Current Clinical Neurology
Series Editor: Daniel Tarsy

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Functional Movement Disorder

An Interdisciplinary Case-Based Approach

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Current Clinical Neurology

Series Editor

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Preface

Functional movement disorder (FMD), a subtype of functional neurological disorder (FND), is a prevalent, potentially disabling, and costly condition at the intersection of neurology and psychiatry. After being of significant interest to a number of early leaders across neurology, psychiatry, and psychology (e.g., Charcot, Freud, Janet, Briquet, Babinski), the late twentieth century was unfortunately marked by limited interest in the field of FND. Part of the difficulties arise from the notion that FND inherently challenges the conceptualization of modern-day medical specialties and societal views of physical health and mental health more broadly. Thankfully, interest in FMD and related conditions has renewed in the last 20 years. Improvements in diagnostic specificity, an expanding "toolbox" of evidence-based treatments, and an international, multidisciplinary professional society (www.FNDsociety.org) are all driving interest in this field among clinicians and researchers alike. Furthermore, FMD is scientifically compelling, teaching us about a range of cognitive-affective neuroscience principles, and when patients respond well to treatment, effect sizes rival those seen throughout the brain sciences.

In this case-based textbook on FMD, we offer readers a practical, evidence-based approach to the assessment and management of FMD presentations over 32 chapters. Chapter 1 provides a historical perspective on FMD and FND more broadly, with Chaps. 2 and 3 outlining emerging neural mechanisms and the importance of the biopsychosocial model, respectively. Chapter 4 offers an integrated clinical neuroscience approach to FMD. Thereafter, Chaps. 5, 6, 7, 8, 9, 10, 11, 12, and 13 detail case-based examples (including videos) and practical discussion on the assessment and management of the full range of functional movement symptoms – including but not limited to functional limb weakness, tremor, dystonia, parkinsonism, tics, jerks, gait difficulties, and speech/voice abnormalities. Non-motor symptoms found in patients with FMD are outlined in Chap. 14, while Chaps. 15 and 16 offer assessment recommendations for pediatric and elderly populations. Informed by the latest research and expert opinions, Chaps. 17, 18, 19, 20, 21, 22, 23, 24, 25, and 26 provide practical suggestions in regard to therapeutic approaches, including education, physical/occupational/speech and language therapies, and psychotherapy. The role of placebo treatment and transcranial magnetic stimulation are visited in Chaps. 27 and 28, respectively, potentially promising interventions requiring considerably more research. Chapters 29, 30, and 31 are also on noteworthy topics, including measuring symptoms, managing obstacles in longitudinal care, and the treatment of pediatric

FMD. Chap. 32 succinctly details the career narratives of the four co-editors, as a way of hopefully inspiring the next generation of clinicians and researchers toward the field of FMD and related conditions. Throughout the text, we advocate for a patient-centered, biopsychosocially informed clinical neuroscience perspective. Given that the clinical landscape of FMD and related conditions have undergone transformative changes in the last several decades, it can be expected that portions of the content put forth here will be updated in the coming decades.

Overall, we believe that this case-based textbook will be a valuable resource for trainees and seasoned clinicians alike across the fields of neurology, psychiatry, medicine, psychology, social work, allied mental health disciplines, and physical rehabilitation (including physical, occupational, and speech-language therapists). Given the overlap between FMD, other FND subtypes, chronic pain, and the range of functional disorders seen across medicine, clinicians working in these spaces will likely also find this text useful.

We would like to express our sincere and utmost gratitude to our chapter authors, who generously provided their expertise and time to help disseminate their knowledge and skills. We are also indebted to our patients, mentors, and current and former colleagues from whom we have learned so much. Lastly, we thank our families for their unwavering support and encouragement.

Saratoga Springs, NY, USA — Kathrin LaFaver
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Series Editor's Introduction

This volume, *Functional Movement Disorder: An Interdisciplinary Case Based Approach* provides a very useful and much needed comprehensive review of *functional movement disorder* as a subtype of *functional neurological disorder* which recently have become recognized as common and worthy of the serious attention of both neurologists and psychiatrists. The editors of this book, Drs. LaFaver, Maurer, Nicholson, and Perez, have collected many careful and thoughtful contributions from their colleagues, all of whom have taken a very serious interest in functional neurological disorder. In Part I of this book, the historical perspective provided by Richard Kanaan in Chap. 1 sets the stage for this discussion by reminding the reader of currently outmoded historical terms such as "conversion disorder", "psychogenic disorder", and "hysterical disorder" which have been of very limited value in characterizing, understanding, and treating patients with functional neurological disorder. In Chap. 2, the pathophysiologic underpinnings of functional movement disorder is imaginatively laid out by Dr. Mark Hallett, who has emphasized the concept of loss of "self-agency" by the patient as a way of understanding the occurrence of abnormal movements which closely resemble abnormal movements that arise from physical lesions of the brain. In Part II of the book, specific case descriptions will assist the clinician who may believe they are encountering a patient with functional tremors, jerky movements, tics, gait disorders, or some other functional movement disorder. Finally, Part III of the book provides a variety of useful approaches to the management of functional movement disorder by a variety of therapeutic techniques.

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Part I

Framework

1 A Historical Perspective on Functional Neurological Disorder

Richard A. A. Kanaan

A history of one of mankind's oldest maladies would be a monumental work, but that is not this chapter. This chapter explores the history of functional neurological disorder (FND), a malady of much more recent birth. How recent? A search on PubMed for "functional neurological disorder" suggests its first English-language appearance was perhaps in 2015 [1]. That year there were 2 publications using the term; the following year there were 6, and a further 9 using the Americanized "neurologic". In 2016, mere months after this first appearance, the authoritative 700-page handbook of clinical neurology on the subject chose "Functional Neurologic Disorders" as its title [2]. Strikingly, for a term that was not the official choice of DSM-5, ICD-10, or the proposed ICD-11, it ("FND") was incorporated into the name of every patient group in the field. FND had very clearly arrived, seemingly out of the blue.

But this magical manifestation is of course an illusion, and in the following I will explore the history of its arrival and its conquest. In discussing 'FND' as opposed to 'hysteria', or 'conversion disorder', I am not indulging in mere pedantry: as any historian will tell you, one cannot simply transplant the concepts of the present into the past – and, make no mistake, the concept of FND involves profound changes from the past. So, in addition to celebrating its birth, I will discuss what the differences in FND are, and what difference they may make.

The Way It Was

It would be hard to exaggerate how dire the situation of conversion disorder was at the turn of the millennium. After a burst of popularity 100 years earlier, when some of the greatest minds in medicine (including leading neurologists and psychiatrists) shared a fascination for the disorder at the Salpêtrière Hospital in Paris (Fig. 1.1), it had sunk into a prolonged and profound decline. Psychiatry seemed to have found a way to ignore it entirely, announcing its disappearance [3]; Neurologists could only wish it had disappeared, left grappling with a problem they did not understand, and did not think was really theirs [4]. Clinicians of all specialties would describe the disorder with dislike, or worse, and question whether it was really all just deliberate feigning [5, 6]. For a disorder of such prevalence and such morbidity, clinical research was astonishingly sparse, with not one single properly-powered clinical trial ever conducted during the twentieth century, and a medley of treatment approaches proliferated, based on the lowest grades of evidence [7]. The public, meanwhile, was barely aware of its existence: though 'hysteria' remained a popular cultural motif, it was viewed almost

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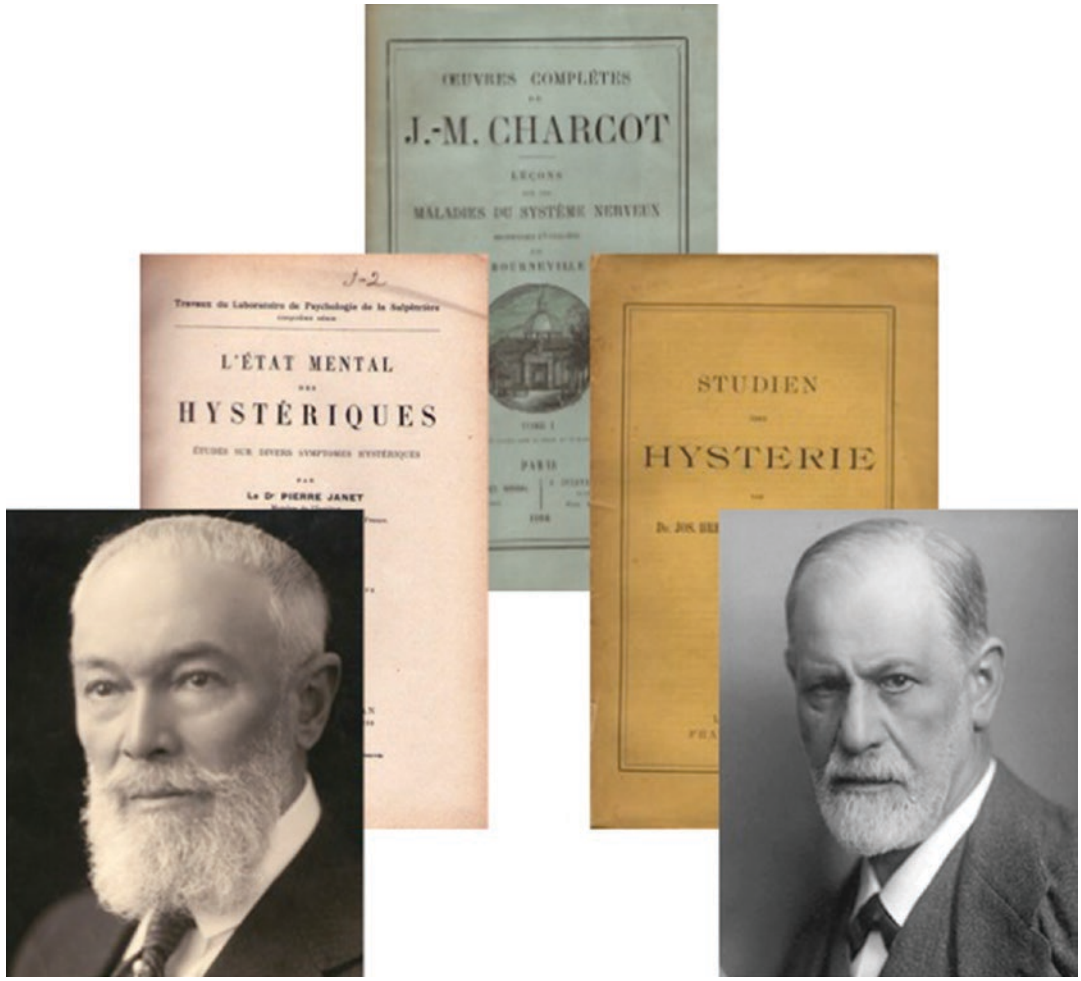

Fig. 1.1 Jean-Martin Charcot's *Lectures on the Diseases of the Nervous System* (1872), and two of the critical works of his students, Pierre Janet's *The Mental State of Hysterics* (1892) and Sigmund Freud's *Studies on Hysteria* (1895). (Images are available in the public domain)

exclusively as an historical artefact of medical misogyny [8] or Victorian absurdity [9]. The public shared in clinical suspicions of feigning [10], and patients newly diagnosed with conversion disorder would be so outraged they would go to any lengths to have the diagnosis overturned [11]. Unsurprisingly, there were no conversion disorder patient advocacy groups, and public demand for services in many places was non-existent [12].

Out With the Old…

The diagnostic criteria reflected these problems, and appeared to play a critical role. ICD-10 [13] and DSM-IV [14] shared the view that conversion disorder required the exclusion of both neurological and factitious alternatives, and the inclusion of a psychiatric explanation (see Box 1.1). There were real problems with each of these [15, 16].

Box 1.1 Comparison of the diagnostic criteria for Conversion Disorder in the Diagnostic and Statistical Manual (DSM), 4th and 5th editions

DSM-5 Conversion Disorder (Functional Neurological Symptom Disorder) - 2013

A. One or more symptoms of altered voluntary motor or sensory function.
B. Clinical findings provide evidence of incompatibility between the symptom and recognised neurological or medical conditions.
C. The symptom or deficit is not better explained by another medical or mental disorder.
D. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.

DSM-IV Conversion Disorder - 1994

A. One or more symptoms or deficits affecting voluntary motor or sensory function suggesting neurological or other general medical condition.
B. Psychological factors are judged to be associated to the symptom or deficit because conflicts or other stressors precede initiation or exacerbation of the symptom or deficit.
C. The patient is not feigning or intentionally producing his or her symptoms or deficits.
D. The symptom or deficit cannot, after appropriate investigation, be fully explained by a general medical condition, by the direct effects of a substance, or as a culturally sanctioned behaviour or experience.
E. The symptom is not limited to pain or to a disturbance in sexual functioning and is not better explained by another mental disorder.

The exclusion of neurological alternatives was not a problem in practice - despite the alarming reports of large-scale neurological misdiagnosis from the 1960s [17], overall, and particularly more recently, conversion disorder was not misdiagnosed more often than other disorders [18] - but it remained a serious problem in principle. For it meant that the neurologist would be required to exclude every alternative - known or unknown - something that appears impossible in principle, and a concerned patient could never be sure that something had not been overlooked, or had not yet been discovered. A patient's confidence in the diagnosis would depend on their confidence in their neurologist and second- or third- neurological opinions might seem only prudent.

The exclusion of feigning, or factitious disorder, was problematic both in principle and practice. In practice, it is usually impossible for a psychiatrist, or anyone else, to exclude feigning, particularly with neurological symptoms [19]. In principle, that this was an exclusion criterion for conversion disorder alone served to confirm all those suspicions that conversion disorder was uniquely close to feigning [20]. As far as a neurologist need be concerned [21], or anyone could prove, they might *all* be feigning [22].

The requirement for a psychiatric formulation was primarily a problem in practice. There were always patients in whom no plausible psychiatric explanation could be constructed, which meant the requirement was effectively ignored. Perhaps this was due to these patients' reluctance to disclose psychopathology [23], or clinician reluctance to explore it [24] – or perhaps because it was not present. But in almost every study, however it was studied, a relevant trauma history could not be identified in a significant minority of patients [25]. Though trauma was obviously still important, this also challenged the principle: it was difficult to support a monolithic, PTSD-type trauma model for conversion disorder when most studies appeared to provide evidence against it.

So, the diagnostic criteria brought the disorder into disrepute while being of little practical value. They drew heavily on psychodynamic ideas that, if not exactly disproven, were cer-

tainly old-fashioned, and perhaps no longer readily interpretable by most psychiatrists [9]. There was a once-in-a-generation opportunity for change, with both DSM-5 and ICD-11 in preparation.

… in With the New

For such 'old-fashioned' ideas to be so enduring suggests that replacing them would not be straightforward, however. They certainly had high-level endorsement, after all, and perhaps there were simply no better alternatives. That the neurological symptoms would not fit with identifiable pathology was established by Jean-Martin Charcot (Fig. 1.1), arguably the greatest neurologist of all time, in the nineteenth century [26]. The suspicions around feigning were part of the same discussion. Neurologists at the time were inclined to explain these apparently voluntary movements as malingering [27], since no better explanation was on offer [3]. The idea that these symptoms would be instead 'psychogenic' dates from the same era, most famously in Sigmund Freud's seminal "Studies on Hysteria", where he essentially argues for a post-traumatic aetiology – the novelty being that the traumas were 'emotional' ones (psychological conflicts), which became 'converted' into physical symptoms [28]. This construction survived largely intact for a century in conversion disorder, while all around it every other psychodynamic construction was being torn down [9]. Why it showed such unique longevity is unclear, given the ethos of the times was so strongly against aetiological criteria. Perhaps the loss of psychiatric interest allowed it to 'slip under the radar'. Or perhaps it just could not be understood psychiatrically without it. The symptoms, after all, were not psychiatric: what else was there about the disorder that would justify it being a psychiatric condition, if not its aetiology [29]? Suggestions for improving the criteria by simply removing the requirement for a psychiatric formulation [15] would have left it looking distinctly neurological, but a neurological syndrome for which no neurological explanation could be found.

There was a real reputational danger there, as outlined above: a criterion which depended on a neurologist's ability or zeal in finding an alternative explanation risked becoming the wastebasket for every patient the neurologist did not take seriously or whose symptoms they could not quite put together. The psychiatric aetiology requirement had protected against that, in theory, since the patient has to pass that test as well, and without it the risk would be clearly exposed. There was a solution, however: make the neurologist *prove* that the case was functional – make a 'positive diagnosis'. If they could identify features specific to the diagnosis that would mean they could demonstrate what it was, not just what it was not, and that risk would be removed.

Or nearly so. Unfortunately, distinguishing it from feigning would be as difficult as ever for most features of conversion disorder [30], but if excluding feigning was removed from the criteria, and feigning no longer considered as a likely alternative, the new criterion could still be considered 'positive' in regards to neurological alternatives. And this 'positive diagnosis' was to become the new principal diagnostic criterion in DSM-5 (Box 1.1).

But there were more changes in store. The name 'conversion disorder' still clearly harked back to Freud, and suggested a "one size fits all" psychodynamic process, even if the criteria did not. Neurologists had been using the term 'functional' to refer to these symptoms since at least the time of Charcot, who argued that any lesion invisible to his microscopes must be a 'functional' lesion of the underlying brain region [31]. Though the meaning of the term was opaque [32], it appeared to be a popular alternative with both patients and neurologists [33]. We, and others, suggested 'functional neurological symptoms' [15], or 'functional neurological symptom disorder' [16], which latter DSM-5 finally adopted. But in an early draft, perhaps following an earlier suggestion [34], it proposed 'functional neurological disorder' – a small difference, but one which shifted the emphasis from the *symptoms* being neurological to the *disorder* being neurological, a shift too far for most neurologists [20, 32]. Yet, this is the name that emerged trium-

phant, despite DSM-5's choice, and has become the standard bearer for the disorder: as of March 2020, a search on PubMed for "Functional Neurological Disorder" yields 55 references since 2019, while "Functional Neurological Symptom Disorder" yields only seven.

With regard to movement disorders more specifically, the subject of this book, similar changes were underway. Long known as 'psychogenic movement disorder', arguments were made on the same grounds [35] that they should instead be known as 'functional movement disorder' (FMD) [36]. Turning again to PubMed, 'functional movement disorder' first appears in 2012 [37], though a publication a year earlier uses the term with 'functional' in scare quotes [38]. But it has already come to dominate the field: since 2019, as of May 2020, "functional movement disorder" yields 25 references, and "psychogenic movement disorder" only six.

What Difference Does It Make?

Much has changed since the turn of the millennium. Clinical interest in the disorder has grown considerably – the first international conference (2017) open to all was hugely over-subscribed. A vibrant research community has developed and properly-powered clinical trials are finally underway. Public interest is increasing. Writing from Australia, FND has featured on 3 prime-time current affairs TV programs in the last few years, having never featured before. And patient attitudes are changing – most patients in my clinic have now actively sought an appointment in the clinic, instead of grudgingly accepting their neurologist's referral, or failing to attend once they understand what it's about – a position colleagues tell me is true more globally. The world of FND is a clearly different world to the world of Conversion Disorder. It's different in its appeal, to neurologists and patients in particular. We cannot be sure what caused these changes, and it seems unlikely that it was any one thing, but it is easy to see the new criteria and the new name as either causes or embodiments of this difference.

The embrace of the disorder by patient groups represents a tremendous advance for the field. As noted in the introduction, they have universally adopted FND into the names of their groups, which seems unlikely to be a coincidence. Patients have consistently expressed a preference for the term 'functional' over the more psychiatric sounding alternatives [33], but clearly the groups also preferred 'FND' over 'FNSD' - the advantage, other than perhaps euphony, presumably being that it more strongly implies the disorder is neurological. The stigma of a psychiatric diagnosis is unfortunately commonplace, and has very real consequences [39], but in the case of FND it appears bound up with the idea that the disorder is not real – is imaginary, or hallucinatory, or simply made up [10]. Though these attributions are not fixed [40], they are virtually absent from neurological disorders: a shift to a neurological disorder definitively makes the disorder seem more 'real'. Dropping the 'exclusion of feigning' criterion should also have helped in that regard. Patient groups also expressed concern with the psychogenic diagnostic criterion, and its implication that they had suffered a trauma, when not all reported such experiences [29] - so dropping that criterion should also be welcome. Finally, a 'positive' demonstration of FND could only help patient acceptance, alleviating doubts about the diagnosis being an excuse for medical incompetence or a way of dismissing unwelcome patients.

There were clear potential advantages for neurologists too. Conversion disorder was a source of great anxiety to neurologists, who found their patient encounters uniquely uncomfortable [4]. Their diagnostic approach of discovering inconsistency encouraged them to consider their patients as deceptive, while also involving the neurologist in a deception - of tricking their patients into revealing the truth [22]. But when it came to explaining their diagnosis, it involved them in discussions of such concepts as the subconscious, on which they felt unqualified; these could be interpreted as implying feigning, or dismissal, or incompetence, and greeted with anger [24]. The new criteria were in these areas a major advance. All mention of feigning was dropped, so that the 'positive' demonstration could be inter-

preted as of FND alone. Moreover, this meant the inconsistencies could be shown to the patient, removing any deception on the neurologists' part, and, in an inspired maneuver, turning these 'tricks' into ways of creating patient insight and support for the diagnosis [41]. Without the need to discuss some psychogenic aetiology, neurologists could stay within a zone more comfortable for them and the patient, so that their encounters need no longer be hostile [42].

So, Are the Problems All Over Now?

The above-discussed developments represent critical, and very welcome, changes. They have already transformed the disorder and have the potential to solve most of the problems that conversion disorder faced. But it would be naïve to think this most intractable of medical conundrums 'finally sorted', and these changes may yet lead to new problems.

Firstly, the thrust of the changes can be seen as rendering the disorder more neurological. The 'psychogenic' criterion is no longer essential; the diagnosis can now be made by neurologists alone; the diagnosis appears in the neurological section of ICD-11 as well [43]. The name FND, as we noted before, now appears to state that it is so. That initial alarm about the name has quietened. A steady stream of neuroimaging studies, while not, as a class, telling us anything we did not already know [44], make a neurological view of the disorder seem more comfortable, and more natural. This is no bad thing, inherently. The division of disorders into psychiatric and neurological has always been somewhat arbitrary, and many have suggested the two should be considered one. But it does encourage the view that the psychiatric aspects are secondary, and can perhaps be ignored in management. We are currently riding a wave of optimism that a neurological model will prevail [45], and that physical treatments may be enough, at least for most patients [46]. They may not, and the effect of that failure on 'FND' as a concept is hard to predict.

Secondly, not everyone will be happy. Among those who will be looking to the results of trials of transcranial magnetic stimulation (TMS) or physiotherapy most keenly may be the psychiatrists and psychologists who thought that 'conversion disorder' was fundamentally more valid, for all its flaws. Interviews with psychiatrists who specialize in the disorder suggests considerable discomfort with the new approach – that they certainly do not all think it is valid [47]. A survey of psychiatrists in the UK and Australia confirmed this, with respondents overwhelmingly reporting they think conversion disorder is psychogenic, and that a psychiatric formulation is not merely helpful for management, but essential to the diagnosis [48].

Thirdly, the new criteria will not diagnose the same patients as before. Any change in the sense of the criteria will inevitably affect their reference – the patients whom they do or do not fit. One obvious change from an aetiological criterion to a symptomatic criterion is that a larger number of seemingly straightforwardly neurological patients will now be included if they have any symptoms that are functional. Previously, the patient with a stroke who also developed a functional weakness need not have been diagnosed with conversion disorder if the psychiatrist did not formulate them as such: now, a 'positive sign' should be enough to make the diagnosis, as long as it is enough to cause distress. This view, of the symptom as sufficient, is not new [17], and not wrong, but it clearly broadens the number of potential FND patients enormously. Conversely, the diagnosis was 'positive' before, in theory, in terms of a 'positive psychiatric formulation', and removing this will exclude others. There will be patients who do have a positive psychiatric formulation but in whom no 'positive signs' are found, so would no longer be diagnosed with FND. Just how many of these there may be is not clear, as large-scale studies have not been conducted outside of specialist centres, but it could be a lot. One recent study, for example, suggested that most paediatric patients diagnosed with FND did not meet that criterion [49]. Some of the 'positive signs' are of doubtful discriminatory value [50], and certainty is typically rationed in neurological diagnoses: insisting on certainty, or on only the most valid signs, would doubtless lead to

a more tightly defined, but inevitably smaller group, particularly in FMD [51]. Inevitably, some patients with Conversion Disorder diagnoses are going to miss out by the new criteria. Again, this is not necessarily wrong, and we cannot conclude from this that the new criteria are more or less valid than the old, but one of the problems with the old psychogenic criterion was that it could not be fulfilled in practice, and was therefore routinely ignored. The danger for the new criterion is precisely the same. And for FND, like Conversion disorder before it, the danger is that it may therefore become dependent on an idea that nobody really believes any longer.

This chapter has focused heavily on DSM-5 in the preceding. It is of course only one iteration of that classification system, and only one of the systems in use. ICD-11 has trodden a different path, in nomenclature and criteria, based on the principle of dissociation espoused by the great rival theorist from the Salpêtrière, Pierre Janet (Fig. 1.1). If anyone needed reminding, Freud's is not the only way to formulate a patient with 'Dissociative Neurological Symptom Disorder', as ICD-11 would have it [43], and this history is not simply of the struggle between the psychodynamic and biological views that is the particularly American history of psychiatry [9]. That struggle may obscure the extent of agreement. As the later chapters in this book will show, life events and the biopsychosocial model remain at the forefront of modern FMD conceptualizations. And even DSM-5 is perhaps less definitive than its criteria may suggest: the title of the disorder retains both Conversion Disorder and FNSD (indeed, with FNSD in parentheses), and the accompanying text make very clear the importance of psychiatric formulation. Perhaps the pendulum, restrained for so long, swung further than anyone quite intended with this version. Perhaps the next iteration will more faithfully capture the diversity of the disorder, and of opinion. Perhaps it will restore psychiatric formulation to an equal footing with neurological signs, as factors which contribute to, but do not define, FND - and confirm FND as a core, interdisciplinary neuropsychiatric disorder [52].

Summary

- Conversion Disorder was profoundly neglected and stigmatized by clinicians, patients and the public 20 years ago.
- The diagnostic criteria were a major contributor to this, based on principles established in the nineteenth century.
- The criteria of DSM-IV made a trauma history essential, which not all patients had, and required the exclusion of neuropathology and of feigning, which was not usually possible.
- The revised criteria for DSM-5 dropped these requirements, instead requiring the diagnostician to make a rule-in diagnosis, by demonstrating incompatibility or finding 'positive signs'.
- The name 'functional neurological disorder' is not the official name in DSM-5, but was proposed in an early draft, and has been adopted by the field despite concern that it suggests the disorder is solely a neurological disorder.
- The name and the new criteria have proven very popular, and the diagnosis is experiencing a transformation in public awareness and patient acceptability.
- As we consider the present and future of FND, an appreciation of neurological signs and psychiatric formulation are likely both to be important in the assessment and management of this condition at the interface of neurology and psychiatry.

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2 Free Will, Emotions and Agency: Pathophysiology of Functional Movement Disorder

Mark Hallett

Introduction

In many movement disorders, the brain is hijacked by a neurodegenerative process or a structural lesion and does not function normally. In some neuropsychiatric conditions, the brain is so altered that it is not capable of normal function in certain domains. In a severe stroke, for example, so many neurons are lost that full recovery is impossible. In functional movement disorder (FMD) the brain also is not functioning normally, but, at least in some circumstances normal function is possible. Patients may present with weakness or involuntary movements, but these symptoms may only occur some of the time, and other instances the patient can be normal. Even during the physical examination, certain maneuvers can reverse the weakness or dampen the involuntary movements. Thus, the manifestations are a product of the disordered central nervous system function; hence the name of *functional movement disorder*. In group analyses comparing patients with FMD to those without, some subtle structural differences have been found which might represent one end of the normal distribution and, in any event, do not prevent normal function [1]. It is important to note that patients with FMD can have another disorder as well, and the FMD would refer to only those symptoms due to reversibly altered brain function.

Abnormal movements in FMD are perceived as involuntary; generally, patients will say that they have no control over the symptoms. There are two other entities that may look somewhat similar, with the patients saying that the symptoms are involuntary, but the individuals are feigning, and the movements are actually voluntary. These are factitious disorder, that has an underlying psychiatric disorder, and malingering, that does not [2]. In most medical practice settings, such patients are not commonly encountered. Better clinical tools and physiological tests are needed to help differentiate between these entities. This chapter will not discuss these other diagnoses further, and the pathophysiology of FMD is certainly distinct from factitious disorder and malingering.

The underpinning of any disorder rests on the two pillars of etiology and pathophysiology [3]. The etiology is the fundamental causes, and the pathophysiology (neural mechanisms), which is a direct result of the etiology, is what produces the symptoms. This chapter will focus on the pathophysiology, and the next chapter will focus on etiological factors. Suffice to say for here that the etiology is best understood to be multifactorial within the context of the biopsychosocial formulation. These factors can predispose,

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precipitate or perpetuate the FMD. Predisposing factors, such as early life stress, can influence the developing central nervous system and render a person less resilient to stress later-on in life [4].

Normal Function

In order to understand the pathophysiology, it is necessary to understand the normal processes for making movement [5] (Fig. 2.1). Movement is generated by muscles, and the muscles are under control of the spinal cord (or for the cranial muscles, the brainstem). The spinal cord receives controlling signals in the corticospinal tract and the reticulospinal tract. The rubrospinal tract, present in monkeys, has disappeared in humans. The reticulospinal tract is primitive and generally just deals with automatic and reflex movements. It also receives input from the cortex. The cortex is the main controller of voluntary movement. A small stroke involving just the corticospinal tract will produce a severe hemiplegia.

The motor part of the corticospinal tract originates in the primary motor cortex (M1, area 4) with contributions from the premotor cortex (PMC, lateral area 6) and the supplementary motor area (SMA, medial area 6). Contributions to the corticospinal tract from the post-central cortex go mostly to the dorsal horn of the spinal

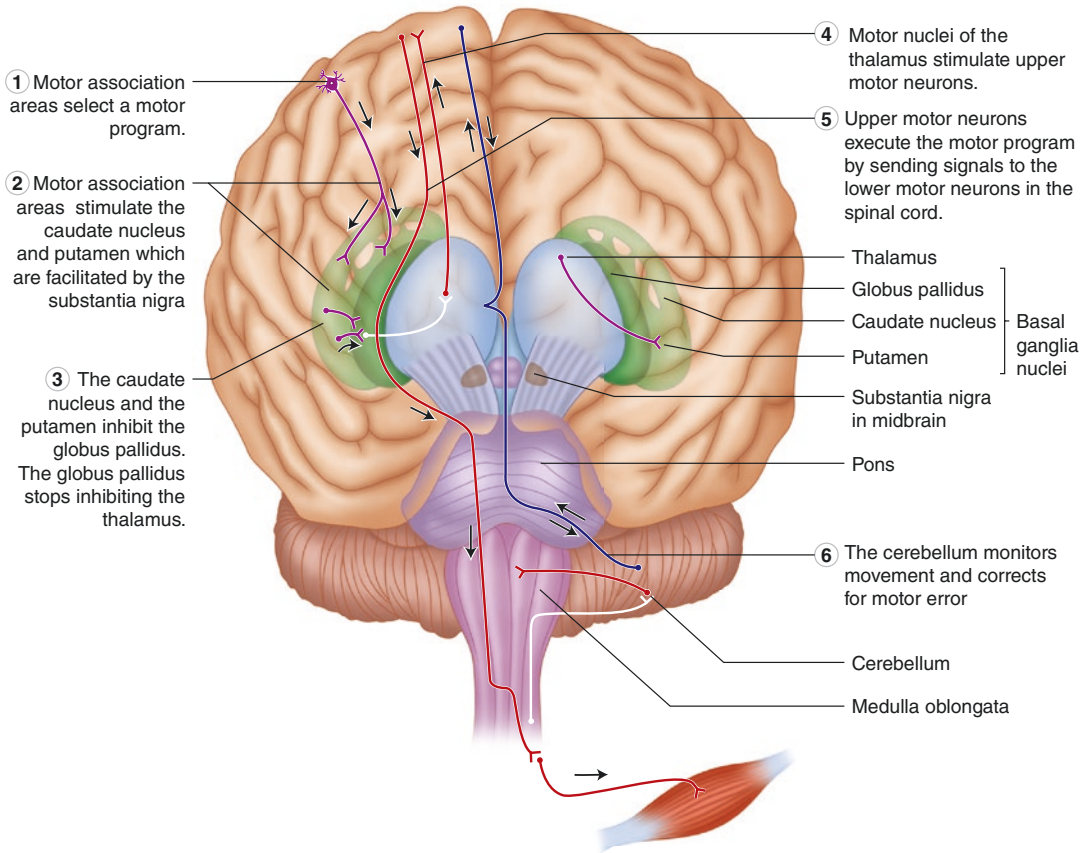

Fig. 2.1 Illustration of the parts and pathways of the brain that generate the movement command (see text for details). (Redrawn with modifications from Tortora and Derrickson [6])

cord and gate sensory information rather than adding to the motor command. What controls the motor cortex, controls movement. Such influences come from subcortical and cortical connections.

The two major subcortical networks involve the basal ganglia and the cerebellum. Both get important input from the cortex and then have their principal output back to the cortex via the thalamus. The loop involving the basal ganglia appears to help the cortex in controlling when and what will be moved, including both whole motor programs and the individual muscles that contribute to the motor programs. Abnormal function of the basal ganglia can lead to symptoms such as bradykinesia and dyskinesias. The loop involving the cerebellum appears to help the cortex in the precise control of the spatial-temporal features of movement, and the principal result of abnormality is ataxia. There are important connections between these loops and the functions may not be as distinct as was once thought. Also, relevant for aspects covered below, both the basal ganglia and cerebellum are involved in non-motor cognitive and affective processes.

The entire cortex provides direct or indirect input to the motor areas. Thus, the motor output at any one time reflects a complex calculation of an enormous number of competing influences. M1 receives strong input from PMC and SMA, and the latter two structures appear to synthesize much of this information for M1 as well as contribute to the corticospinal tract. As a generality, the posterior part of the brain provides information about the external world while the anterior part of the brain provides information about the internal world, the body and its regulation. The posterior part of the brain does receive all the sensory input, visual, somatosensory and auditory. Information is then routed from there to prefrontal and premotor areas where it can influence motor actions. The anterior part of the brain receives information about body function, homeostasis, emotions, and drives such as hunger, thirst, sex, reward, and therefore is likely to develop long and short-term goals. Information from the anterior part of the brain funnels to the anterior midline structures, the anterior/middle cingulate, pre-SMA, SMA and then to PMC and M1.

If you are hungry and felt rewarded recently in this situation when eating a chocolate bar, you might go toward the kitchen to find some chocolate. This is top down control [7]. Seeing the chocolate bar then triggers picking it up and eating it. If the chocolate bar is moldy, then this would discourage eating it. This is bottom up control. The details of skilled movements like picking up the chocolate bar are stored in parietal-motor pathways, perhaps because they are learned with trial and error feedback from the periphery [8].

The brain events associated with an internally triggered, top down movement, activation of the SMA and PMC, can be identified in the EEG as the Bereitschaftspotential (BP), also known, in the English translation, as the readiness potential (RP) [9]. This is a slowly rising negative potential over the anterior midline of the brain. It can be identified in the 1 to 2 seconds prior to the onset of movement.

Some brain events have correlates in consciousness. We do not understand how the contents of consciousness arise, but we are all aware of them. Each content element is a quale. Goals of behavior are appreciated as intentions, and the final command signal for making a movement is appreciated as willing. The quale of willing often has the sense that the action has been freely chosen by the person. This is the sense of free will [10]. When the brain generates goals and motor commands, there are feedforward signals to the inferior parietal lobule and temporoparietal junction (TPJ) in the posterior part of the brain. If these same regions get sensory input in accord with what was willed, this can be an indication that the brain caused that movement. This will be appreciated as the quale of agency, the person is the agent of the movement, and this is another aspect of the sense of free will. In one study of agency, the amount of control of a representation of a hand on a computer screen by the subject's hand was systematically varied. The right TPJ is an important node in the multimodal integration network that identifies the match of command

and result [11]. When there is a match, all is well, and the TPJ is relatively quiet; it becomes more active with mismatch. Other relevant areas in the network concerned with bodily awareness are the right anterior insula, right precuneus, and several regions in the frontal lobe [12].

The brain can focus on only one (or perhaps a few) thing at a time. This includes the qualia. The focusing mechanism is called attention, and this is a distributed system in the brain with at least two networks that have been defined, dorsal and ventral [13]. The dorsal attention network is bilateral including the intraparietal sulcus and the junction of the precentral and superior frontal sulcus (in the region of the frontal eye fields) and is said to be involved with voluntary or "top-down" goal-oriented attention. The ventral attention network is right lateralized and includes the TPJ and the ventral frontal cortex and is thought to be involved, bottom-up, with orienting to salient stimuli. In relation to salience, there is a separate, but related [14], salience network including the anterior insula and dorsal anterior cingulate as well as some subcortical structures including the amygdala, substantia nigra and periaqueductal gray. For an illustration of these and other brain circuits implicated in the pathophysiology of FMD, see Fig. 2.2.

There is another important aspect to brain function that appears relevant. The brain creates reality for each person on an individual basis, framed as its predictive capabilities. Perception

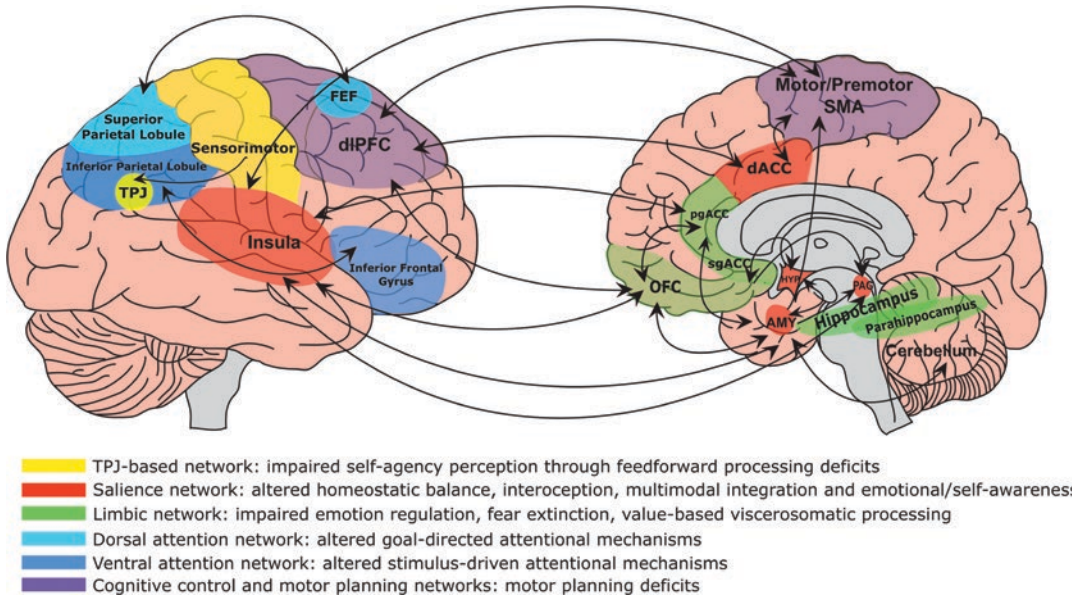

Fig. 2.2 Display of brain circuits (and related constructs) that are emerging as important in the pathophysiology of functional neurological disorder (FND). As depicted, FND is a multi-network disorder involving abnormalities within and across brain circuits implicated in self-agency, emotion processing, attention, homeostatic balance, interoception, multimodal integration, and cognitive/motor control among other functions. Circuits are described by their related dysfunction in the pathophysiology of FND. It should also be noted that several areas cut across multiple networks; for example, the dorsal anterior insula is most strongly interconnected with the dorsal anterior cingulate cortex (dACC), while the posterior insula receives afferent projections from the lamina I spinothalamocortical pathway and somatosensory cortices. Similarly, the amygdala is part of both the salience and limbic networks. Prefrontal brain regions are interconnected with striatal-thalamic areas (not shown), and these pathways should also be factored into the neural circuitry of FND. TPJ indicates temporoparietal junction, FEF frontal eye fields, dlPFC dorsolateral prefrontal cortex, pgACC perigenual anterior cingulate cortex, sgACC subgenual anterior cingulate cortex, OFC orbitofrontal cortex, SMA supplementary motor area, AMY amygdala, HYP hypothalamus, PAG periaqueductal gray. (From: Drane DL, Fani N, Hallett M, Khalsa SS, Perez DL, Roberts NA, A framework for understanding the pathophysiology of functional neurological disorder [15], published with permission)