SCIENCE INQUEST
Excellent in sharing scientific knowledge

Open Access
Research Article

Volume 3 Issue 1 *Journal of Neurology and Psychiatric Disorders*

Functional Neurological Disorders and Psychogenic Nonepileptic Seizures: Neurologic Disease NOT a 'Functional Etiology'

Carlson CA*

Doctor of Clinical Psychology, Licensed Psychologist, Minnesota Judicial Branch, Minneapolis, Minnesota 55487, USA

***Corresponding Author:** Carlson CA, Doctor of Clinical Psychology, Licensed Psychologist, Minnesota Judicial Branch, Minneapolis, Minnesota 55487, USA, Tel: 651 340-4967, E-mail: carlsonc32@gmail.com

Citation: Carlson CA (2020) Functional Neurological Disorders and Psychogenic Nonepileptic Seizures: Neurologic Disease NOT a 'Functional Etiology'. J Neurol Psychiatr Disord 3(1): 103

Abstract

Neurologic symptoms deemed inconsistent, incongruent, or incompatible with recognized neurologic disease will likely garner a functional (formerly psychogenic) diagnosis, or Conversion Disorder in modern nomenclature. The absence of organic (neurologic) findings is what *distinguishes a functional disorder from ordinary neurologic disease*. The theory underlying the functional diagnosis presumes that in the absence of organic findings, the neurologic symptom must have a psychological etiology and thus, symptom remission rests on psychological intervention. *The most data ever amassed on patients diagnosed with functional disorders has debunked this hypothesis and all its presumptions.* Studies show pervasive neurologic disease in FND and PNES patient populations. In many patients diagnosed with a functional disorder, there is no discernible psychological factor that could be responsible for the 'non-neurologic' symptom. Physical therapies, not psychotherapy, have emerged as highly effective treatments for movement disorders labelled functional. Like a substantial proportion of untreated epilepsy patients, many FND and PNES patients show spontaneous remission of symptoms without any psychotherapy at all. The empirical findings starkly expose the inherent flaws of the diagnostic practice promoted in the FND and PNES literature. The diagnosis of PNES relies on the ictal vEEG, a 'gold standard' that does not capture all epileptiform discharges, particularly those of a frontal lobe origin. The 'positive signs' underlying the diagnosis of functional movement disorders are fallible, have been observed in neurologic conditions, and label *atypical* presentations, and *likely early stage disease*, as inconsistent with *classic* neurologic disorders. Despite compelling evidence these patients indeed suffer from ordinary neurologic disease and epilepsy, FND and PNES investigators remain *highly invested* in the 'functional' etiology and show *confirmation bias* in their interpretation of the empirical data. The ongoing misdiagnosis of neurologic disease and epilepsy as FND and PNES is the *real crisis in neurology.*

Keywords: *Neurological Disorders; Psychogenic Nonepileptic Seizures; Neurologic Disease; Functional Etiology; Atypical; FND and PNES*

Introduction

Functional Neurological Disorders (FND), or Conversion Disorder in modern nomenclature [1], embody a broad phenomenological spectrum encompassing psychogenic nonepileptic seizures (PNES), abnormal movements, gait abnormalities, and sensorimotor deficits [2]. Functional symptoms (aka hysterical, non-organic, psychogenic, and medically unexplained) [3] are common [4], and not only mimic organic disease such as epilepsy [5] and movement disorders [6], but are *just as disabling* as neurologic disease [7-10]. Functional neurological disorders are associated with a poor outcome [11,12], occupy a grey area between neurology and psychiatry [13], and have been deemed a crisis in neurology [14].

Functional Neurological Disorders are a legacy of the clinical sensation known as hysteria. The latter has an enduring history [15] but that does not equate with a uniform or proven entity. In the early 20th century, both the definition and nature of hysteria were subject to animated debates among neurologists and psychiatrists [16]. Jean-Martin Charcot is regarded as a preeminent neurologist who made substantial contributions to the field of neurology, achieving celebrity status in large part due to his demonstrations of hysterical symptoms during his 'Tuesday Lessons' at the Salpêtrière Hospital in the late 19th century [17]. He utilized hypnosis to both create and suppress hysterical symptoms which he concluded were triggered by psychological factors [17]. He is often heralded by FND investigators as an original

authority on hysteria [18] but he was ridiculed by his contemporaries who thought he was being duped by his patients, and later in life, he regretted his work on hysteria and wanted to rewrite his basic tenets on the subject [17].

Arguably the most famous case of 'hysteria' was that of Anna O. In the early 1880's, she was treated by Dr. Josef Breuer, who discovered the 'talking cure' further developed by his protégé' Sigmund Freud [18-20]. During the course of her treatment, in addition to inexplicable physical symptoms, Anna O demonstrated the following psychiatric symptoms and markers: hallucinations; disorganized and incoherent speech; paranoia (accused others of persecuting her, telling her nothing but lies); extended periods of mutism and continuous trance (read catatonia); non-responsive when spoken to (interpreted as hysterical deafness); irritability; periods of high excitement and agitation that involved aggression towards others; decreased need for sleep (went days without sleep); and rapid mood swings wherein she oscillated between exaggerated high spirits and anxious melancholy [19]. Anna O told Dr. Breuer she thought she was going mad, and while he did refer to her as *psychotic* at one point, he attributed all her symptoms to 'hysteria,' a serious psychical disturbance [19]. He hypothesized that a two-week period of muteness was caused by an unidentifiable event wherein Anna felt acutely offended and had determined not to speak about it [19]. He suggested the origins of her intermittent 'deafness' included: "Her father had asked her in vain for wine" and "Her young brother shook her angrily when he caught her listening at the door of the sickroom [20]." Anna O was not cured by hypnosis or talk therapy and in her early to late 20's, she was in and out of sanitariums [19]. *In modern psychiatry, Anna O's presentation would likely garner a diagnosis other than Conversion Disorder* especially if her 'hysterical' symptoms remitted on antipsychotic and mood stabilizing medication.

In 1975, in preparation for an International Symposium on Dystonia, Stanley Fahn and Roswell Eldridge apologized to the "many victims" of dystonia who had been *misdiagnosed with a psychogenic disorder* [20]. They told attendees that psychologically based dystonia was a rare or *non-existent condition* and pointed out how past attempts to manage generalized dystonia along psychiatric lines had *ignored strong evidence for organic causation* [22]. Three years later, Lesser and Fahn [23] reported the "first case of psychogenic dystonia" in a teenager who subsequently admitted to feigning her dystonic symptoms which then disappeared. In 1988, Fahn and Williams [24] published a classification for psychogenic dystonia that included intentional feigning as a variant. The conflation of intentional feigning with unconsciously generated psychogenic symptoms has been a longstanding conceptual problem [25] but Conversion Disorders should be distinguished from malingering and factitious conditions [26] and most FND and PNES investigators make this discrimination [25,27-30].

In 2006, William G. Ondo, M.D., reviewed the book *Psychogenic Movement Disorders* for the New England Journal of Medicine [18]. Dr. Ondo found that Jean-Martin Charcot and other 19th century researchers were cited throughout the book as frequently as current investigators and that this reliance on the past, "attests to the uncertainty of the subject matter [18]." He described the content as "highly theoretical" with much of it "based on the opinions of experts" and concluded that "the evidence-oriented physician might be appalled by the lack of good data [18]."

A great deal of literature on functional disorders has been amassed in recent decades but with very few exceptions [31,32], it *presumes* that the 'psychogenic' or 'functional' diagnostic entity does in fact exist and proceeds from that position. The terms 'psychogenic' and 'functional' are used interchangeably in the clinical literature and refer to neurologic symptoms that most investigators *presume* are primarily psychological in origin, though 'biopsychosocial' models are increasingly popular [33-35].

Methods and Terminology

To generate and support the analysis, a wide sampling of articles on FND and PNES were reviewed. The term 'non-neurologic' refers to neurologic symptoms that have been labelled as having a 'psychogenic' or 'functional' etiology.

Literature Review and Analysis of Findings

Functional (psychogenic) Neurological Disorders are common [36-38], challenging to diagnose given the resemblance of symptoms to recognized neurologic disease [5,36,40], and just as disabling as neurologic disorders like epilepsy [8] and Parkinson's Disease [41].

Functional Neurological Disorders (Conversion Disorder in modern nomenclature) have long been attributed to a psychogenic etiology [42]. *In the absence of organic findings*, investigators *presumed* the neurologic symptom had a psychological origin [29,43-50], and that thorough neurologic and psychiatric histories would confirm the psychogenic source [51]. Patients with functional disorders purportedly have a mysterious capacity to unconsciously transform unspecified psychic distress into often disabling symptoms resembling organic disease [5]. By exposing

and examining the psychological source, the patient can develop the necessary insight resulting in the abandonment of the functional (psychogenic) symptom [5]. The Freudian influence on FND and PNES investigators is unmistakable [5,28,29,50,52-54] and *the validity of the functional diagnostic entity is never questioned*.

Modern neurology is immensely intrigued by functional neurological disorders [55] which is evident by the rapid rate of publications on these conditions [35,37]. Functional Neurological Disorders are now a 'rule in' diagnosis based on validated neurologic signs [40] with examinations "aimed at excluding" neurologic disease [56]. While the etiology of these 'non- neurologic' symptoms remains a mystery [57-60], and the diagnostic challenges in neurology are well known [29,40,61,62], investigators express confidence in the ability to accurately and *rapidly* diagnosis functional disorders [4,40,58,63-65].

Functional symptoms are generally attributed to "psychodynamic causes" [28] and the cornerstone of treatment is psychotherapy [6,66-71]. Without treating and resolving the underlying psychological issues, the "curing" of functional symptoms is considered unlikely [70]. Treatment for Conversion Disorder may require years of intensive counseling to get to the point where patients can safely approach the inner conflicts causing the psychogenic symptom [70].

Investigators have emphasized the importance of identifying psychological factors relevant to the etiology and maintenance of functional symptoms [72]. A problem list with predisposing, precipitating, and perpetuating factors, or "the 3 Ps," is a key component to the clinical formulation [73]. These factors may be remote, occurring during development and early adulthood, and can include psychiatric co-morbidities, maladaptive personality traits, insecure attachment, adverse life events, alexithymia, family dysfunction, intellectual disability, or comorbid medical/ neurological conditions, among other factors [74]. In PNES patients, one common scenario presented is being raised in an alcoholic home leading to a people-pleasing and perfectionistic personality style (predisposing), with a recent motor vehicle accident leading to job loss (precipitating), and ongoing family stressors (perpetuating) [72]. Of course, these psychological factors are also found in people who do not have functional disorders [75] and psychiatric comorbidity is *common* in patients with neurologic disease [76].

Investigators *presumed* that the psychological origin of functional symptoms would be discernible in FND and PNES patients [51] *but epidemiological studies did not support this presumption* [77,78]. The contribution of psychiatric and traumatic contributions have been inconsistent and mixed [75]. The conversion hypothesis, that intrapsychic distress is unconsciously converted into somatic symptoms, is unproven [34]. "Childhood trauma, recent negative life events, depression, and anxiety are all more common in patients with functional neurological symptoms than in the healthy population, but many, even the majority, have none of these [80]."

The epidemiological data led to a controversial change in terminology. Some investigators thought replacing 'psychogenic' with 'functional' was obfuscating and motivated by the desire to increase patient acceptance of the diagnosis [81] which is famously poor [30,82,83]. Other investigators considered the term 'psychogenic' and its proposed etiology "poorly defined" and not supported by current evidence [34]. The term 'functional' was recommended because like other psychiatric disorders, "the cause of Conversion Disorder is unknown and it is desirable that it remain an atheoretical category in which primacy is given to the symptoms without making assumptions about their etiology [84]." While the empirical evidence may have led to a change in terminology, it has not disabused most FND and PNES investigators of their *conviction* that the origin of functional symptoms is primarily psychological [37,53,54,57,75,81,85-87]. Thus, the search continues for the mysterious etiology producing these 'non-neurologic' symptoms with seasoned investigators acknowledging, "we really do not know the cause [57]" and welcoming alternative explanations of functional symptoms which "should be given due consideration [88]."

Studies show that *physical therapy*, not psychotherapy, has emerged as a "surprisingly effective [89]" treatment for functional motor and gait disturbances [46,90-93]. These studies led to the development of an expert consensus recommending the use of physical therapy in functional movement disorders [74]. The question has been posed, "Why offer a physical treatment for a mental problem? [70]" and the short answer is because it works. The more salient question is why does it work so well? Occam's razor, the law of parsimony, concludes that the abnormal movements labelled 'functional' respond surprisingly well to targeted physical interventions because these patients have ordinary neurologic disease, *not a disorder with a psychogenic or otherwise 'non-neurologic' etiology.*

Patients diagnosed with PNES have demonstrated improvements with psychotherapy [8] but the same response to psychotherapy has been reported in patients with drug-resistant epilepsy [94]. Both populations have shown a reduction in seizure frequency and improvements in clinical co-morbidities [8,94,95]. A positive response to psychotherapy *does not differentiate* patients with PNES from patients with epilepsy.

The absence of neurologic (organic) findings is what *distinguishes* functional conditions from genuine neurologic disorders. The literature is *unequivocal* on this point. For decades, the terms 'hysterical' and 'psychogenic' were synonymous with a 'non-organic' etiology [15,96]. Conversion Disorder (FND) is a diagnosis of exclusion requiring that any neuropathological explanation of symptoms has been ruled out [29,70,97]. Psychogenic Movement Disorders are to be distinguished from organic diseases [24]. Functional neurological symptoms are somatic symptoms superficially resembling organic disorders of the nervous system but for which no physical explanation can be found [98]. The brains of patients diagnosed with FND are structurally normal [86]. Functional neurological symptoms are not explained by organic pathology [48,49,82,99,100]. Functional symptoms are not caused by organic damage [101]. Patients with FND have no neurological disease [102]. Functional neurological disorders are not due to irreversible brain damage [103,104]. Functional symptoms refer to weakness or movement disorders that are genuine but do not relate to an underlying neurologic disease [13,105]. The potential for *mistaking a neurologic condition* for a functional disorder has *decreased considerably with advances in neuroimaging* [67]. Patients were told that their 'functional' symptoms involved a "software problem with the brain rather than a hardware problem [104]."

The sole distinguishing characteristic- that patients with symptoms deemed 'functional' do not have neurologic disease- has been *debunked by empirical studies* showing pervasive brain pathology in the FND and PNES populations [37,106]. In a review of the literature, Szaflarski and LaFrance [106] found both structural imaging and sub-macroscopic abnormalities in patients with FND and PNES to the extent both are now considered network disorders *just like epilepsy* [107] *and dystonia* [108]. Seasoned investigators acknowledged that these findings were unexpected: The structural imaging in patients with functional disorders "should be" normal [106] and "the identification of such neurobiological correlates does not sit well with the understanding of PNES as a purely 'psychological' or 'psychogenic' disorder without any discernible 'physical correlates' [88]." It 'does not sit well' because for decades PNES and FND investigators asserted *unequivocally* that the *exclusion of neurologic disease was the basis* for a 'psychogenic' or 'functional' diagnosis. Now that the 'absence of neurologic disease' in the FND and PNES populations has been disproven, all that remains are 'neurologic symptoms' and 'evidence of pervasive neurologic disease.' The unanticipated data must then be plugged into the original hypothesis; In the *absence of organic findings*, investigators *presumed* that the neurologic symptom had a psychological origin [29,43-50]. The new equation reveals when the 'absence of neurologic disease' is replaced with 'presence of neurologic disease,' there is *no longer any basis to presume a psychological or otherwise 'non-neurologic' etiology*. The law of parsimony concludes that evidence of neurologic disease is simply evidence of a neurologic disorder.

Rather than revisiting the original hypothesis to examine the validity of the 'functional etiology,' FND and PNES investigators *readily incorporated* the 'evidence of neurologic disease' into their 'functional' frameworks. Functional neurological disorders are now a "software" and a "hardware" problem [55]. "Neuroimaging research is at the forefront of establishing neurobiological models" for functional disorders [60] and will "provide a foundation for postulating the neurobiological underpinnings of PNES [106]." The neurobiology of FNDs is not well understood but neural network dysfunction may underlie the symptomatic manifestation in FNDs [53]. It remains unclear whether structural alterations relate to predisposing vulnerabilities or are consequences of the functional disorder [55]. Functional neuroimaging has elucidated dysfunction in FNDs at the level of brain network activity, connectivity, and specific anatomic areas of altered metabolic demand during tasks [4]. Brain imaging techniques provide unprecedented opportunities to study the neural mechanisms underlying FND, which have long remained a mystery and clinical challenge for physicians, as they arise with no apparent underlying organic disease [109]. Neuroimaging is employed to exclude co-morbid organic disease in the diagnostic phase *and* explore the brain abnormalities in functional populations [37]. So, the 'evidence of neurologic disease' in patients diagnosed with 'non-neurologic' (functional) symptoms is being interpreted by investigators as 'neurologic evidence' of the 'non-neurologic' (functional) disorder. This amalgam of incoherence is born of *confirmation bias*.

While 'evidence of neurologic disease' has been absorbed into the theoretical framework, at the individual level, a functional diagnosis still rests on the 'absence of neurologic findings' (i.e. symptoms that cannot be explained by a neurologic disorder) [1]. But why is it that patients with symptoms deemed 'functional' (non-neurologic) show pervasive neurologic abnormalities when studied in-depth and en masse? The answer lies in the phenomenology of neurologic disease and the faulty diagnostic practice promoted in the FND and PNES literature.

Manifestations of neurologic disease are known for their kaleidoscopic and sometimes bizarre presentations. The clinical features of movement disorders can be various and heterogeneous, and the correct clinical diagnosis is often a challenge, even for expert neurologists [61]. The dyskinesias encountered in neurology clinics entail a

"hotch potch of miscellaneous and largely unclassified phenomena" which are "strikingly situation specific and variable in severity [110]." The symptoms of dystonia are *unusual, inconsistent* (e.g. can run but not walk, sing but not talk, have spasms of the eyelids that occur while reading but not while watching TV), fluctuate over time, worsen with anxiety, lessen while the patient is under hypnosis, and remit during sleep [23]. The clinical features of idiopathic torsion dystonia are *highly variable* and severity is largely determined by age of onset [111]. Because there are so many different clinical manifestations and causes, there are *no simple algorithms* for diagnosis addressing all dystonias [112]. The clinical presentation of movement disorders is complex, often variable, and sometimes bizarre [113]. The clinical signs of neurologic disease are often varied and confusing [114]. Medical teaching programs typically take *classic clinical presentations as the starting point* and present students with a representative constellation of features; however, patients *rarely present in this way* to a physician in clinical practice, *particularly in the early stages of a disease* [115]. Renowned neurologist David Marsden (who was instrumental in establishing dystonia as a neurologic disease rather than a psychogenic condition) [15] concluded that the bizarre and inconsistent presentation of dystonias, and their relief by certain inexplicable trick maneuvers, *were erroneously considered signs of a psychogenic etiology* [116].

Studies indicate that during the neurologic examination, clinical signs viewed as *atypical* or non-prototypical will likely garner a functional diagnosis. Functional Neurological Disorders; comprise neurologic symptoms unexplained by a *classical* neurologic disease [2]; have positive signs and symptoms that are *not typically seen* in other movement disorders [34]; may appear bizarre and should be inconsistent or incongruent with *classic* organic syndromes [117]; demonstrate *atypical* neurologic symptoms that do not conform to any neurologic disorder [58]; are positively identified as not being due to *recognized neurologic disease* [84]; show signs that are inconsistent and incongruent with the *normal rules* of pathology [118]; are characterized by inconsistent character with *unusual* presentations in amplitude, frequency, and distribution [83]; show internal inconsistency or incongruity with *known patterns* of neurologic disease [119].

The demand for a 'classic presentation' is a *wholly ill-suited* bar for phenomenon that is strikingly variable, confusing, largely unclassified, and sometimes bizarre. Eminent neurologist Jean-Martin Charcot stressed that classic signs (e.g. absent reflexes in patients with tabes dorsalis) are not always present and that it would be a "grave error" to rule out a condition based on the absence of a prototypical sign [17]. Manifestations of neurologic disease *do not lend themselves to rigid classification* and the scientific literature is replete with studies showing that non-prototypical presentations are not that atypical [97,114,119-127]. A diagnostic standard that demands a *prototypical* presentation, produces as an artifact, a not insubstantial subgroup of patients whose neurologic symptoms will be considered 'incongruent,' 'inconsistent' or 'incompatible' with *classic* neurologic disease (i.e. the FND population *during the diagnostic phase*).

The diagnosis of FND is based on 'positive signs' deemed inconsistent with neurologic disease [62]. The Hoover sign for example is hailed as a reliable test for functional leg weakness [64] but this sign has *well documented limitations*. Pain may affect the sign in several ways [64] and many clinicians have pointed out that the test can yield variable or equivocal results [128-133]. Entrainment, distractibility, and variability are all considered 'positive' evidence of FNDs [134,135] but these clinical signs are also found in patients with recognized neurologic disease [116,135,136]. Inexplicably, spontaneous remission is promoted as 'positive' evidence of a functional disorder [83,134]. Why would a symptom with a 'psychogenic' or otherwise 'non-neurologic' etiology simply remit without any intervention? The *incongruence* with the 'functional theory' aside, spontaneous remission has been observed in many neurologic disorders including Tourette's Syndrome [136], dystonia [137] and *a substantial proportion of untreated epilepsy patients* [138-140]. A placebo response is considered evidence of a functional disorder [24,75] but studies show these effects are ubiquitous across diseases, patient populations, and experimental paradigms, making them a key consideration in the design of clinical trials [141]. While these 'positive signs' are fallible and have been observed in neurologic disorders, they are presented in the literature as pathognomonic for a 'functional' disorder.

In functional gaits, walking is often *bizarre* and does not conform to any of the *usual patterns* observed with neurologic gait disorders [142]. In a recent case study, a 28-year-old woman developed transient weakness in her limbs and a gait disturbance that was deemed "bizarre" and functional by several neurologists [58]. Her neurologic examination was considered normal and no brain imaging had been completed because of the "obviously atypical nature of her symptoms [58]." During her fourth work-up, the patient finally got a CT scan which showed a large left frontal meningioma with considerable edema and midline shift. After the tumor was removed, her neurologic symptoms resolved, including the 'functional' gait. The author defended the FND diagnosis, pathologized the patients' sense of vindication, and emphasized that every medical professional involved was confident "the odd gait was functional" and

"less certainty was expressed on the role of such a large brain tumor in her presentation [58]." The law of parsimony concludes that the patient's neurologic symptoms, including her atypical gait, were caused by the tumor and the 'positive sign' that convinced the neurologists that her gait was 'functional,' was *erroneous and invalid.*

'Medically unexplained' symptoms are often equated with a functional etiology [3,143] but the logic is faulty [144] and constitutes a diagnostic approach not universally accepted in medicine. "Many patients with chronic diseases remain without a diagnosis despite extensive medical evaluation [145]." The Undiagnosed Diseases Program (UDP) was established at the National Institutes of Health (NIH) to meet the needs of patients with undiagnosed diseases and investigate the biologic characteristics of the diseases [123]. This program has defined "entirely new syndromes, rare diseases, and unusual presentations of common diseases [123]." A diagnostic approach inferring a 'functional' etiology from 'medically unexplained' symptoms has the *obvious potential to increase the incidence of 'functional' disorders*.

Psychogenic nonepileptic seizures have been *eliminated and induced by epilepsy surgery* [146-148]. In one study, nine of thirteen patients with PNES and co-morbid epilepsy stopped having PNES, and of those, seven also became free of epileptic seizures [146]. The PNES investigators conclusion: "That 7 of our patients became free of both seizures types does not necessarily mean that psychogenic seizures were also caused by focal, organic disease, which was cured by resection of the ictogenic part of the brain. Epilepsy surgery, whether successful or not, represents a significant life event, and the reasons for an improvement of psychogenic seizures could well be psychological [146]." In another study, five of nine patients with PNES and co-morbid epilepsy became seizure-free and eight subsequently developed PNES [147]. Why would 'psychogenic' or 'functional' seizures disappear or emerge following epilepsy surgery? The interpretation that the impact of neurosurgery "could well be psychological" [148] *shows confirmation bias*. The law of parsimony concludes PNES and epileptic seizures are significantly affected by epilepsy surgery because *both are epileptic events*.

The ictal vEEG test result determines whether a seizure is designated epileptic or PNES [63,149-151]. While studies confirm that epileptic seizures can *elude* scalp and intracranial electrodes [149-155], the fallible vEEG is *still employed as a litmus test* and hailed worldwide as the diagnostic 'gold standard' [5]. Though investigators assert PNES are "not associated with ictal electrical discharges in the brain [28]" this is merely a *presumption* and one unsupported by the objective evidence. Patients diagnosed with PNES and patients with epilepsy are *identical populations* separated only by a test with known limitations [32]. The condition known as PNES is just as disabling as epilepsy [8] because it is epilepsy [32]. The high incidence of PNES diagnoses [157,158] speaks to how frequently epileptic seizures are not captured by vEEG electrodes.

There is no condition precluding a functional diagnosis. People with intellectual disabilities make up a subgroup of PNES patients [159]. In a 2-year-old with a transient dystonic sign deemed 'functional,' poor frustration tolerance and emphasis on toilet training were identified as the probable origin of the 'functional' sign [160]. In one study, a 2-month-old infant was diagnosed with PNES [161]. Apparently PNES are "quite commonly" encountered in infants and young children and in most cases, "a careful history and examination will elucidate their nature [161]." The authors did not speculate as to what psychological factors could possibly be the origin of PNES in a newborn. Do these patients suffer from neurologic disease and epilepsy or a 'non-neurologic' disorder with a mysterious etiology? The law of parsimony concludes the former.

'Functional overlay' refers to the co-morbidity of neurologic disease and functional symptoms and apparently it is common [162]. Epilepsy and PNES is a well-documented variant of 'functional overlay' which may co-occur more frequently than previously thought [163]. Patients with Parkinson's Disease (PD) are also prone to functional symptoms [57,164] *especially 'functional' tremors that are located on the most (PD) affected side* [165]. Investigators of FND and PNES submit that while patients with PD and epilepsy suffer from recognized neurologic disorders with global neurologic impact, many of them have comorbid 'functional' symptoms distinct from the neurologic disease [55] and that stem from a mysterious condition with a 'psychogenic' or otherwise 'non-neurologic' etiology. The position for 'functional overlay' in these patients is insupportable. The law of parsimony concludes that *all tremors* observed in patients with PD are a manifestation of *the neurodegenerative disease (PD)*, and that *all seizures* observed in patients with epilepsy are *epileptic*.

Objective data is the arbitrator, not highly theoretical expert opinion, and it points *unwaveringly* to epilepsy and neurologic disease. Traumatic brain injury is a significant risk factor for *both* epileptic seizures [166,167] and seizures labelled PNES [168,169]. After a woman was kicked in the head by a horse, she started having seizures that were labelled PNES [170]. Epilepsy and PNES populations demonstrate pervasive brain abnormalities and *both* are considered

network disorders [106,107]. No single biomarker successfully differentiates PNES from epileptic seizures [151]. The semiology of PNES and epileptic seizures are so similar [5,45,171], there is *no clinical sign that has diagnostic value* [156]. Seizure trained dogs, who *recently demonstrated the existence of an epileptic odor* in humans [172], have reliably *alerted to both epileptic seizures* [172,175] and *seizures labelled* PNES [170,176,177]. The mortality rate of patients diagnosed with PNES is 2.5 times that of the general population and *similar to most patients with epilepsy* [178]. Patients 'mistakenly' treated for epilepsy, and later diagnosed with PNES, had their seizures *remit, or experienced a substantial reduction in seizure frequency* after they started antiepileptic medication [179]. Patients with seizures labelled PNES have shown mesial temporal lobe sclerosis on MRIs [180]. In two adolescents with drug resistant epilepsy, the semiology of their 'psychogenic' seizures showed a *striking resemblance* to their epileptic seizures [181]. Patients diagnosed with FND show pervasive brain abnormalities [37,106] to the extent that FND is considered a network disorder [106] *just like dystonia* [108]. Patients with cervical dystonia and patients diagnosed with FND *both* show structural brain abnormalities [37,106,182]. Functional neurological disorders are *just as disabling* as neurologic disorders [9,10]. *Physical injuries* are frequently associated with the *onset* of movement disorders labelled 'functional [183,184]. A patient with a family history of Huntington's Disease received a diagnosis of *psychogenic chorea* [185]. Physiotherapies are a highly effective treatment for patients with organic movement disorders [186,187] and patients with abnormal movements labelled 'functional' [74]. Transcranial magnetic stimulation (TMS) studies have shown that psychogenic and organic dystonia exhibit *similar neurophysiological abnormalities*, as compared to controls [188,189]. A patient diagnosed with 'functional' weakness showed significant improvement following repetitive transcranial magnetic stimulation (rTMS) [190]. A randomized, double-blind controlled study concluded that repetitive transcranial magnetic stimulation could represent a valuable intervention for patients with tremors labelled 'functional' [191]. Patients with dystonic tremor and patients diagnosed with FND have demonstrated significant improvement following administration of transcutaneous electrical stimulation (TENS) [192,48]. The objective evidence is compelling and the law of parsimony concludes that patients diagnosed with FND and PNES *suffer from ordinary neurologic disease and epilepsy*.

Conclusion

Empirical evidence debunks the psychogenic presumption underlying the functional diagnostic entity. Patients diagnosed with 'non-neurologic' (psychogenic or functional) symptoms show pervasive brain abnormalities. When this data is plugged into the originating hypothesis, and the 'absence of organic findings' is replaced with the 'presence of neurologic disease,' *there is no longer any basis to presume an etiology other than neurologic disease*. Evidence of neurologic disease is simply evidence of a neurologic disorder.

The diagnostic practice as promoted in the PNES and FND literature is fatally flawed and the proof of this *lies in the data*. Conditions labelled FND and PNES are *just as disabling* as neurologic disorders and epilepsy for a reason. The vEEG test result and the 'positive signs' employed to identify functional or 'non-neurologic' disorders have *isolated* patient populations with pervasive brain disease. In other words, *the 'functional' diagnostic markers are identifying patients with serious neurologic disorders*. While the theory underlying the 'functional' diagnosis has been dismantled by the data, modern investigators, like the neurologists who were called out by Eldridge and Fahn years ago [21], *are ignoring strong evidence of neurologic causation.* The prevalent misdiagnosis of patients with neurologic disease and epilepsy is the *real crisis in neurology*.

References

1. American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders, (5th Edn) (DSM-5), APA, Arlington, VA, USA.

2. Galli S, Béreau M, Magnin E, Moulin T, Aybek S, et al. (2020) Functional movement disorders. Rev Neurol (Paris) 176: 244-51.

3. Thenganatt MA, Jankovic J (2015) Psychogenic movement disorders. Neurol Clin 33: 205-24.

4. Espay AJ, Aybek S, Carson A, Edwards MJ, Goldstein LH, et al. (2018) Current Concepts in Diagnosis and Treatment of Functional Neurological Disorders. JAMA Neurol 75: 1132-41.

5. Goldstein LH, Mellers JDC (2012) Recent developments in our understanding of the semiology and treatment of psychogenic nonepileptic seizures. Curr Neurol Neurosci Rep 12: 436-44.

6. Sharma VD, Jones R, Factor SA (2017) Psychodynamic Psychotherapy for Functional (Psychogenic) Movement Disorders. J Mov Disord, 10: 40-4.

7. Stone J, Sharpe M, Rothwell PM, Warlow CP (2003) The 12 year prognosis of unilateral functional weakness and sensory disturbance. J Neurol Neurosurg Psychiatry 74: 591-6.

8. LaFrance WC Jr, Baird GL, Barry JJ, Blum AS, Frank Webb A., et al. (2014) Multicenter Pilot Treatment Trial for Psychogenic Nonepileptic Seizures: A Randomized Clinical Trial. JAMA Psychiatry 71: 997-1005.

9. Anderson KE, Gruber-Baldini AL, Vaughan CG, et al. (2007). Impact of psychogenic movement disorders versus Parkinson's on disability, quality of life, and psychopathology. Mov Disord 22:2204-9.

10. Gendre T, Carle G, Mesrati F, Hubsch C, Mauras T, et al. (2019) Quality of life in functional movement disorders is as altered as in organic movement disorders. J of Psychosomatic Research, 116: 10-6.

11. Gelauff J, Stone J, Edwards M, Carson A (2014) The prognosis of functional (psychogenic) motor symptoms: a systematic review. J Neurol Neurosurg Psychiatry, 85: 220-6.

12. Thomas M, Vuong KD, Jankovic J (2006) Long-term prognosis of patients with psychogenic movement disorders. Parkinsonism Relat Disord, 12: 382-7.

13. Edwards M, Bhatia K (2012) Functional (psychogenic) movement disorders: merging mind and brain. Lancet Neurol 11: 250-260.

14. Hallett M (2006) Psychogenic movement disorders: a crisis for neurology. J Clin Neurosci 6: 269-71.

15. Munts AG & Koehler PJ (2010) How psychogenic is dystonia? Views from past to present. Brain 133: 1552-64.

16. Philippon J, Poirier J (2009) Joseph Babinski A Biography. Oxford University Press: NY, USA.

17. Goetz, CG (1987) Charcot the Clinician: The Tuesday Lessons. Translated from: Lecons du Mardi a la Salpêtrière. Ravens Press.

18. Ondo WG (2006) Book review of- Psychogenic Movement Disorders: Neurology and Neuropsychiatry. N Engl J Med 354: 26.

19. Guttman MG (2001) The Enigma of Anna O. Moyer Bell, Wickford, Rhode Island & London.

20. Freud S, Breuer J (1978) Studies in Hysteria. Harmonsworth, UK: Penguin.

21. Eldridge R, Fahn S (1976) Preface. Adv Neurol 14: V.

22. Fahn S, Eldridge R (1976) Definition of dystonia and classification of the dystonic states, in Dystonia. Adv Neurol 14: 1-5.

23. Lesser RP, Fahn S (1978) Dystonia: A Disorder Often Misdiagnosed as a Conversion Reaction. Am J Psychiatry 135: 349-52.

24. Fahn S, Williams DT (1988) Psychogenic dystonia. Advances in Neurology, 50: 431-455.

25. Galli S, Tatu L, Bogousslavsky J, Aybek S (2018) Conversion, Factitious Disorder and Malingering: A Distinct Pattern or a continuum? Front Neurol Neurosci 42: 72-80.

26. Ford CV (2005) Deception syndromes: factitious disorders and malingering. In: Levenson JL, ed. The American Psychiatric Publishing Textbook of Psychosomatic Medicine. Washington, DC: American Psychiatric Publishing Inc; 2005: 297-309.

27. Voon V, Gallea C, Hattori N, Bruno M, Ekanayake V, Hallet M (2010) The involuntary nature of conversion disorder. Neurology 74: 223-228.

28. Reuber M (2009) The etiology of Psychogenic Non-epileptic Seizures: Toward a Biopsychosocial Model. Neurol Clin 27: 909-24.

29. Kanaan RA, Carson A, Wessely SC, Nicholson TR, Aybek S, David AS (2010) What's so special about conversion disorder? A Problem and a Proposal for Diagnostic Classification. Br J Psychiatry 196: 427-8.

30. McKee K, Glass S, Adams C, Stephen CD, King F, Parlman K, Perez DL, Kontos N. (2018) The Inpatient Assessment and Management of Motor Functional Neurological Disorders: An Interdisciplinary Perspective. Psychosomatics 59: 358-368.

31. Tuller D (2019) Trial by Error: Shaky Evidence for Signs of Functional Neurological Disorders. Virology Blog.

32. Carlson CA (2019) A Proposed Etiology of Psychogenic Nonepileptic Seizures. J Neurol Psychiatr Disord 1: 201.

33. Popkirov S, Hoeritzauer I, Colvin L, Carson AJ, Stone J (2019) Complex regional pain syndrome and functional neurological disorders- time for reconciliation. J Neurol Neurosurg Psychiatry, 90: 608-14.

34. Edwards MJ, Stone J, Lang AE (2014) From psychogenic movement disorder to functional movement disorder: it's time to change the name. Mov Disord, 29: 849-852.

35. Wiseman H, Reuber M (2015) New insights into psychogenic nonepileptic seizures 2011-2014. Seizure 29: 69-80.

36. Carson A, Lehn A, Ludwig L, Stone J (2016) Explaining functional disorders in the neurology clinic: a photo story. Pract Neurol 16: 56-61.

37. Roelofs JJ, Teodoro T & Edwards MJ (2019) Neuroimaging in functional movement disorders. Curr Neurol Neurosci Rep 19: 12.

38. Baslet G (2012) Psychogenic Nonepileptic Seizures: A Treatment Review. What Have We Learned Since the Beginning of the Millennium? Neuropsychiatr Dis Treat 8: 585-98.

39. Apartis E (2014) Clinical neurophysiology of psychogenic movement disorders: how to diagnose psychogenic tremor and myoclonus. Neurophysiol Clin 44: 417-24.

40. Anderson JR, Nakhate V, Stephen CD, Perez DL (2019) Functional (Psychogenic) Neurological Disorders: Assessment and Acute Management in the Emergency Department. Semin Neurol 39: 102-14.

41. Anderson KE, Gruber-Baldini AL, Vaughan CG, et al. (2007) Impact of psychogenic movement disorders versus Parkinson's on disability, quality of life, and psychopathology. Mov Disord 22: 2204-09.

42. Trimble M, Reynolds EH (2016) Chapter 1- A brief history of hysteria: From the ancient to the modern. Handb Clin Neurology, 139: 3-10.

43. Kranick S, Ekanayake V, Martinez V, Ameli R, Hallett M, Voon V (2011) Psychopathology and psychogenic movement disorders. Mov Disord 26: 1844-50.

44. Brandt J, Puente AN (2015) Update on Psychogenic Nonepileptic Seizures. Psychiatric Times 32.

45. Baslet G, Seshadri A, Bermeo-Ovalle A, Willment K, Myers L, et al. (2016) Psychogenic Non- epileptic Seizures: An Updated Primer. Psychosomatics 57: 1-7.

46. Jacob AE, Kaelin DL, Roach AR, Ziegler CH, LaFaver K, et al. (2018) Motor Retraining (MoRe) for Functional Movement Disorders: Outcomes from a 1-week Multidisciplinary Rehabilitation Program. PM R, 10: 1164-72.

47. Morgante F, Edwards MJ, Espay AJ (2013) Psychogenic Movement Disorders. Continuum (Minneap Minn), 19(5 Movement Disorders): 1383-96.

48. Ferrara J, Stamey W, Strutt AM, Adam O R, Jankovic J, et al. (2011) Transcutaneous electrical stimulation (TENS) for Psychogenic Movement Disorders. J Neuropsychiatry Clin Neurosci 23: 141-8.

49. Amar JY, Killinger RL, Borden K, Lollo V (2018) A seizure by any other name: Challenges and long-term implications of psychogenic nonepileptic seizures. The American Journal of Psychiatry Residents Journal 13: 3-6.

50. Keynejad RC, Carson AJ, David AS, Nicholson TR (2017) Functional neurological disorder: psychiatry's blind spot. The Lancet Psychiatry, 4.3: E2-E3.

51. Devinsky O, Gazzola D, LaFrance Jr WC (2011) Nature Reviews Neurology 7: 210-20.

52. Beghi M, Negrini PB, Perin C, Peroni F, Magaudda A, et al. (2015) Psychogenic non-epileptic seizures: so-called psychiatric comorbidity and underlying defense mechanisms. Neuropsychiatr Dis Treat 11: 2519-27.

53. O'Neal MA & Baslet G (2018) Treatment for patients with a Functional Neurological Disorder (Conversion Disorder): An Integrated Approach. American J of Psychiatry 175: 307-14.

54. Kanaan RAA, Craig TKJ (2019) Conversion disorder and the trouble with trauma. Psychological Medicine, 49: 1585-8.

55. Bèque I, Adams C, Stone J, Perez DL (2019) Structural alterations in functional neurological disorder and related conditions: a software and hardware problem? Neuroimage Clin 22: 101798.

56. Daum C, Hubschmid M, Aybek S (2014) The value of 'positive' clinical signs for weakness, sensory and gait disorders in conversion disorder: a systematic and narrative review. J Neurol Neurosurg Psychiatry 85: 180-90.

57. Hallett M (2018) The most promising advances in our understanding and treatment of functional (psychogenic) movement disorders. Parkinsonism Relat Disord 46: S80- 2.

58. Feinstein A (2018) Conversion Disorder. Continuum (Minneap Minn) Behavioral Neurology and Psychiatry 24: 861-72.

59. Picks S, Goldstein LH, Perez DL, Nicholson TR (2019) Emotional processing in functional neurological disorder: a review, biopsychosocial model and research agenda. J Neurol Neurosurg Psychiatry 90: 704-11.

60. Pal PK (2011) Electrophysiologic evaluation of psychogenic movement disorders. Journal of Movement Disorders 4: 21-32.

61. Peila E, Mortara P, Cicerale A, Pinessi L (2015) Paroxymal non-kinesigenic dyskinesia post-streptococcal syndromes and psychogenic movement disorders: a diagnostic challenge. BMJ Case Reports 2015: 207449.

62. Walzl D, Carson A J, Stone J (2019) The misdiagnosis of functional disorders as other neurological conditions. Journal of Neurology 266: 2018-26.

63. LaFrance WC Jr, Baker GA, Duncan R, Goldstein LH, Reuber M, et al. (2013) Minimum requirements for the Diagnosis of Psychogenic Nonepileptic Seizures: A Staged Approach. Epilepsia, 54: 2005-18.

64. Stone J, Sharpe M (2001) Hoover's sign. Practical Neurology 1: 50-3

65. Stone J, Edwards M (2012) Trick or Treat? Showing Patients with Functional (Psychogenic) Motor Symptoms Their Physical Signs. Neurology, 79: 282-4.

66. Fahn S, Williams D, Reches A, Lesser RP, Jankovic J, et al. (1983) Hysterical Dystonia, a Rare Disorder: Report of Five Documented Cases. Neurology 33: 161.

67. LaFrance WC Jr (2008) Diagnosing conversion weakness with the Spinal Injuries Center test: when Hoover doesn't help. Neurology 71: e57.

68. Mayor R, Smith PE, Reuber M (2011) Management of patients with nonepileptic attack disorder in the United Kingdom: a survey of health care professionals. Epilepsy Behav 21: 402-6.

69. Ellenstein A, Kranick SM, Hallett M (2011) An Update on Psychogenic Movement Disorders. Curr Neurol Neurosci Rep 11: 396-403.

70. Kirschner KL, Smith GR, LorishP, Antiel RM, Frost F, et al. (2012) "Why Can't I Move, Doc?" Ethical dilemmas in Treating Conversion Disorders. American Academy of Physical Medicine and Rehabilitation, 4.4: 296-303.

71. Nielsen G, Buszewicz M, Stevenson F, Hunter R, Holt K, et al. (2017) Randomised feasibility study of physiotherapy for patients with functional motor disorders. J Neurol Neurosurg Psychiatry 88: 484-90.

72. LaFrance WC Jr., Reuber M, Goldstein L (2013) Management of psychogenic non- epileptic seizures. Epilepsia 54: 53-67.

73. Lafrance WC Jr, Devinsky O (2002) Treatment of nonepileptic seizures. Epilepsy Behav 3.5: 19-23.

74. Nielsen G, Stone J, Matthews A, Brown M, Sparkes C, et al. (2015) Physiotherapy for functional motor disorders: a consensus recommendation. J of Neurol Neurosurg Psychiatry 86: 1113-9.

75. Edwards MJ, Fotopoulou A, Pareès I (2013) Neurobiology of functional (psychogenic) movement disorders. Curr Opin Neurol, 26.4: 442-7.

76. Reich SG (2006) Psychogenic movement disorders. Semin Neurol 26: 289-96.

77. Nicholson TR, Stone J, Kanaan RA (2011) Conversion Disorder: a problematic diagnosis. J Neurol Neurosurg Psychiatry, 82: 1267

78. Pringsheim T, Edwards M (2017) Functional movement disorders: Five new things. Neurol Clin Pract 7: 141-7.

79. Fobian, AD & Elliott, LE (2019) A review of functional neurological symptom disorder etiology and the integrated etiological summary model. J Psychiatry Neurosci 44: 8-18.

80. Edwards M (2016) Functional neurological symptoms: welcome to the new normal. Pract Neurol 16: 2-3.

81. Fahn S & Olanow CW (2014) "Psychogenic Movement Disorders": they are what they are. Mov Disord 29: 853-6.

82. Bolton C & Goldsmith P (2018) Complaints from patients with functional neurological disorders: a cross-sectional UK survey of why patients complain and the effect on the clinicians who look after them. BMJ Open, 8: e021573.

83. Peckham EL, Hallett M (2009) Psychogenic Movement Disorders. Neurol Clin 27.3: 801-vii.

84. Stone J, LaFrance WC Jr, Brown R, Spiegel D, Levenson JL, et al. (2011) Conversion Disorder: Current problems and potential solutions for DSM 5. J Psychosom Res 71: 369-76.

85. Brown RJ, Reuber M (2016) Towards an integrative theory of psychogenic non-epileptic seizures (PNES). Clin Psychol Rev 47: 55-70.

86. Barbey A & Aybek S (2017) Functional movement disorders. Current Opinion in Neurology 30: 427-34.

87. Nicholson TR, Aybek S, Craig T, Harris T, Wojcik W, David AS, Kanaan RA (2016) Life events and escape in conversion disorder. Psychol Med 46: 2617-26.

88. McSweeney M, Reuber M & Levita L (2017) Neuroimaging studies in patients with psychogenic non-epileptic seizures: A systematic meta-review. Neuroimage Clin 16: 210-21.

89. Matthews A, Brown M, Stone J (2016) Inpatient physiotherapy for functional (psychogenic) gait disorder: A case series of 35 patients. Mov Disord Clin Pract 3: 603-6.

90. Nielsen G, Ricciardi L, Demartini B, Hunter R, Joyce E, et al. (2015) Outcomes of a 5-day physiotherapy programme for functional (psychogenic) motor disorder. J Neurol 262: 674-81.

91. Jordbru AA, Smedstad LM, Klungs-o with line thru it-yr, Martinsen EW (2014) Psychogenic gait disorder: a randomized controlled trial of physical rehabilitation with one-year follow-up. J Rehabil Med 46: 181-7.

92. Czarnecki K, Thompson JM, Seime R, Geda YE, Duffy JR, et al. (2012) Functional movement disorders: successful treatment with a physical therapy rehabilitation protocol. Parkinsonism Relat Disord 18: 247-51.

93. Dallocchio C, Arbasino C, Klersy C, Marchioni E (2010) The effects of physical activity on psychogenic movement disorders. Mov Disord 25: 421-5.

94. Tang V, Poon WS, Kwan P (2015) Mindfulness-Based Therapy for Drug-Resistant Epilepsy: An Assessor-Blinded Randomized Trial. Neurology 85: 1100-7.

95. Carlson P, Perry KH (2017) Psychological Interventions for Psychogenic Non-epileptic Seizures: A Meta-Analysis. Seizure 45: 142-50.

96. Edwards MJ, Adams RA, Brown H, Paree's I, Friston KJ (2012) A Bayesian account of 'hysteria.' Brain 135: 3495-12.

97. Barnum R (2014) Problems with diagnosing Conversion Disorder in response to variable and unusual problems. Adolesc Health Med Ther 5: 67-71.

98. Markus Reuber, Stephanie Howlett, Ajjaz Khan, Richard A Grünewald, et al. (2007) Non-epileptic seizures and other functional neurological symptoms: predisposing, precipitating, and perpetuating factors. Psychosomatics 48: 230-8.

99. Carson AJ, Ringbauer B, Stone J, McKenzie L, Warlow C, et al. (2000) Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. J Neurol Neurosurg Psychiatry 68: 207-10.

100. Yasuda T, Hatakeda J, Yoshimasu H (2018) Assessments and treatments for neurological symptoms that do not match organic findings. Brain Nerve, 70: 971-9.

101. Vuilleumier P (2014) Brain Circuits Implicated in Psychogenic Paralysis in Conversion Disorders and Hypnosis. Neurophysiol Clin, 44: 33-7.

102. Carson A, Lehn A, Ludwig L, Stone J (2016) Explaining functional disorders in the neurology clinic: a photo story. Pract Neurol 16: 56-61.

103. Garcin B, Mesrati F, Hubsch C, Mauras, Iliescu I, et al. (2017) Impact of Transcranial Magnetic Stimulation on Functional Movement Disorders: Cortical Modulation or a Behavioral Effect? Frontiers in Neurology 8: 338.

104. Stone J (2014) Functional neurological disorders: The neurological assessment as treatment. Clinical Neurophysiology 44: 363-73.

105. Stone J, Warlow C, Sharpe M (2010) The symptom of functional weakness: a controlled study of 107 patients. Brain 133.5: 1537-51.

106. Szaflarski JP, LaFrance WC Jr (2018) Psychogenic nonepileptic seizures as a network disorder- Evidence from neuroimaging of functional (psychogenic) neurological disorders. Epilepsy Curr 18.4: 211-6.

107. Whelan CD, Altmann A, Botia JA, Jahanshad N, Hibar DP, et al. (2018) Structural Brain Abnormalities in the Common Epilepsies Assessed in a Worldwide ENGIMA Study. Brain 141: 391-408.

108. Prudente CN, Hess EJ, Jinnah HA (2014) Dystonia as a Network Disorder: What Is the Role of the Cerebellum? Neuroscience 260: 23-5.

109. Aybek S, Vuilleumier P (2016) Imaging studies of functional neurologic disorders. Handb Clin Neurol 139: 73-84.

110. Lees AJ (2002) Odd and unusual movement disorders. J Neurol Neurosurg Psychiatry 72.1: i17-i21.

111. Harding A (1994) Movement disorders: genetic aspects. In Movement Disorders 3, edited by C David Marsden and Stanley Fahn. Butterworth-Heinemann Ltd, Linacre House, Jordan Hill, Oxford OX2 2DP

112. Jinnah HA, Factor SA (2015) Diagnosis and Treatment of Dystonia. Neurol Clin 33: 77-100.

113. Abdo WF, van de Warrenburg BPC, Burn DJ, Quinn NP, Bloem BR (2010) The clinical approach to movement disorders. Nat Rev Neurol 6: 29-37.

114. O'Dell C (1988) Atypical presentations of neurological illness in the elderly. Geriatrics 43: 35-7.

115. Nonnekes J, Goselink RJM, Ruzicka (has marks above) E, Fasano A, Nutt JG, et al. (2018) Neurological disorders of gait, balance and posture: a sign-based approach. Nat Rev Neuol 14: 183-9.

116. Marsden CD (1976) The problem of adult-onset idiopathic torsion dystonia and other isolated dyskinesias in adult life (including blepharospasm, oromandibular dystonia, dystonic writer's cramp, and torticollis, or axial dystonia). Adv Neruol 14:259-76.

117. Factor SA, Podskalny GD, Mohlo ES (1995) Psychogenic movement disorders: frequency, clinical profile, and characteristics. J Neurol Neurosurg Psychiatr 59: 406-12.

118. Hassa T, de Gel E, Tuescher O, Schmidt R, Schoenfeld MA (2016) Functional networks of motor inhibition in conversion disorder patients and feigning subjects. NeuroImage:Clinical 11: 719-27.

119. Teodoro T, Oliveira R, Afonso P (2019) Atypical lyme neuroborreliosis, Guillain-Barre (accent e) Syndrome or Conversion Disorder: Differential diagnosis of unusual neurological presentations. Case Rep Neurol 11: 142-7.

120. Agarwal T, Palm M, Bhavaraju-Sanka R, Jackson C (2018) An atypical presentation of Congenital Myasthenic Syndrome masquerading as Acquired Myasthenia Gravis. Neurology 90: 15.

121. Bakheit AMO (2006) Recognition of acute illness in people with chronic neurological disability. Postgrad Med J 82: 267-9.

122. Elmali AD, Ayşegül G, Poyraz BC, Kiziltan ME, Ertan S, et al. (2017) A case illustrating how tremor of Wilson's Disease may mimic functional tremor. Acta Neurol Belg 117: 351-3.

123. Gahl WA, Markello TC, Toro C, et al. (2012) The National Institutes of Health Undiagnosed Diseases Program: insights into rare diseases. Genet Med 14: 51-59.

124. Pillai J, Krishnan K, Rothenberg KG (2019) Bridging the gap between neurology and psychiatry- atypical presentation of common neurodegenerative disorders. AAGP Annual Meeting. Am J Gertiatr Psychiatry 27:3S.

125. Mabrouk EE, Achour NB, Turki A, Benrhourma H, Klaa H, et al. (2017) Atypical presentations of Guillain-Barr'e Syndrome in children. European Journal of Paediatric Neurology, 21: e230.

126. O'Brien MD (2005) Unusual presentations of neurological conditions: make your diagnosis. J R Soc Med 98.12: 569-70.

127. Kwon DY, Kim J, Yoon H, Park MH (2018) Progressive myoclonic tremor mimicking functional tremor in Hirayama disease. Acta Neurol Belg 118: 517-8.

128. Magee KR (1962) Hysterical hemiplegia and hemianesthesia. Post Grad Med 31: 339-45.

129. Merskey H (1986) The importance of hysteria. Br J Psychiatry 149: 23-8.

130. Jones JB (1990) Conversion disorder camouflage for brain lesion in two cases. Arch Intern Med 150: 1343-5.

131. Hirose G (2016) Hoover's sign. Clinical Neuroscience (Tokyo) 18: 606.

132. Pryse-Philips W (1995) Companion to clinical neurology. Boston: Little Brown: 422.

133. Sonoo M (2004) Abductor sign: a reliable new sign to detect unilateral non-organic paresis of the lower limb. J Neurol Neurosurg Psychiatry 75: 121-5.

134. Shill H, Gerber P (2006) Evaluation of clinical diagnostic criteria for psychogenic movement disorders. Mov Disord 21: 1163-8.

135. Kenney C, Diamond A, Mejia N, Davidson A, Hunter C, et al. (2007) Distinguishing psychogenic and essential tremor. J Neurol Sci 263.1-2: 94-9.

136. Ahmed MAS, Martinez A, Yee A, Cahill D, Besag FMC, et al. (2008) Psychogenic and organic movement disorders in children. Developmental Medicine & Child Neurology 50: 300-4.

137. Eldridge R, Ince SE, Chernow B, Milstien S, Lake CR, et al. (1984) Dystonia in 61-year-old identical twins: observations over 45 years. Ann Neurol 16: 356-8.

138. Kwan P, Sander JW (2004) The Natural History of Epilepsy: An Epidemiological View. J Neurol Neurosurg Psychiatry 75: 1376-81.

139. Nicoletti A, Sofia V, Vitale G, Bonelli SI, Bejarano V, et al. (2009) Natural History and Mortality of Chronic Epilepsy in an Untreated Population of Rural Bolivia: A Follow-up After 10 years. Epilepsy 50: 2199-206.

140. Watts AE (1992) The Natural History of Untreated Epilepsy in a Rural Community in Africa. Epilepsy 33: 464-68.

141. Kaas BM, Humbyrd CJ, Pantelyat A (2018) Functional Movement Disorders and Placebo: A Brief Review of the Placebo Effect in Movement Disorders and Ethical Considerations for Placebo Therapy. Mov Disord Clin Pract 5: 471-8.

142. Miyasaki JM (2019) Functional Movement Disorders- UpToDate.

143. Stone J, Carson A, Duncan R, et al. (2011) Which neurological diseases are most likely to be associated with "symptoms unexplained by organic disease." J Neurol 259: 33-8.

144. Sykes R (2010) Medically Unexplained Symptoms and the Siren "Psychogenic Inference." Philosophy, Psychiatry & Psychology: PPP 17: 289-299.

145. Splinter K, Adams DR, Bacino CA, Bellen HJ, Bernstein JA, et al. (2018) Effect of genetic diagnosis of patients with previously undiagnosed disease. New England Journal of Medicine 379: 2131-9.

146. Reuber M, Kurthen M, Fernández G, Schramm J, Elger CE, et al. (2002) Epilepsy surgery in patients with additional psychogenic seizures. Arch Neurol 59: 82-6.

147. González Otárula KA, Tan YL, Dubai F, Correa JA, Knowlton RC, et al. (2017) Psychogenic Nonepileptic Seizures in Patients with Surgically Treated Temporal Lobe Epilepsy: Presurgical and De Novo Post-Surgical Occurrence. Epilepsy Behav 75: 252-5.

148. Gobbi G, Peroni F, Filippini M, Beghi M, Giulioni M, et al. (2016) PNES recovery after surgery: an unusual evolution of PNES. Clinical Cases Rev Epilepsy 1: 149-54.

149. Asadi-Pooya AA, Sperling MR (2015) Epidemiology of Psychogenic Nonepileptic Seizures. Epilepsy Behav 46: 60-5.

150. Perez DL, LaFrance WC Jr (2016) Nonepileptic Seizures: An Updated Review. CNS Spectr 21: 239-46.

151. Sundararajan T, Tesar GE, Jimenez XF (2016) Biomarkers in the Diagnosis and Study of Psychogenic Nonepileptic Seizures: A Systematic Review. Seizure 35: 11-22.

152. Cantrell V (2016) Psychogenic Nonepileptic Seizures versus Epileptic Seizures: An Unusual Case Report. Neurodiagn J 56: 165-77.

153. Williamson PD, Spencer DD, Spencer SS, Novelly RA, Mattson RH, et al. (1985) Complex Partial Seizures of Frontal Lobe Origin. Ann Neurol 18: 497-504.

154. Limura Y, Sugano H, Nakajima M, Higo T, Ssuzuki H, et al (2016) Analysis of Epileptic Discharges from Implanted Subdural Electrodes in Patients with Sturge-Weber Syndrome. PloS One 11: e0152992.

155. Marsh ED, Peltzer B, Brown MW, Wusthoff, et al. (2010) Interictal EEG Spikes Identify the Region of Seizure Onset in Some but Not All Pediatric Epilepsy Patients. Epilepsia 51: 592-601.

156. Gasparini S, Beghi E, Ferlazzo E, Beghi M, Belcastro V, et al. (2019) Management of psychogenic non-epileptic seizures: a multidisciplinary approach. Eur J Neurol 26: 205-e15.

157. Gillig PM (2013) Psychogenic Nonepileptic Seizures. Innov Clin Neurosci 10: 15-8.

158. Smith BJ (2014) Closing the Major Gaps in PNES research Finding a Home for a Borderland Disorder. Epilepsy Curr 14: 63-7.

159. Kanemoto K, LaFrance Jr WC, Duncan R, Gigineishvili D, Park SP, et al. (2017) PNES around the world: Where are we now and how we can close the diagnosis and treatment gaps- an ILAE PNES Task Force Report. Epilepsia Open, 2.3: 307-16.

160. Ghosh D (2017) A 2 year-old girl with functional dystonia manifesting as forceful finger flexion producing fixed posture of clenched hands. Mov Disord Clin Pract. 4.6: 893-4.

161. Kotagal P, Costa M, Wyllie E, Wolgamuth B, et al. (2002) Paroxysmal nonepileptic events in children and adolescents. Pediatric 110.4: e46.

162. Stone J, Carson A, Duncan R, Roberts R, Coleman R, et al. (2011) Which Neurological Diseases Are Most Likely to Be Associated With "Symptoms Unexplained by Organic Disease." J Neurol 259.1: 33-8.

163. Yon MI, Azman F, Irsel Tezer F, Saygi S, et al. (2020) The coexistence of psychogenic nonepileptic and epileptic seizures in the same patient is more frequent than expected: Is there any clinical feature for defining these patients? Epilepsy Behav 105: 106940.

164. Wissel BD, Dwivedi AK, Merola A, Chin D, Jacob C, et al. (2018) Functional neurological disorders in Parkinson disease. J of Neurol Neurosurg Psychiatry 89: 566- 71.

165. Akkaoui MA, Geoffroy P, Roze E, Degos B, Garcin B, et al. (2019) Functional Motor Symptoms in Parkinson's Disease, and Functional Parkinsonism: A Systematic Review. International Parkinson and Movement Disorder Society. 2019 International Congress. Abstract Number: 386.

166. Lowenstein DH (2009) Epilepsy After Head Injury: An Overview. Epilepsia 50: 4-9.

167. Webster KM, Sun M, Crack P, O'Brien TJ, Shultz SR, et al. (2017) Inflammation in epileptogenesis after traumatic brain injury. J Neuroinflammation 14: 10.

168. LaFrance WC Jr, Deluca M, Machan JT, Fava JL, et al. (2013) Traumatic brain injury and psychogenic nonepileptic seizures yield worse outcomes. Epilepsia 54: 718-25.

169. Salinsky M, Storzbach D, Goy E, Evrard C, et al. (2015) Traumatic brain injury and psychogenic seizures in veterans. J Head Trauma Rehabil 30: E65-E70.

170. Ortiz R and Liporace J (2005) "Seizure alert dogs": Observations from an inpatient video/EEG unit. Epilepsy Behav 6: 620-2.

171. Mellers JDC (2005) The Approach to Patients with Non-Epileptic Seizures. Postgrad Med J 81: 498-504.

172. Catala A, Grandgeorge M, Schaff J-L, Cousillas H, Hausberger M, et al. (2019) Dogs demonstrate the existence of an epileptic seizure odour in humans. 9: 4103.

173. Strong V, Brown SW, Walker R (1999) Seizure-alert dogs - fact or fiction? Seizure 8: 62-5.

174. Strong V, Brown S, Huyton M, Coyle H, et al. (2002) Effect of trained seizure alert dogs on frequency of tonic-clonic seizures. Seizure 11: 402-5.

175. Kirton A, Winter A, Wirrell E, Snead OC, et al. (2008) Seizure response dogs: Evaluation of a formal training program. Epilepsy Behav 13: 499-504.

176. Krauss GL, Choi JS, Lesser RP (2007) Pseudoseizure dogs. Neurology 68: 308-9.

177. Doherty MJ, Haltiner AM (2007) Wag the dog: skepticism on seizure alert canines. Neurology 68: 309.

178. Borreli L (2018) December 6 2018. Neurology Today.

179. Alessi R, Valente KD (2014) Psychogenic nonepileptic seizures: should we use response to AEDS as a red flag for the diagnosis? Seizure 23: 906-8.

180. Lowe MR, De Toledo JC, Rabinstein AA, Giulla MF (2001) MRI evidence of mesial temporal sclerosis in patients with psychogenic nonepileptic seizures. Neurology 56: 823.

181. Ostrowsky-Coste K, Montavont A, Keo-Kosal P, Guenot M, Chatillon CE, et al. (2013) Similar Semiology of Epileptic and Psychogenic Nonepileptic Seizures Recorded During Stereo-EEG. Seizure 22: 897-900.

182. Prell T, Peschel T, Köhler B, Bokemeyer MH, Dengler R, et al. (2013) Structural brain abnormalities in cervical dystonia. BMC Neurosci 14: 123.

183. Stone J, Carson A, Aditya H, Prescott R, Zaubi M, et al. (2009). The role of physical injury in motor and sensory conversion symptoms: A systematic and narrative review. Journal of Psychosomatic Research 66: 383-90.

184. Schrag A, Trimble M, Quinn N, Bhatia K (2004) The syndrome of fixed dystonia: an evaluation of 103 patients. Brain 127: 2360-72.

185. Fekete R, Jankovic J (2010) Psychogenic chorea associated with family history of Huntington disease. Mov Disord 25: 503-4.

186. Queiroz MAR, Chien HF, Sekeff-Sallem FA, Barbosa ER (2012) Physical therapy program for cervical dystonia: a study of 20 cases. Functional Neurology 27: 187-92.

187. Hohler AD, Tsao JM, Katz DI, Dipiero J, Hehl CL, et al. (2012) Effectiveness of an Inpatient Movement Disorders Program for Patients with Atypical Parkinsonism. Parkinson Disease 2012: 871974.

188. Espay AJ, Morgante F, Purzner J, et al (2006) Cortical and spinal abnormalities in psychogenic dystonia. Ann Neurol 59: 825-34.

189. Avanzino L, Martino D, van de Warrenburg BP, et al.. (2008) Cortical excitability is abnormal in patients with the "fixed dystonia" syndrome. Mov Disord 23: 646-52.

190. Portaro S, Milardi D, Naro A, Chillura A, Corallo F, et al. (2018) Chasing the chameleon: Psychogenic paraparesis responding to non-invasive brain stimulation. Psychiatry Investing 15: 428-31.

191. Taib S, Ory-Magne F, Brefel-Courbon C, Moreau Y, Thalamas C, et al. (2019) Repetitive transcranial magnetic stimulation for functional tremor: A randomized, double-blind, controlled study. Mov Disord 34: 1210-9.

192. Bending J, Cleeves L, (1990) Effect of electrical nerve stimulation on dystonic tremor. Lancet 336: 1385-6.