validity of the proposed model of frontal lobe pathology and stress-related phenomena in PMD patients, (2) clarifying the relationship between posttraumatic symptomatology, psychopathology, and neuropsychological functioning in this patient population, (3) examining the validity of PMD classification systems proposed by previous researchers, and (4) utilizing such information to elucidate characteristic differences that may be utilized to facilitate and inform differential diagnosis and treatment planning for PMD patients with different clinical presentations.

In contrast to previous neuropsychological research with PMD patients, which has either approached analyses from the theoretical perspective of cognitive functioning in the neurological counterparts of PMD subgroups, or has neglected to employ a theoretical perspective altogether, statistical analyses in the current study will be guided by the aforementioned theory of frontal lobe dysfunction and HPA-axis disinhibition, which the extant data seem to implicate in PMD patients. Considering that both affective distress and frontal lobe dysfunction can produce impairments in attention, working memory, and executive functioning, it is hypothesized that such deficits will be among the most prominent in the current sample. Additionally, given the toxic effect of stress-related hormones on neuroanatomic structures mediating memory, it is expected that PMD patients with more severe anxiety symptoms will demonstrate poorer memory performance than those with minimal anxiety symptoms. Finally, with respect to subgroup differences, it is hypothesized that patients with more dramatic motor

manifestations (i.e., PNES versus other PMDs) will demonstrate greater neuropsychological impairments, exhibit more severe psychopathology, and report either a greater chronicity or severity of traumatic experiences.

Method

Participants

Data collection for the current study was conducted at Baylor College of Medicine (BCM) and St. Luke's Methodist Hospital in Houston, Texas, as well as Martin Neurobehavioral Center (MNC) in Tyler, Texas. Potential participants were identified based on previous neurological evaluations and appropriate medical assessments (i.e., video-EEG monitoring or adherence to Fahn and Williams criteria) establishing a psychogenic diagnosis. Inclusion criteria included: 1) English-speaking adults; 2) both males and females; 3) eighteen to sixty-five years of age. Exclusion criteria included: 1) presence of an underlying organic neurological disorder; 2) current or past psychotic symptoms that could interfere with assessment; 3) substance abuse disorder within the past six months; 4) traumatic brain injury; 5) unstable medical condition or clinically significant abnormal laboratory results; 6) mixed etiologies (e.g., concurrent epilepsy and PNES).

Altogether, 84 PMD patients were identified as potential participants and screened for possible inclusion in the current study. Of those patients screened, 27 were excluded due to age (9), poor English mastery (2), traumatic brain injury (3), cerebrovascular accident (4), multiple sclerosis (1), and other comorbid neurological conditions (8). Additionally, eighteen patients declined the initial invitation to participate in the current study, and seven patients were scheduled to participate but ultimately did not complete the evaluation. The final sample consisted of thirty-two PMD patients.

Each patient's motor manifestations were characterized based on symptoms documented by the neurologist in their electronic medical chart, resulting in the following symptom classifications: 16 PNES (50.0%), 2 dystonia (6.3%), 1 bilateral tremor (3.1%), 1 left-sided tremor (3.1%), 3 right-sided tremor (9.4%), 1 myoclonus (3.1%), 2 gait disturbance (6.3%), 2 bilateral tremor and gait disturbance (6.3%), 1 tic and stereotypies (3.1%), and 3 mixed facial symptoms (e.g., dystonia, tics, orofacial dyskinesia, and blepharospasms; 9.4%).

As ten of these patients had previously completed a comprehensive neuropsychological evaluation at MNC, their data were obtained via archival review. The remaining sample had yet to complete a neuropsychological assessment and were therefore evaluated by study investigators, including one participant tested as an inpatient at St. Luke's and twenty-one participants tested as outpatients at BCM. This assessment is not a routine component of standard clinical care for individuals with these diagnoses, and thus, the current evaluation was offered as a free service. The present study was approved by the Institutional Review Board at BCM as well as the University of Texas at Tyler, and all participants evaluated by study investigators provided informed consent.

Study Design

The current study employed a quasi-experimental design, wherein the following bi-level quasi-independent variables were utilized to define groups for comparison: 1) Motor manifestations (PNES versus other hyperkinetic PMDs); 2) Treatment with psychotropic agents versus those without. Such variables were established through review of medical records and clinical interviews conducted with each patient. These groups provided the basis for comparison among multiple dependent variables, including

symptom characteristics, psychosocial variables, and performance on the outcome measures (*described below*).

Outcome Measures

The following neuropsychological measures were administered and scored according to standardized procedures: Montreal Cognitive Assessment (MoCA: Nasreddine et al., 2005), a screening tool assessing general mental status; Digit Span subtest of the Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV: Wechsler, 2008), a measure of attention and working memory; Wechsler Test of Adult Reading (WTAR: Wechsler, 2001), an estimate of premorbid intellectual functioning; Trail Making Test, Parts A and B (TMT-A and TMT-B: Reitan, 1992), a measure of visuomotor integration and set shifting; Lexical (FAS) and Semantic (Animals) Fluency (Heaton, Miller, Taylor, & Grant, 2004; Reitan & Wolfson, 1985), a measure of speeded retrieval of exemplars from a phonemic and semantic category, respectively; Test of Memory Malingering (TOMM: Tombaugh, 1996) and Rey Fifteen Item Memory Test (Rey-15: Rey, 1964; Lezak, Howieson, & Loring, 2004), two measures of symptom validity; Wisconsin Card Sorting Test, 64 Card Version (WCST-64: Kongs, Thompson, Iverson, & Heaton, 2000), a measure of set-shifting and problem solving ability.

Participants were also asked to complete the following self-report measures: Minnesota Multiphasic Personality Inventory, Second Edition, Restructured Format (MMPI-2-RF: Tellegen & Ben-Porath, 2008), a measure of personality, psychopathology, and affective symptomatology; Beck Depression Inventory, Second Edition (BDI-II: Beck, Steer, & Brown, 1996), a measure of depressive symptoms; Penn State Worry Questionnaire (PSWQ: Meyer, Miller, Metzger, & Borkovec, 1990), a measure of

cognitive symptoms of anxiety; PTSD Checklist (PTSD-C: Weathers, Litz, Herman, Huska, Keane, 1993), a measure of posttraumatic symptomatology; Dissociative Experiences Scale, Second Edition (DES-II: Stockdale, Gridley, Balogh, & Holtgraves, 2002), an assessment of dissociative symptoms; Trauma Symptom Inventory (TSI: Briere, 1995), a screening tool for symptoms of posttraumatic stress; and Emotion Regulation Questionnaire (ERQ: Goss, & John, 2003), a measure of individual differences in emotion regulation strategies.

The units of measurement for each test typically utilized in clinical practice for assessment purposes, were used in the present study to examine group performance. Specifically, raw scores were utilized for measures commonly assessed in relation to raw score cutoffs (i.e., MoCA, TOMM, Rey-15, BDI-II, PSWQ, PTSD-C, ERQ and DES-II), age-adjusted standard scores were used for the WTAR (Wechsler, 2001), and demographically adjusted t-scores were used for the TSI (Briere, 1995), MMPI-2-RF (Tellegen & Ben-Porath, 2008), WAIS-IV Digit Span (Wechsler, 2008), WCST-64 (Kongs et al., 2000), TMT and Lexical and Semantic Fluency (Heaton et al., 2004).

Procedure

Neuropsychological evaluations were conducted under the supervision of licensed clinical neuropsychologists by psychometricians with formal training in the administration and scoring of the aforementioned instruments. Participants were first briefed on the purpose of the study, potential risks and benefits of participating, as well as the voluntary nature of their participation. Verbal feedback was then elicited from patients to address any questions or concerns they may have posed and to ensure comprehension. Written consent was subsequently obtained, followed by a clinical

interview eliciting pertinent demographic, developmental, medical and psychosocial information. Participants were then administered the aforementioned neuropsychological instruments according to the standardized procedures outlined in their respective administration manuals. Following completion of the test battery, participants were asked to complete the aforementioned self-report measures to assess their psychological functioning. While administration time varied based on the patient's functional status, the majority of patients were able to complete the assessment within two to three hours. The administration order and approximate duration of each component of the assessment session is proved in Appendix A. Additional data regarding each participant's current condition, treatment, and medical history were later obtained via review of medical records.

Statistical Analyses

Statistical analyses were conducted via IBM® SPSS version 18.0 for Windows. Pearson's correlations were utilized to examine relationships between variables, while multi-dimensional chi-square tests were used to compare categorical variables between groups, including demographic variables and classifications of performance on the outcome measures. Given the limited size of the current sample, each continuous variable was carefully screened for potential violations of assumptions underlying parametric procedures. Examination of graphical displays (probability-probability plots and histograms), values of skew and kurtosis, as well as the Kolmogorov-Smirnov Test were used to assess normality, while homogeneity of variance was assessed by means of Levene's Test and other graphical depictions. Standard statistical transformations as outlined by Tabachnick & Fidell (2001) were applied to those variables violating the

assumption of normality. Mann-Whitney tests were utilized to examine between-group differences for variables that failed to achieve normalization with statistical transformations, while independent sample t-tests were used to compare all other continuous variables between groups.

As age and education are demographic variables known to significantly influence performance on neuropsychological tests, these two variables were screened for their potential utility as covariates for group comparisons on neurocognitive measures not already corrected for both of these demographic variables (i.e., WTAR and WAIS-IV Digit Span). However, when examined as covariates, age was not significantly related to either outcome measure, and education was only significantly related to WTAR scores. As such, analysis of covariance (ANCOVA) utilizing education as a covariate was used to examine between-group differences in WTAR scores, while all other neurocognitive measures were assessed via independent sample t-tests and Mann-Whitney tests, as outlined above. Additionally, multiple regression was utilized to examine potential predictors of general cognitive impairment for the total sample. The stepwise method was used in this analysis due to the exploratory nature of the present study. Finally, a binary logistic regression analysis was employed to identify potential predictors of group membership (i.e., PNES versus other PMD).

Results

Demographic Comparisons

A detailed comparison between PNES and PMD groups along demographic variables is provided in Table 1. As shown, no significant between-group differences were observed for age at testing, gender, handedness, ethnicity, medical/psychiatric history or current treatment. However, the two groups differed significantly in education, $U = 73.0$, $N_1 = 16$, $N_2 = 16$, $p = 0.03$, with PMD participants reporting a higher level of educational attainment than those in the PNES group. The two groups also differed significantly in marital status, $\chi^2(1) = 8.15$, $p = 0.04$, and functional status, $\chi^2(1) = 6.79$, $p = 0.009$, with more PMD participants being married and employed at the time of testing than their PNES counterparts.

While PMD participants tended to report a later age of symptom onset than members of the PNES group, this difference did not reach significance. Additionally, no significant differences were observed between groups in self-reported factors exacerbating their motor symptoms, which included stress (50.0%), fatigue (21.9%), physical pain (21.9%), strong positive or negative emotions (21.9%), heat exposure (12.5%), and hormonal changes (6.30%). However, a significant between group difference was observed for onset type, $\chi^2(1) = 6.15$, $p = 0.01$, with a greater proportion of PNES patients (75.0%) reporting a sudden onset of their motor symptoms and the majority of PMD participants (68.8%) reporting a gradual symptom onset.

While the proportion of patients receiving psychotherapy and psychotropic medications was nearly equivalent between groups, twice as many PMD as compared to PNES patients were taking an antidepressant at the time of testing. However, no significant differences were observed in depressive symptomatology on the BDI-II, MMPI-2-RF or TSI between patients receiving antidepressant medications (PNES = 31.3%; PMD = 62.5%) and those who were not. Moreover, patients receiving treatment with a benzodiazepine at the time of testing (PNES = 50.0%; PMD = 43.8%) demonstrated no significant differences in anxiety symptomatology on the PSWQ, MMPI-2-RF or TSI than those who were not. However, those being treated with a benzodiazepine demonstrated significantly greater impairment on the TMT-B, $t(30) = 2.33$, $p = 0.03$; Benzodiazepine: $M = 31.0$, $SD = 13.3$; No Benzodiazepine: $M = 42.7$, $SD = 14.9$. Finally, no significant differences were found in somatic symptomatology including pain complaints on the MMPI-2-RF between patients being treated with an opiate agonist (PNES = 43.8%; PMD = 56.3%) and those who were not.

Trauma History

While the majority of participants reported a history of some form of traumatic experience, the frequency of reported sexual abuse was the only traumatic experience found to differ significantly between groups, $\chi^2(1) = 4.57$, $p = 0.03$, PNES = 37.5%, PMD = 6.30%. On the TSI, no significant differences were observed between groups in either mean T-scores or the frequency of clinical scale elevations. Additionally, no significant between-group differences were observed on the PTSD Checklist or DES-II (Table 2). However, while 76.0% of the current sample obtained clinically significant scores on the

PTSD Checklist, only 29.2% and 22.2% of participants produced clinically elevated scores on the DES-II and dissociation subscale of the TSI, respectively.

Although PTSD Checklist scores accounted for 42.6% of the variance in DES-II total scores, the former measure demonstrated a stronger relationship with the absorption, $r = 0.66$, $p < 0.001$, and depersonalization, $r = 0.65$, $p = 0.001$, subscales than the amnesia, $r = 0.41$, $p < 0.05$, subscale of the DES-II. Alternatively, PTSD Checklist scores accounted for 50.2% of the variance in dissociation subscale scores of the TSI, which exhibited a stronger relationship with the absorption, $r = 0.80$, $p < 0.001$, and amnesia, $r = 0.64$, $p = 0.005$, subscales than the depersonalization, $r = 0.49$, $p = 0.04$, subscale of the DES-II.

Assessment of Personality & Psychopathology

PNES and PMD participants did not differ significantly in either affective symptomatology or emotion regulation strategies (Table 3). On the BDI-II, 43.4% of the total sample obtained scores in the minimal range, while 16.7% reported mild and 40.0% moderate to severe depressive symptoms, respectively. Clinical classifications on the PSWQ were as follows: 28.0% low worry, 40.0% moderate worry, and 32.0% high worry. Examination of ERQ subscale scores revealed 64.0% of participants utilize reappraisal over suppression strategies, which was also not found to differ significantly between groups.

On the MMPI-2-RF, no significant between-group differences were observed in mean T-scores, with the exception of the Restructured Clinical (RC) scale 3: Cynicism, $t(24) = 2.34$, $p = 0.03$; PNES: $M = 54.7$, $SD = 8.91$; PMD: $M = 45.6$, $SD = 10.5$. A comparison of the RC scale profiles of PNES versus other PMD participants is depicted

in Figure 1. In terms of the frequencies of clinical scale elevations, a significantly higher proportion of PMD participants produced elevated scores on the Negative Emotionality/Neuroticism scale, $\chi^2(1) = 4.21$, $p = 0.04$, PNES = 6.30%, PMD = 43.8%, while a significantly greater number of PNES patients obtained clinically elevated scores on the Suicidal Ideation scale, $\chi^2(1) = 5.11$, $p = 0.02$, PNES = 18.8%, PMD = 0.0%.

PNES and PMD participants did not differ significantly on the Anxiety scale of the MMPI-2-RF. However, an examination of the differences between participants' with clinically elevated scores on the Anxiety scale versus those within normal limits, indicated that the former group reported significantly greater symptoms of depression, BDI-II: $t(30) = -3.11$, $p = 0.005$, dissociation, DES-II: $t(30) = -2.50$, $p = 0.02$, and posttraumatic symptomatology, PTSD Checklist: $t(30) = -5.76$, $p < 0.001$. These individuals also obtained significantly lower Memory subscores on the MoCA, $t(30) = 3.42$, $p = 0.002$, than those with Anxiety scores within normal limits.

Assessment of Neuropsychological Functioning

All participants obtained valid scores on two measures of suboptimal effort, including the TOMM (PNES: $M = 49.2$, $SD = 1.60$; PMD: $M = 49.9$, $SD = 0.26$) and Rey-15 (PNES: $M = 12.7$, $SD = 0.90$; PMD: $M = 14.4$, $SD = 1.24$), indicating that they put forth valid effort during their neuropsychological assessment (Table 4). While group mean WTAR scores were in the average range, the mean MoCA total scores for both groups were in the impaired range, with approximately 75.0% of PNES and 56.3% of PMD participants demonstrating deficient performance on this measure. Although no significant between-group differences were observed for MoCA total scores, PMD participants significantly outperformed their PNES counterparts on the Attention subscale

of the MoCA, $t(30) = -2.88$, $p = 0.007$. MoCA performance by domain in comparison to the extant normative data is provided for the combined sample in Figure 2.

Mean Digit Span total scores for PNES and PMD participants were in the low average and average range, respectively. However, significantly more PNES (37.5%) than PMD (6.25%) participants obtained Digit Span total scores in the impaired range, $\chi^2(1) = 5.04$, $p = 0.03$. TMT group means varied from low average to mildly impaired and did not differ significantly between groups. While PMD and PNES participants demonstrated comparable semantic fluency performance, their lexical fluency scores differed significantly between groups, $t(30) = -3.44$, $p = 0.002$, with PNES participants evidencing mild to moderate impairment on this measure in comparison to the low average classification of the PMD group's performance. Additionally, both PNES and PMD group means were in the low average range for WCST-64 total and perseverative errors, while PNES patients demonstrated significantly more perseverative responses than PMD participants on this measure, $t(30) = -3.87$, $p = 0.001$. The neuropsychological profile of the combined groups in relation to demographically corrected normative data is provided in Figure 3.

A linear regression analysis employing the stepwise method was utilized to identify potential predictors of general cognitive impairment as evidenced by performance on the MoCA. Only variables demonstrating a significant relationship with the outcome measure at $p < 0.01$ and having a theoretical basis for inclusion were used as independent variables for the analysis (i.e., years of education, BDI-II and PTSD Checklist total raw scores, WCST-64 perseverative errors and MMPI-2-RF Psychoticism scale T-scores). As a result, the first step yielded a significant model, $F(1, 23) = 15.4$, p

= 0.001, with only PTSD Checklist scores identified as a significant predictor and accounting for 38.5% of the variance, Adjusted $R^2 = 0.385$. However, in the second and final step, a significant model emerged, $F(2, 23) = 14.8$, $p < 0.001$, with both years of education, $\beta = -0.46$, $p = 0.007$, and PTSD Checklist scores, $\beta = 0.45$, $p = 0.007$, identified as significant predictors of MoCA total scores. The final model explains 54.6% of the variance in the population, Adjusted $R^2 = 0.546$.

Exploratory Analyses

Given the small sample size of the current study, a conservative approach was adopted in subsequent analyses to identify potential predictors of group membership—utilizing only variables with significant between-group differences at $p < 0.01$. Based on this criterion, two significant models were generated by means of binary logistic regression analyses. The first model included functional status, MoCA Attention subscale scores, WCST-64 perseverative responses and lexical fluency T-scores, with the combination of these four variables significantly predicted group membership, omnibus $\chi^2(4) = 21.8$, $p < 0.001$. The model accounted for between 52.8% and 70.6% of the variance in motor symptom group membership, with 86.2% of the total sample correctly classified. Separately, however, only functional status and WCST-64 perseverative responses were statistically significant predictors ($p < 0.05$). A second model was generated consisting of functional status and WCST-64 perseverative responses. This combination of variables was also found to be statistically significant, omnibus $\chi^2(2) = 20.8$, $p < 0.001$, with both variables identified as significant predictors of group membership ($p < 0.05$). This second model accounted for 47.8 – 63.7% of the variance in group membership, with 75.0% of both groups correctly classified. These findings are

considered preliminary, however, and must be interpreted with caution given the limited sample size.

Discussion

The present study was undertaken to advance the current conceptualization of hyperkinetic psychogenic movement disorders through an examination of psychological and neuropsychological functioning of increasingly homogeneous clinical phenotypic groups. Unlike previous investigations approaching assessment from either an atheoretical perspective or a model of functioning established in neurological disorders with similar clinical presentations, the neurocognitive and psychosocial variables examined in the present study were guided by a theoretical lens of frontal lobe pathology and informed by previous neuroimaging studies with this patient population. While the results of the present study are consistent with previous investigations observing a higher prevalence of affective and posttraumatic symptomatology in this patient population as compared to the general population (Fizman et al., 2004; Hallett et al., 2006), the current findings also suggest that patients with PNES and other PMDs demonstrate specific neurocognitive deficits mediated by frontal lobe structures in the context of average premorbid intellectual functioning and the provision of valid effort during neuropsychological assessment.

Frontal Lobe Pathology in PMD

Considering that both affective distress and frontal lobe dysfunction have been shown to produce impairments in attention, working memory, and executive functioning (Lezak et al., 2004), it was hypothesized that such deficits would be among the most

prominent in the current sample. Indeed, the results of the present study are generally consistent with this hypothesis as the combined sample, including both PNES and other PMD patients, demonstrated moderately impaired performance in the attention and abstraction domains of the MoCA, while their visuospatial/executive scores were low average and their scores in all other domains were in the average range. The mean lexical fluency performance for the total sample fell in the mildly impaired range, while performance on other measures of language functioning were within normal limits. Simple auditory attention as assessed by the WAIS-IV was generally in the low average range, while simple visual attention and tracking skills demonstrated on TMT-A were mildly impaired. On a complex visual attention and tracking task requiring flexibility of thought and motor sequencing ability (i.e., TMT-B), the current sample also demonstrated mildly impaired performance. Finally, on a more complex measure assessing cognitive flexibility, set shifting and problem solving abilities (i.e., WCST-64), the current sample demonstrated performance in the low average range. While clinically significant levels of anxiety and depressive symptoms were observed in both PNES and other PMD participants, measures of affective symptomatology failed to demonstrate utility in predicting neurocognitive performance. Alternatively, both education level and posttraumatic symptoms as assessed by the PTSD-Checklist were identified as significant predictors of MoCA total scores, with lower levels of education and more severe posttraumatic symptoms being associated with greater impairments in general cognitive status (*discussed further below*).

The present findings appear to be consistent with data from previous neuroimaging studies implicating functional abnormalities in several prefrontal cortical

regions (Cojan et al., 2009; DeLange et al., 2010; Marshal et al., 1997; Schrag et al., 2013; Spence et al., 2000; Stone et al., 2007; Voon et al., 2010a, b). Specifically, the poor lexical fluency, problem-solving and response inhibition demonstrated by PMD participants in the current study may be suggestive of abnormal information processing in the DLPFC (Shallice & Burgess, 1991; Stahl, 2008), while the emotional dysregulation characteristic of this patient population and also observed in the present study would seem to implicate VMPFC dysfunction (Cojan et al., 2009; Lezak et al., 2004). Additionally, the attentional disturbances and affective dysregulation demonstrated by PMD patients in the present study may be indicative of abnormal information processing in the dorsal and ventral ACC, respectively (Stahl, 2008). Along with the causal relationship observed between posttraumatic symptomatology and general cognitive impairment, these findings would seem to provide further support for the proposed model of frontal lobe dysfunction in PMD involving abnormal cortico-limbic interactions. In addition, such disruptions in higher-order functions may account for the inconsistent findings demonstrated across neuropsychological investigations with this patient population, as such prefrontal areas subserve functions that facilitate memory (e.g., learning and retrieval strategies, organizational approaches, consolidation, etc.) and play a principal role in inhibiting interference from competing stimuli (Lezak et al., 2004).

Etiological Considerations & Conceptual Implications

Given that neurobiological research has demonstrated elevated cortisol levels in this patient population (Bakvis et al., 2010) and persistently elevated glucocorticoids are known to exert a toxic effect on neuroanatomic structures mediating memory, it was hypothesized that PMD patients with more severe symptoms of anxiety would

demonstrate poorer performance on measures of memory than those with minimal anxiety symptoms. The present findings are consistent with this premise, as participants with clinically elevated scores on the anxiety scale of the MMPI-2-RF demonstrated significantly poorer performance on the memory subscale of the MoCA than patients reporting sub-clinical symptoms of anxiety. As would be expected given the high rates of comorbidity frequently observed between such conditions (Hallett et al., 2006), these patients also reported significantly greater depressive and dissociative symptoms, as well as posttraumatic symptomatology. While the prevalence of trauma symptoms observed in the present study is consistent with previous research (Fizman et al., 2004), the stronger association observed between posttraumatic symptomatology and the absorption and depersonalization subscales of the DES-II in comparison to other dissociative sequelae assessed by this measure, would seem to suggest that a more clinical form of dissociation may characterize this patient population (Putnum et al., 1996).

Altogether, such findings would appear to lend credence to the notion of HPA-axis dysregulation and the conceptualization of this form of psychogenic illness as a type of anxiety disorder, or perhaps more specifically a subtype of PTSD, as has been suggested by previous investigators (Brewin, Andrews, Rose, & Kirk, 1999; Bryant & Harvey, 2000; Fizman et al., 2004). This line of reasoning stems from investigations of the neuronal circuitry underlying responses to script-driven trauma imagery in PTSD patients (Hopper, Frewen, Van der Kolk, & Lanius, 2007; Lanius et al., 2002), in which two distinct subgroups have been identified: one consistent with the DSM definition emphasizing hyperarousal; and another principally characterized by dissociative symptoms. Such research indicates that PTSD patients in the latter subgroup demonstrate

a dissociative response to fear-inducing stimuli, which is neurophysiologically characterized by increased activation of the medial prefrontal cortex and anterior cingulate gyrus along with increased inhibition of amygdala processing and decreased activation of the right inferior frontal cortical region associated with movement inhibition (Aron & Poldrack, 2005; Hopper et al., 2007; Lanius et al., 2002). Moreover, factors similar to those reported by PMD patients in the current study (e.g., stress, strong emotions, etc.) have also been reported to exacerbate the dissociative symptomatology in such patients (Sierra, & Berrios, 1998). Although research with PMD patients has yet to employ such paradigms, the overlap between functional abnormalities observed in this patient population and the proposed dissociative subtype of PTSD provide compelling evidence for a cortico-limbic disconnection conceptualization of psychogenic illness.

Utility of PMD Phenotypic Classifications

With respect to subgroup differences, it is was hypothesized that patients with more dramatic motor manifestations (i.e., PNES versus other PMDs) would demonstrate greater neuropsychological impairments, exhibit more severe psychopathology, and report either a greater chronicity or severity of traumatic experiences. The present findings are partially consistent with these predictions, as patients with other hyperkinetic motor symptoms generally outperformed their PNES counterparts across neuropsychological measures. However, the only neurocognitive discrepancies between motor groups to reach statistical significance were on measures assessing simple auditory attention, lexical fluency and response inhibition. While the psychological profiles of PNES and other PMD participants were generally comparable and clinically depressive symptoms were observed in both groups, a significantly greater prevalence of negative

emotionality and cynicism was observed in patients with other hyperkinetic PMDs. Such findings seem to suggest that the depressive symptoms experienced by these patients may be more accurately characterized as symptoms of increased negative affect, which are theoretically related to serotonergic and noradrenergic dysfunction as opposed to symptoms of decreased positive affect, which entails a greater involvement of dopaminergic dysregulation (Stahl, 2008). Alternatively, a significantly higher prevalence of suicidal ideation was reported by PNES patients, which may be suggestive of more severe abnormalities in serotonergic projections from brainstem raphe nuclei to the amygdala, VMPFC and OFC (Stahl, 2008).

Consistent with previous findings (Stone et al., 2004), participants with other hyperkinetic motor manifestations tended to be older, more educated, and report a later age of symptom onset than PNES patients—although only the educational discrepancy reached statistical significance. While similar rates of self-reported trauma and posttraumatic symptomatology were observed in both groups, PNES patients reported a significantly higher prevalence of sexual abuse as compared to patients with other hyperkinetic PMDs. Moreover, in comparison to the PNES group, a significantly greater number of PMD patients were married and employed at the time of testing. While functional status and response inhibition skills were observed to significantly predict group membership, it may be the case that greater executive deficits characterize PNES versus PMD patients resulting in a diminished capacity to navigate social interactions and appropriately modify behavioral strategies according to environmental feedback.

Treatment Implications

The results of the present study are also consistent with the well-established fact that effective treatment modalities for this patient population are decisively lacking. While the proportion of patients receiving psychotherapy and psychotropic medications was comparable between PNES and other PMD patients, those receiving specific psychotherapeutic agents failed to demonstrate characteristic advantages over those not receiving similar treatment at the time of testing. Specifically, patients taking antidepressant medications reported depressive symptoms comparable to those not receiving such psychotropic agents, while the subset of patients receiving treatment with a benzodiazepine at the time of testing demonstrated anxiety symptoms comparable to those not receiving such treatment. Finally, no marked decrements in somatic symptomatology, including pain complaints, were observed in those being treated with an opiate agonist at the time of testing in comparison to those receiving no such treatment. Although a potential confounding variable may include symptom severity prior to treatment, such findings, nevertheless, raise concerns regarding the efficacy of these psychopharmacological agents with this patient population and would seem to reinforce the need for future research investigating the utility of these pharmacological interventions.

Considering the traditional conceptualization of PMD as a purely "functional" rather than "organic" condition, cognitive behavioral therapy (CBT) is currently the treatment of choice for this patient population (Goldstein et al., 2010; LaFrance et al., 2009). However, given that CBT has been found to produce neurochemical changes in patients with mood and anxiety disorders, the remediation of motor symptoms in PMD as

a result of such therapeutic intervention may simply be due to an improvement in affective symptomatology (DeRubeis, Siegle, & Hollon, 2008). Consistent with such hypotheses are the results yielded from recent psychopharmacological studies employing the antidepressants sertraline (Zoloft) and venlafaxine (Effexor) with PNES patients. Of interest, however, is the fact that despite both drugs producing variable decreases in the frequency of non-epileptic seizures experienced by these patients, only the latter produced significant improvement in PNES patients' affective symptomatology (LaFrance et al., 2010; Pintor et al., 2010). Given that no appreciable reduction in seizure frequency was achieved with the additional psychopharmacological actions of norepinephrine reuptake, such findings would seem to implicate serotonergic abnormalities in the production of psychogenic movements and suggest a viable target for research into more efficacious drug therapies for PMD patients.

Moreover, despite notable improvements in affective functioning and somatic complaints, CBT has failed to demonstrate any appreciable effects on functional status in patients with psychogenic illness (Thomas & Jankovic, 2004). In the present study, however, significant differences were observed between PNES and other PMD patients with respect to functional as well as marital status. When considered in the context of previous research demonstrating greater borderline personality features in this PMD subgroup (Stone et al., 2004), such findings may be suggestive of greater disturbances in interpersonal and communication skills. If this is indeed the case, then perhaps integrating interpersonal/social therapeutic techniques into a CBT framework may be a beneficial approach to treating PNES patients. In addition, only PNES patients in the present study reported clinically significant symptoms of suicidal ideation, suggesting the

need for more aggressive therapeutic interventions with this motor subgroup, which may include recently developed CBT models specifically tailored to reduce suicidal ideation/behavior (Berk, Henriques, Warman, Brown, & Beck, 2004) and perhaps off-label use of psychopharmacological agents with demonstrated efficacy in reducing suicidal ideation in other clinical populations (e.g., lithium).

Alternatively, patients with other hyperkinetic PMDs demonstrated significantly greater negative emotionality and neuroticism, which previous research has found to be associated with general deficits in attentional control including difficulty disengaging attentional resources from negatively valenced stimuli (Bredemeier, Berenbaum, Most, & Simons, 2011). As such, perhaps patients with similar hyperkinetic symptoms may receive particular benefit from therapeutic interventions incorporating biofeedback training, in which patients learn to restructure targeted patterns of brainwaves through the provision of information on their cortical electrical activity. Considering that similar treatment paradigms have been successfully employed in clinical populations with similar symptoms, including posttraumatic stress disorder (Morina et al., 2012; Zotev, Phillips, Young, Drevets, & Bodurka, 2013), major depressive disorder (Choi et al., 2010; Sacchet et al., 2013), fibromyalgia (Kayiran, Dursun, Dursn, Ermutlu, & Karamürsel, 2010), generalized anxiety disorder (Kerson, Sherman, & Kozlowski, 2009), and attention deficit disorder (Thompson & Thompson, 1998), this novel approach to treating psychogenic illness appears promising. However, given the apparently diminished capacity of PNES patients to modify behavioral strategies appropriately in response to environmental cues, these patients may be less likely to benefit from this form of treatment.

Diagnostic Considerations

Over time, the American Psychiatric Association (APA) has come to occupy a position of considerable power and influence within the field of mental health, as their Diagnostic and Statistical Manual of Mental Disorders (DSM) currently guides the direction and scope of clinical practice as well as scientific inquiry (Eriksen & Kress, 2005). During the preparation of this manuscript, the legacy of the APA was extended with the publication of the long anticipated and sharply criticized DSM, Fifth Edition (DSM-V: APA, 2013). According to the APA, their current nosological system is based on clinical utility, is grounded firmly on empirical research, and provides a simple and succinct format which facilitates communication among professionals (APA, 2000; APA, 2013; Jablensky, 2009; Spritzer, 2005). Advocates of this clinical instrument further assert that the descriptive phenomenological approach characterizing the current categorical scheme of the DSM results in highly reliable identification of mental disorders, which enables an accurate comparison of available treatment modalities and identification of psychosocial correlates, and is the only viable classification scheme at present, because there is currently not enough known about the etiology and pathophysiological processes underlying mental disorders to structure the diagnostic compendium according to etiology (APA, 2000; APA, 2013; Spitzer, 2005; Widiger & Mullins-Sweatt, 2007).

However, the validity of the current system is dubious at best, as politics and financial conflicts of interest have and continue to play a stronger role in its development and revision rather than empirical research data (Conner, 2004; Eriksen & Kress, 2005; Frances, 2012). In fact, the DSM appears almost resistant to scientific evidence as it

retains its former classification schema despite advancements in technology enabling the identification of biological substrates associated with a wide variety of mental health conditions, including psychogenic illness (Anderson, Maes, & Berk, 2012; Labate et al., 2012; McHugh, 2005; Tagay, Schlegl, & Senf, 2010). While the APA purportedly considered reclassification of PMD in the new DSM-V, this condition continues to share a diagnostic category with conditions previously referred to as somatization, hypochondriasis, and factitious disorder (APA, 2013)—thereby retaining the inherent implication of either interpretive bias or conscious intent as causal mechanisms underlying PMD. In contrast to such stark resistance to change, research linking PMDs with dissociative pathology has been sufficiently convincing such that the ICD-10 now catalogues PMD as a dissociative type of condition (Brown, Cardena, Nijenhuis, Sar, & Van der Hart, 2007; World Health Organization, 1992). Regardless of the specific organizational scheme advocated, the fact remains that the DSM-V's current classification of PMD is inconsistent with accumulating research demonstrating neurobiological underpinnings of PMD symptomatology. Such dissonance between a requisite clinical instrument and existing empirical data will continue to hinder advancements with this clinical population as the development of increasingly efficacious therapeutic interventions is dependent upon an accurate conceptualization of underlying pathology—neither of which the current nosological system provides. Only through restructuring the classification system to actually conform to empirical data will the field of psychiatry effectively redirect research efforts toward uncovering pathogenic processes, and thereby advance beyond reliance on a mere field guide that may be

reliable but not necessarily valid (Eriksen & Kress, 2005; Zalaquett, Fuerth, Stein, Ivey, & Ivey, 2008).

Limitations & Future Directions

Given the small sample size and the high number of statistical comparisons utilized in the current study, these findings must be interpreted with caution. It must also be acknowledged that the limited size of the current sample prevented further subdivision of the other hyperkinetic PMD group, which may have potentially obscured psychological and neurocognitive differences between more homogeneous motor subgroups (e.g., tremor versus gait disturbances, etc.). Additionally, the sample employed may have consisted of more severe cases of PMD as these individuals were seen at specialized centers, to which they were likely referred by previous healthcare professionals. Future research should strive to investigate a larger sample and provide comparisons between more homogeneous motor subgroups, while also specifically requesting information concerning previous psychogenic diagnoses as well as the frequency and type of previous therapeutic interventions undertaken.

As the development of psychogenic illness is predicated upon an inability to regulate emotions and available data suggests abnormalities in neuronal networks subserving affective regulation, future investigations should also undertake a more comprehensive assessment of the affective expression and emotion regulation strategies employed by PMD patients. Moreover, given extant data implicating abnormalities in serotonergic projections from brainstem neurotransmitter centers to prefrontal cortical and limbic structures, future avenues of research may include controlled drug trials as well as genomic testing to identify potential genotypes for the serotonin transporter and

other receptors that may convey greater risk to decompensation of cortical circuitry upon exposure to environmental stressors. Along those same lines, future research should be undertaken to examine stress hormones, including cortisol levels, and plasma BDNF in relation to abnormal cortico-cortical and cortico-limbic connectivity in PMD patients, as some research suggests that chronic glucocorticoid exposure may lead to the downregulation of serotonergic 1A receptors, which play a critical role in regulating serotonergic neurotransmission, and by extension, the development and maintenance of neural circuits fostered by BDNF synthesis (Savitz, Lucki, & Drevets, 2009; Stahl & Briley, 2004). Finally, longitudinal research tracking the progression of neurobiological markers as well as the observed functional abnormalities in relation to normal age-related atrophy may also be beneficial.

Closing Remarks

In sum, PMD represents a complex and severe form of psychopathology that is currently poorly understood. As a shorter duration of motor symptoms has been associated with a better prognosis (Hallett et al., 2006), early detection and treatment of this condition is required. However, if as has been suggested (Stone et al., 2004), the only feature unifying these forms of psychogenic illness is the imitation of neurological disorders, then further research with mixed samples confounded under the general label of PMD or even "pseudoseizures" will continue to yield inconsistent results and the therapeutic interventions, subsequently developed from a poor understanding of the etiology of such afflictions, will remain less than comprehensive. While "psychogenic movement disorders" has long served as an umbrella term for patients with psychological disturbances in the presence of medically unexplained symptoms, the results of the

current study suggest that PMD patients with different hyperkinetic motor manifestations do not represent a unitary group, but instead, may be characterized by subtle differences that could have important implications for uncovering the etiopathogenesis of this debilitating condition as well as developing increasingly efficacious treatment strategies for this clinical population. As such, future research is needed to determine the origin of such similarities and differences in these forms of psychogenic illness, as well as the utility of such characteristics in delineating appropriate therapeutic interventions for various PMD subgroups.

What is clear, however, is that in order to advance scientific understanding and treatment of psychogenic illness, research endeavors must be released from the shackles of reliable but invalid classification schema that holds fast to outdated conceptualizations derived from a theorist relying solely on his faculties of reason and clinical observation in an era prior to the advent of modern medicine (i.e., neuroimaging, neurobiological and psychopharmacological knowledge). While clinical correlations may provide utility in directing scientific inquiry, it is well-known that correlation is not equivalent to causation. Thus, just as depression is not effectively remediated with the simple application of an appetite stimulant and hypnotic agent, so too is psychogenic illness not effectively treated from a conceptual standpoint limited to clinical correlations. Although the assumption that somatic manifestations of PMD patients function as an expression of underlying psychological distress is supported by research demonstrating that a reduction in motor symptoms is typically accompanied by an amelioration of emotional turmoil, the fact remains that the causative mechanisms underlying this so-called "conversion" process have yet to be identified. Therefore, future investigators working with this

patient population are encouraged to adopt a multi-disciplinary approach employing recent technological advances and utilizing theoretical models guided by empirically established principles of neurocognitive functioning.

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Appendix A: Procedural Outline of Assessment Session

1. Informed Consent *(approximately 5-10 minutes)*
2. Clinical Interview *(approximately 15-30 minutes)*
3. Administration of Neuropsychological Measures *(approximately 45-60 minutes)*
    i. Rey Fifteen Item Memory Test (Rey-15)
    ii. Montreal Cognitive Assessment (MoCA)
    iii. Test of Memory Malingering (TOMM)
    iv. Wechsler Test of Adult Reading (WTAR)
    v. Digit Span subtest of the WAIS-IV
    vi. Lexical (FAS) and Semantic (Animals) Fluency
    vii. Trail Making Test, Parts A and B (TMT-A and -B)
    viii. Wisconsin Card Sorting Test, 64 Card Version (WCST-64)
4. Administration of Self-Report Measures *(approximately 65-90 minutes)*
    i. Beck Depression Inventory, Second Edition (BDI-II)
    ii. Penn State Worry Questionnaire (PSWQ)
    iii. PTSD Checklist (PTSD-C)
    iv. Emotion Regulation Questionnaire (ERQ)
    v. Dissociative Experiences Scale, Second Edition (DES-II)
    vi. Trauma Symptom Inventory (TSI)
    vii. Minnesota Multiphasic Personality Inventory, Second Edition, Restructured Format (MMPI-2-RF)

Appendix B: Tables and Figures

Table 1. Demographic profiles of motor symptom subgroups.

Variable	PNES	PMD	t/χ^2	*p*
Age (years)				
At Time of Testing	39.8 (10.3)	44.6 (14.0)	—	*ns*
At Symptom Onset	36.2 (10.2)	41.3 (12.1)	—	*ns*
Education[a]	12.9 (2.36)	14.3 (2.08)	73.0	0.03
Gender (% female)	15 (93.8%)	15 (93.8%)	—	*ns*
Handedness (% right)	13 (81.3%)	11 (68.8%)	—	*ns*
Ethnicity				
Caucasian	11 (68.8%)	10 (62.5%)	—	*ns*
Hispanic	3 (18.8%)	3 (18.8%)		
African American	2 (12.5%)	3 (18.8%)		
Marital Status				
Married	6 (37.5%)	13 (81.3%)	8.15	0.04
Single	6 (37.5%)	1 (6.30%)		
Widowed	2 (12.5%)	-		
Divorced	2 (12.5%)	2 (12.5%)		
Functional Status				
Employed	2 (12.5%)	9 (56.3%)	6.79	0.009
Disability/Unemployed	14 (87.5%)	7 (43.8%)		
Medical History				
Fibromyalgia	3 (18.8%)	1 (6.30%)	—	*ns*
Migraine headaches	13 (81.3%)	11 (68.8%)		
Head injury w/o LOC	5 (31.3%)	4 (25.0%)		
Psychiatric History				
Mood disorder	6 (37.5%)	6 (37.5%)	—	*ns*
Anxiety-related disorder	7 (43.8%)	6 (37.5%)		
Somatoform disorder	2 (12.5%)	2 (12.5%)		
Dissociative disorder	-	2 (12.5%)		
Personality disorder	1 (6.30%)	-		
Current Treatment				
Antidepressant	5 (31.3%)	10 (62.5%)	—	*ns*
Benzodiazepine	8 (50.0%)	7 (43.8%)		
Anticonvulsant	10 (62.5%)	10 (62.5%)		
Opiate agonist	7 (43.8%)	9 (56.3%)		
Counseling	5 (31.3%)	5 (31.3%)		

Note. Mean (SD) and frequency (%) are provided for each variable.
ns = not statistically significant at $p < 0.05$
[a]Mann-Whitney U test was performed for this variable.

Table 2. Trauma history and posttraumatic symptomatology across groups.

Variable	PNES	PMD	t/χ^2	*p*
DES-II Total	20.3 (16.3)	20.9 (18.1)	—	*ns*
PTSD Checklist Total	43.7 (14.8)	42.4 (16.9)	—	*ns*
Trauma History				
Sexual abuse	6 (37.5%)	1 (6.30%)	4.57	0.03
Physical abuse	2 (12.5%)	5 (31.3%)	—	*ns*
Emotional/Verbal abuse	-	3 (18.8%)	—	*ns*
Neglect/Abandonment	5 (31.3%)	4 (25.0%)	—	*ns*
Natural disaster	4 (25.0%)	5 (31.3%)	—	*ns*
Family Conflict	2 (12.5%)	3 (18.8%)	—	*ns*

Note. Mean (SD) and frequency (%) are provided for each variable.
ns = not statistically significant at $p < 0.05$

Table 3. Affective symptomatology across groups.

Variable	PNES	PMD	*t*	*p*
BDI-II	22.8 (14.4)	16.3 (10.2)	—	*ns*
PSWQ	51.3 (15.1)	48.8 (15.8)	—	*ns*
ERQ				
Suppression	4.02 (1.64)	3.35 (1.37)	—	*ns*
Reappraisal	4.81 (1.01)	4.69 (1.21)	—	*ns*

Note. Mean (SD) are provided for each variable.
ns = not statistically significant at $p < 0.05$

Table 4. Neuropsychological performance across groups.

Variable	PNES	PMD	*t*	*p*
WTAR[a]	95.4 (12.7)	101.5 (14.5)	—	*ns*
MoCA Total Score	23.1 (4.27)	25.0 (3.46)	—	*ns*
Visuospatial/Executive	4.69 (1.25)	5.19 (1.68)	—	*ns*
Naming	3.00 (0.00)	3.00 (0.00)	—	*ns*
Attention	3.94 (1.57)	5.25 (0.93)	-2.88	0.007
Language	1.94 (1.18)	2.37 (0.72)	—	*ns*
Abstraction	1.00 (0.82)	1.19 (0.75)	—	*ns*
Memory	3.56 (1.55)	3.31 (1.99)	—	*ns*
Orientation	5.88 (0.34)	5.94 (0.25)	—	*ns*
Digit Span (WAIS-IV)[b]	41.8 (11.7)	45.1 (6.46)	—	*ns*
TMT				
Trails A	40.9 (15.6)	38.2 (13.4)	—	*ns*
Trails B	38.6 (17.0)	35.8 (13.4)	—	*ns*
Verbal Fluency				
Lexical (FAS)	33.1 (9.26)	43.7 (9.54)	-3.44	0.002
Semantic (Animals)	38.2 (8.87)	42.3 (11.4)	—	*ns*
WCST-64				
Total Errors	42.5 (11.3)	44.4 (9.05)	—	*ns*
Perseverative Errors	41.3 (10.3)	42.6 (7.98)	—	*ns*
Perseverative Responses	55.5 (10.5)	72.8 (14.4)	-3.87	0.001

Note. Mean (SD) t-scores are provided for each variable, with the exception of the WTAR (standard scores) and the MoCA (raw scores).
ns = not statistically significant at $p < 0.05$
[a]ANCOVA with education as a covariate was performed for this variable.
[b]Mann-Whitney U test was performed for this variable.

Figure 1. Comparison of MMPI-2-RF Restructured Clinical (RC) scales between groups.

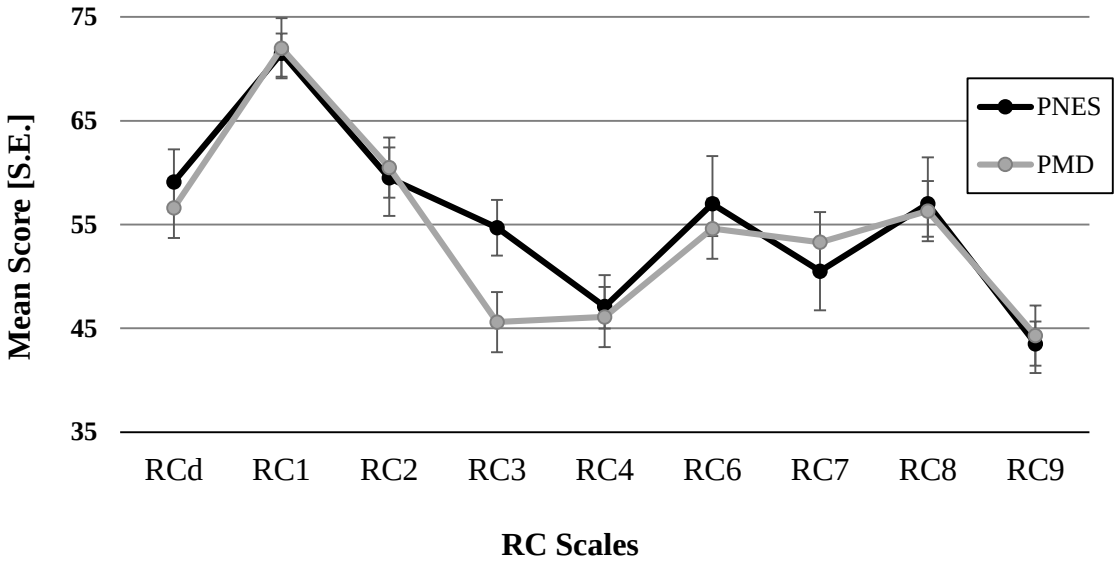

Figure 1. Mean t-scores for PNES and other PMD groups on MMPI-2-RF RC scales. Error bars represent standard errors. Scale abbreviations are as follows: RCd = Demoralization; RC1 = Somatic Complaints; RC2 = Low Positive Emotions; RC3 = Cynicism; RC4 = Antisocial Behavior; RC6 = Ideas of Persecution; RC7 = Dysfunctional Negative Emotions; RC8 = Aberrant Experiences; RC9 = Hypomanic Activation.

Figure 2. MoCA performance by domain for combined groups in relation to normative data.

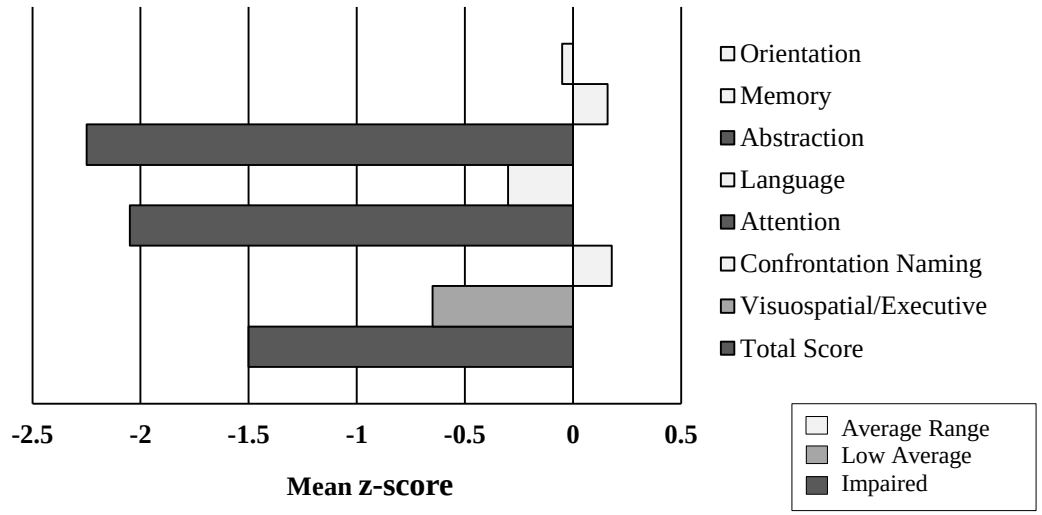

Figure 3. Neuropsychological profile of combined groups in relation to normative data.