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

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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 psychological 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 intense 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, Szaflbarski 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.

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