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NEUROLOGICAL UPDATE

Functional neurological disorders: mechanisms and treatment

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Abstract Functional neurological disorders are common problems in neurologic practice. In the past decade there has been an increasing interest in this group of disorders both from a clinical as well as research point of view. In this review, we highlight some of the most salient and exciting publications from recent years focusing especially on new findings illuminating mechanism and studies examining treatment.

Keywords Functional neurological disorders · Conversion disorder · Psychogenic · Psychological treatment · Physiotherapy · Non-epileptic seizures · Dissociative attacks · Imaging · Neurophysiology

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Introduction

Patients who present with physical symptoms for which no disease pathology can be found are common in medical practice [1, 2]. Terminology includes psychogenic, non-organic, conversion disorder, dissociative motor or seizure disorder, but in this article we will refer to them as functional disorders. Compared with patients suffering from disease pathology, patients with functional disorders suffer from more distress and disability, more social isolation and are more likely to be receiving disability-related state financial benefits [3, 4]. Although functional disorders can remit spontaneously, the prognosis is often poor [5–7].

The last decade has seen a substantial increase in research interest across many areas of functional neurological disorders (FNDs). New insights into the underlying mechanisms of FNDs were gained and several well-conducted randomised controlled trials (RCTs) added much needed evidence base to treatment approaches. There have been developments in classification [8, 9] and consensus recommendations for treatment have been published to guide management and also to build a base for future research [10, 11]. This narrative review, part of the Update series for *Journal of Neurology*, summarises some key publications from recent years covering clinical presentations/diagnosis, pathophysiology and multi-disciplinary treatment.

Advances in diagnosis and classification of functional neurological disorders

There have been substantive changes in the classification of FNDs in recent years. These have been predicated in large part by a realisation that you can only reliably diagnose a functional disorder by demonstrating typical positive

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features on assessment [12, 13]. It cannot be diagnosed on the basis of associated psychosocial factors and neither is it a diagnosis of exclusion. A number of studies have established the reliability of existing signs such as Hoover's sign for functional limb weakness [14, 15] and the entrainment test for functional tremor [16]. A particularly useful study showed that such signs not only have reasonable sensitivity and specificity, but also acceptable inter-rater reliability [17]. Additional studies have described new specific clinical and laboratory features, such as accelerometry for functional head tremor, or using reaction time to assess functional dystonia which may assist in positively identifying the diagnosis [18–20]. In keeping with this shift in thinking the most recent version of the diagnostic and statistical manual of mental disorders (DSM), DSM-5, moved from the DSM-IV definition based on a psychological aetiology (largely following Breuer and Freud's conversion hypothesis) to one based on the positive identification of these typical presenting neurological signs. The DSM-5 criteria published in 2013 [21] removed both the requirement for a "recent psychological stressor" as well as the need to exclude feigning (which is not possible in practice). It replaced them with the need for positive physical signs to support the diagnosis (Box 1). The term "functional neurological symptom disorder" was added to the previous term "conversion disorder" [8, 22].

Box 1: DSM-5 criteria for conversion disorder (functional neurological symptom disorder)

1. One or more symptoms of altered voluntary motor or sensory function

2. Clinical findings provide evidence of incompatibility between the symptom and recognised neurological or medical conditions

3. The symptom or deficit is not better explained by another medical or mental disorder

4. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation

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The DSM-5 definition of functional disorders therefore has been aligned with the way that neurologists have always approached diagnosis. The paradox is that for other functional disorders the classification has moved in the opposite direction with the creation of the new category of somatic symptom disorder (Box 2). This describes physical symptoms which are distressing and causing disruption to daily life. There are two new features, however, compared to the previous DSM-IV somatoform disorder categories. The first is that the symptoms can be explained both by

disease or unexplained by disease (i.e. functional), so it does not matter what the underlying diagnosis is. Secondly, there is a new emphasis on psychological and behavioural response to being ill. In this system a patient with epilepsy, depression and an unusually high rate of contact with the Emergency Department would have the same diagnosis as a patient with dissociative (non-epileptic) attacks and the same additional features. In addition, it is not clear whether a wheelchair patient with chronic fatigue and pain ought to be seen as having 'disproportionate' behaviour to their diagnosis or proportionate response to a disabling illness. It seems likely that these criteria will incorrectly be used as a short hand for patients previously labelled as somatisation disorder and without encouraging psychiatrists to seek clarity in establishing the underlying diagnosis.

Box 2: DSM-5 criteria for somatic symptom disorder

A. One or more somatic symptoms that are distressing or result in significant disruption of daily life

B. Excessive thoughts, feelings, behaviours related to the somatic symptoms or associated health concerns as manifested by at least one of the following: (1) disproportionate and persistent thoughts about the seriousness of one's symptoms. (2) Persistently high level of anxiety about health or symptoms. (3) Excessive time and energy devoted to these symptoms or health concerns

C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months)

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The International Classification of Diseases 11th Revision (ICD-11) is due by 2017. In its current beta draft (http://apps.who.int/classifications/icd11/browse/l-m/en) functional disorders are found as a separate category within the neurologic section for the first time. It includes subcategories with separate codes for specific disorders such as functional tremor or functional speech disorder. Some of the anticipated benefits of these changes are that neurologists will be more comfortable using the new criteria and gain confidence in the diagnosis of functional disorders. They will also hopefully improve collaboration between neurology and psychiatry [9].

New phenotypes of functional disorders

Increasing awareness of functional disorders has led to the description of new phenotypes, particularly in the field of movement disorders. Propriospinal myoclonus describes flexor arrhythmic jerks of the trunk, hips and knees which

increase when the patient is supine and are often stimulus sensitive [23]. Previously, this was considered to be an organic movement disorder, but a recent examination of two large case series (n = 179) has suggested that the majority of these patients (57 %) may have a functional movement disorder (FMD) [24]. The diagnosis of a functional movement disorder was based on acute onset, distractibility and co-occurrence with functional somatic disorders and reinforced by the discovery of a bereitschaftspotential (BP) (pre-movement potential seen in voluntary movement) on jerk-locked EEG back averaging [24, 25]. The authors emphasised that an absence of BP does not exclude an FMD and that imaging is still required to detect secondary causes such as cervical cord tumour, infection and compression which were reported in 7 % of cases [24].

Functional facial movement disorders were recognised in the past but a landmark study of 61 patients from seven tertiary movement disorder centres has re-established this clinical entity which in our experience is relatively common [26]. The key feature of a functional facial movement disorder is unilateral facial contraction, usually of lower lip downward and ipsilateral jaw (Fig. 1). This may give the appearance of facial weakness. There may be ipsilateral tongue deviation as well. Contraction of orbicularis oculis muscle on the same side leading to depression of the eyebrow is also common. In organic blepharospasm the eyebrow is often raised in an attempt to overcome the spasm and the duration of contraction is much shorter. Comorbid functional neurological symptoms, distractibility, rapid onset or spontaneous remissions and response to suggestion or psychotherapy are additional features [27]. Electrophysiological testing such as the R2 blink reflex recovery cycle in patients with psychogenic blepharospasm [28] may be helpful in positively diagnosing functional facial movement disorders; however, BP appears to be unhelpful [26].

Two recent studies have suggested that it is possible to identify patients with functional tic disorders [29, 30]. Such a differentiation is a challenge as tics share many features of FMDs such as suggestibility, distractibility and suppressibility. The authors suggested that adult onset, an inability to suppress the tics, lack of pre-monitory sensations, lack of associated pali-, echo- and copro-phenomena, co-existence with other FMDs and dissociative/non-epileptic seizures rather than the expected OCD or ADHD are associated with functional rather than organic tics.

Studies of patients presenting with cauda equina symptoms such as leg weakness and urinary retention have found a high rate of no detectable structural lesion [31, 32] with some patients having evidence of functional disorder. This may have some parallels with the high frequency of functional disorder recently reported in patients with Fowler's syndrome of chronic urinary retention (24 %) [33].
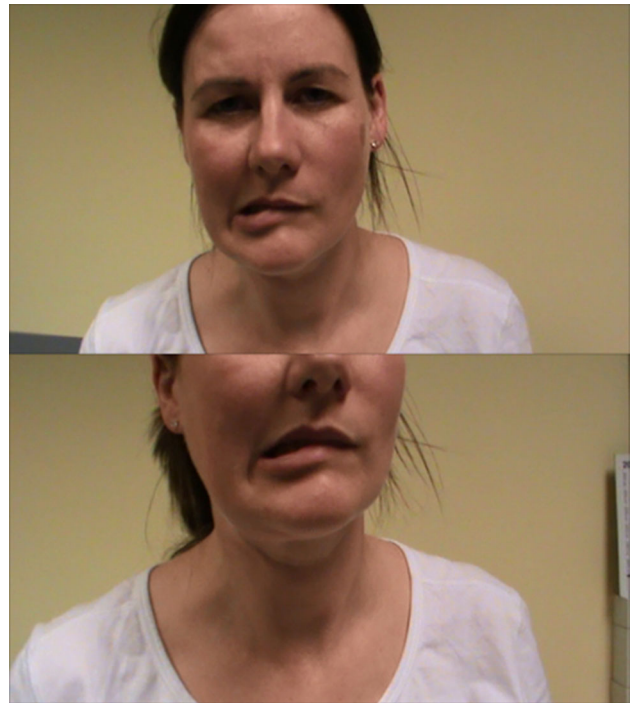

Fig. 1 Functional facial spasm/dystonia is characterised by contraction of platysma and/or orbicularis oculis. Ipsilateral jaw and tongue deviation is common. (Reproduced with permission from Stone [87].)

Finally, palatal tremor/myoclonus, long thought to be a specific sign of organic pathology in the brainstem (triangle of Guillain–Mollaret) has been found in some patients, to be caused by a functional disorder. A group from Queen Square retrospectively analysed 17 cases of palatal tremor and found that 10 had a functional/psychogenic disorder based on the presence of entrainment of the tremor and distractibility with ballistic tasks [34].

Dissociative (non-epileptic) seizures

Research into dissociative (psychogenic) non-epileptic seizures (NES) has escalated, with studies in recent years consolidating knowledge of seizure semiology [35, 36], progressing understanding of subjective seizure experience [37, 38], and exploring brain networks [39, 40].

A meta-analysis of studies looking at objective signs mostly captured during video EEG (vEEG) highlighted long duration events, fluctuation of course, side-to-side head or body movements, closed eyes during episode, ictal crying and memory recall are useful in distinguishing NES from generalised epileptic seizures (ES) and signs such as onset from (EEG confirmed) sleep, and stertorous breathing favoured ES over NES. However, numerous other factors such as gradual onset, tongue biting or urinary incontinence were found to have insufficient supporting

evidence to use to distinguish between NES and ES [35]. An important study of witnesses of vEEG-confirmed cases highlighted how commonly they report features 'sometimes', 'often' or 'always' that might be thought to be typical of ES such as "frothing at the mouth" (19 %), "head turning" (35 %), "lip smacking or chewing" (49 %) and "eyes open" (66 %)—clinicians beware [38]!

An international consensus group collaboration report from the International League Against Epilepsy aimed to 'develop clear guidance on standards for the diagnosis of PNES' [13]. They distinguished four diagnostic levels: possible, non-clinician witnessed event or self-reported, and no epileptiform activity in routine or sleep-deprived interictal EEG; probable, clinician witnessed event with signs typical of NES (as outlined above), with no epileptiform activity found in routine or sleep-deprived interictal EEG; clinically established clinician witnessed event with signs typical of NES, with no epileptiform EEG activity; and documented event with no epileptiform activity immediately before, during or after ictus captured on ictal vEEG, with typical NES semiology. Studies have also attempted to define seizure subtypes, but the clinical utility of this remains uncertain [36, 41, 42].

Research on the subjective symptoms of attacks has only appeared in the last 10 years or so. Increasingly, the evidence supports a hypothesis that in many patients, NES is a dissociative response to a state of arousal similar to panic, maintained as a conditioned response [37, 38, 43]. In this framework, the seizure itself is seen as a dissociative event, whether or not the patient experiences symptoms of depersonalisation or derealisation. In a large study, more than 80 % of patients with NES ($n = 224$) were found to have $\geq$4 panic attack symptoms, vs. 35 % of patients with ES ($n = 130$) [43], and as the number of symptoms increased, the specificity improved, although sensitivity reduced concurrently. Another study of 100 vEEG-confirmed patients also reported self-reported fear and dissociation ("In my attacks I am conscious but I can't react to things") during seizures in over 75 % of patients [38]. A case series of 11 patients described 'wilful submission' to the non-epileptic attacks in order to end these unpleasant prodromal symptoms [37]. This perhaps helps to explain why patients with NES are so often reluctant to discuss seizure symptoms and experiences as shown so elegantly in studies of conversation analysis comparing NES to epilepsy [44].

Although the EEG should not have seizure discharges in NES, non-specific EEG abnormalities may be found 1.8 times as often in NES patients as compared to healthy controls [39]. Recent studies have analysed the EEG quantitatively to explore functional brain networks in NES patients [40]. These highlighted weak local connectedness, excessive rigidity of networks, and an imbalance between local and global connectivity which the authors hypothesised might "predispose to and/or facilitate the occurrence of PNES episodes" [40].

Imaging and neurophysiological experiments in functional neurological disorders

Progress has been made in understanding the mechanism of functional neurological disorders. Edwards et al. proposed an updated model based on earlier models in which belief plays a central role in maintaining an expectation of a sensory or motor outcome which in turn is the subject of abnormally focused attention [45].

In a study by Van der Salm et al. 86 % of patients with functional jerks showed a bereitschaftspotential before an involuntary jerk [46]. Remarkably, 17 out of 29 patients did not show such a bereitschaftspotential before the intended voluntary movement, which indicates that perception of the voluntariness of movements might be part of the problem. In line with this idea, the study of Kranick et al. [47] found a reduced 'intentional binding effect' and in a classical Libet experiment, patients partly lacked the feeling of intention before movement [48]. Also, in an experiment with a force matching task, patients did not overestimate the force required when pressing directly on their own finger as healthy controls did, which suggests sensory attenuation, a measure for motor agency, is impaired [18].

Exploring further the role of attention and self-agency, Parees et al. conducted a series of experiments testing voluntary movements under full conscious control and various degrees of automatic control [49]. They showed that patients with functional neurological disorders performed particularly poorly in tasks that were highly predictable, as these situations allow a switch to an "attentive self-focused action-monitoring mode". The idea that attention plays a major role in the presence of symptoms was illustrated by the finding that patients reported having functional tremor 83.5 % of the day, while the tremor was only registered 3.9 % of the day by actigraphy [50] (Fig. 2). This suggests that at least some patients have the symptoms only when they attend to them.

Several recent functional imaging studies have explored the neural basis of functional motor disorders. Different paradigms and varying inclusion criteria mean that many different activity patterns have been implicated covering almost the entire brain [51]. However, individual studies are of interest. In a study by Schrag et al. [52], a pattern of activation involving basal ganglia, the cerebellum was found in functional dystonia in contrast to 'organic' dystonia where there was activation of primary motor cortex.

Two fMRI studies in motor symptoms, one investigating escape life events [53] and one with emotional stimuli [54]
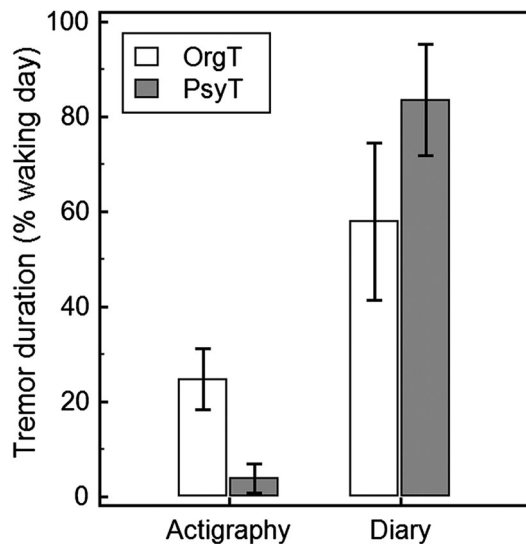
Fig. 2 Tremor duration as percentage of the waking day [mean (SD)], as recorded in self-report diaries and by actigraphy, in patients with organic tremor (OrgT) and psychogenic tremor (PsyT). (Reproduced with permission from Parees et al. [50].)
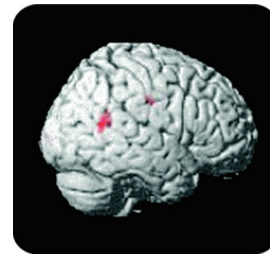

Fig. 3 fMRI study of eight patients comparing spontaneous functional tremor with a voluntary tremor made in the same limb. Hypoactivation of the right temporoparietal junction may be in keeping with a problem in the 'feed-forward' network that gives voluntary movement a sense of agency. (Reproduced with permission from Voon et al. [59].)

found functional connectivity between the SMA (a motor region) and the amygdala. Another fMRI study using emotional stimuli (fearful and sad faces compared to neutral faces), also found enhanced activity in the amygdala, in patients with motor conversion disorder compared to healthy controls. This effect remained significant after correction for a higher level of anxiety in the patient group [55]. A fourth study of motor symptoms showed altered activity in the amygdala as well, but in a non-emotional, action selection task [56].

Another common theme has been altered activity in the insula, a complex multi-functional region amongst others involved in emotion regulation and self-awareness [53, 56–58]. Altered agency and motor planning were suggested as explanations for changes in frontal, parietal and insular regions, for example in the action selection task by Voon et al. [56] and a within-subject comparison of functional tremor and mimicked tremor [59]. In that last study, a comparison of functional tremor and simulated tremor within the same individuals highlighted hypoactivation of the right temporoparietal junction suggesting that although the movements were generated by voluntary motor pathways, the patients may lack agency in feeling that they had intended the movements (Fig. 3).

Resting state fMRI and FDG-PET studies in non-epileptic attacks found stronger connectivity between the precentral sulcus, intraparietal sulcus, insula, and supramarginal gyrus [60], more general frontal–parietal changes [61] and hypometabolism in right inferior parietal and anterior cingulate regions compared to controls (Fig. 3) [62].

Summarising these findings is difficult, but an alteration of areas involved in planning, execution and interpretation/attribution of movement, moderated by those areas involved in emotional regulation does fit very broadly with current models. There are undoubtedly many challenges involved in examining the neural basis of these symptoms. Intrasubject studies 'before' and 'after' treatment [59] would avoid the problem of comorbidity that limits interpretation of many of these studies. Paradigms that directly assess the symptom involved also lessen the risk of finding trait or comorbid abnormalities that are not specific to the functional symptom.

Physiotherapy for functional motor disorders

The last few years have brought exciting developments in evidence for physiotherapy in functional motor disorders which previously consisted of sparse if generally positive case series [63]. A retrospective study described 60 patients who received a 5-day rehabilitation programme with a multi-disciplinary team (MDT) [64]. Patients received a positive explanation of their functional movement disorder, but treatment was largely physical in nature. 69 % were "markedly improved" immediately after this short treatment even though pre-treatment symptom duration was 17 months. Treatment effects were sustained at 25 months. A similar inpatient approach was used in the first randomised trial of physiotherapy [65]. This study used a delayed treatment design to investigate a 3-week physical and sports therapy inpatient programme with 1-year follow-up for patients with functional gait disorder. There was a significant seven-point improvement in a 15-point scale in treated patients that was sustained at 1-year follow-up even with a mean duration of 9 months at the outset of the study (Fig. 4). Positive explanation and consistent approach by all the team was an integral part of this study.
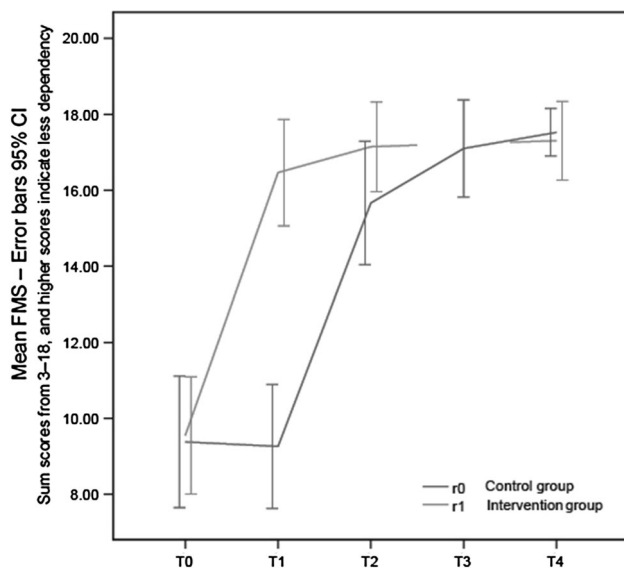
Fig. 4 A RCT of immediate and delayed physiotherapy for functional gait disorder ($n = 60$) showed an increase in Functional Mobility Scale (FMS) scores up to near normal (scale ranges from 3 to 18) even though the mean duration of symptoms prior to treatment was 9 months. The benefit was seen at the end of treatment (T2 and T3) and sustained at 1 year (T4). (Reproduced with permission from Jordbru et al. [65].)

Most recently a group of physiotherapists, neurologists and psychiatrists (including some of the authors of this paper) collaborated to produce detailed consensus recommendations on the nature of the content and intensity of physiotherapy for functional motor disorders [10] to give a base from which therapists can start comparable studies. This had been lacking in literature reviewed to date. The approach includes reducing abnormal self-directed attention by distraction techniques and breaking down learned patterns of abnormal movement to then retrain normal patterns, but recognises the importance of education and entwining psychological approaches.

Nielsen et al. published outcomes of a 5-day intervention using these techniques in 47 patients with functional motor disorders who had experienced symptoms for over 5 years [66]. 65 % of patients were "very much improved or much improved" at the end of treatment and 55 % at 3-month follow-up, remarkable outcomes given that more than 55 % of his cohort of patients had "poor prognosis" [6].

Three studies of inpatient treatment of patients with chronic and severe functional disorders also offer some hope that an MDT approach, which combines physical and psychological treatment, commonly, cognitive behavioural therapy (CBT), can be effective for some patients [64, 67, 68] even those with longstanding and severe disability. A prospective study of 66 patients undergoing this treatment reported improvements in two-thirds, which appeared to be

reasonably sustained over 1 year. HONOS (Health of the Nation Outcome Scale) was most sensitive at predicting outcome. Occupational therapy was especially popular among patients [67]. This data, most of it from the last 2 years, provides an exciting platform from which to build more robust evidence for the role of physiotherapy and MDT treatment.

Psychological treatments

In the last few years, randomised controlled trial (RCT) data has emerged for the first time for psychological therapy in patients with functional neurological disorders. In patients with non-epileptic seizures a UK pilot RCT of 66 patients compared the effect of cognitive behavioural therapy (CBT) plus standard medical care (SMC) with SMC alone [69]. 12-session manualised CBT comprised: (1) techniques for interrupting behavioural/physiological/cognitive warning signals at the start of a seizure; (2) enabling patients to engage in activities they were avoiding and (3) tackling unhelpful thoughts which may have influence on seizure control, self-esteem, mood and anxiety. An intention-to-treat analysis found a greater reduction in seizure frequency for the intervention (group × time interaction $p < 0.0001$) with a number needed to treat for seizure freedom of 5.1. This benefit was maintained at the 6-month follow-up. Limitations of the study included 27 patients who refused to participate, but overall the study was encouraging and has led to a large multicentre trial currently ongoing (http://www.codestrial.org). Another US pilot multisite RCT compared four treatments for non-epileptic seizures but unfortunately, with only 38 patients in total, the study was not powered to detect "between-group" differences [70]. After 16 weeks all groups had fewer seizures: CBT informed psychotherapy (CBT-ip) treatment arm (51 %); combined treatment groups (59 %); patients on Sertraline (27 %) and treatment as usual (34 %). An analysis of secondary measures such as depression, anxiety, quality of life and global functioning suggested a trend towards benefits in the CBT-ip group. The study provides further support for larger RCTs of CBT.

Evidence-based psychological treatment is even more scarce in functional movement disorders (FMD) [71]. A recently published 6 months randomised immediate vs. delayed treatment trial, based on psychodynamic psychotherapy, could not find a significant main effect of treatment assignment but was again underpowered [72]. Only 40 % of eligible patients agreed to participate and a further 35 % of randomised patients dropped out leaving only 15 patients in the trial. A RCT of the effect of liaison psychiatry input on patients with a wide spectrum of functional neurological disorders in 23 patients found

promising results both on symptoms and hospital visits encouraging more research and investment in interdisciplinary working [73]. Finally, an RCT of brief (4 × 30 min) guided self-help based on CBT in 127 patients with a range of functional symptoms and disorders in a neurology outpatient setting showed benefits (odds ratio 2.4 for improved outcome $p = 0.02$) [74]. Given the numbers of patients with these disorders, brief and cost-effective interventions are required to properly support neurology services. These RCTs highlight some of the challenges in gathering evidence in this patient group, especially around recruitment and drop outs for psychological therapy. Nonetheless they present an important step towards a future aim of multicentre trials of manualised therapy involving the large numbers of patients that we know attend neurology services with functional disorders [75].

A recent secondary analysis of the PACE trial [76] looked at adaptive pacing, graded activity and cognitive behaviour therapy compared to usual care in patients with chronic fatigue syndrome (CFS) [77]. Although CFS does not belong within the DSM-5 definition of functional neurological disorder this study offers insights relevant to FNDs. In the study both graded activity and CBT led to significant improvement in physical function with the main effect being mediated through the change of fearful beliefs about engaging in activity. This could help explain why physiotherapy as well as psychotherapy can be beneficial in patients with functional movement disorders. The key point seems to be to induce normal limb movement again and there may be more than one way to do that.

TENS, TMS, biofeedback and sedation

A variety of other physical treatments have been introduced or re-evaluated as potentially helpful in the treatment of functional neurological disorders. Transcutaneous electrical nerve stimulation (TENS) was trialled with some promise in an uncontrolled series of 19 patients with functional movement disorders, using a stimulation intensity sufficient to produce a 'tingling sensation' without muscle twitching or pain [78]. The portability and focus of TENS may make it a useful tool as part of a physiotherapy programme, especially for patients with sensory symptoms such as numbness or allodynia, but cutaneous discomfort may limit use at intensities high enough to generate muscle movement [10].

Transcranial magnetic stimulation (TMS) has been the subject of much recent interest as a potential treatment for functional movement disorders. Applied at supra-motor-threshold intensities to the contralateral motor cortex, TMS can produce jerky movements in a functionally weak, dystonic or tremulous limb. A case report of a patient improving with TMS was first published in 1992 and subsequently several studies have investigated TMS as a treatment for functional movement disorders [79, 80]. Chastan and Parain treated 70 patients with functional limb weakness with a single session of 15 min TMS with a success rate of 89 %. More than half had duration less than 10 days and 49 patients were adolescents or children so many of these may have improved without treatment and there was no systematic data on longer term outcome [81]. Garcin et al. report that 75 % of their 24 patients with functional movement disorders (with a longer median duration of 2.8 years) had sustained benefit from TMS given in a rehabilitative context [82]. It remains to be seen whether these results can be reproduced in other centres. The first small crossover RCT of rTMS using sub motor threshold treatment in 11 patients did not demonstrate any subjective clinical improvement suggesting caution is warranted before endorsing this technique [83].

The mechanism of action of TMS in functional movement disorders is uncertain. Despite described treatments being insufficient to induce neural after-effects, speculation about biological mechanisms is common. Most authors also acknowledge that suggestion and placebo factors may play a part. Another possibility is that by demonstrating to the patient the possibility of normal movement TMS can alter pathologically precise prior beliefs allowing recovery of normal function [45].

A novel study of biofeedback treatment for functional tremor used tactile and auditory external cueing and real-time visual feedback to help 'retrain' their tremor frequency with promising results [84].

Demonstration of reversibility and of normal movement can also be achieved using non-electrical clinical techniques, such as Hoover's sign of functional weakness [85]. In situations where it is not possible to demonstrate reversibility in clinic, therapeutic sedation may be helpful; a standardised anaesthetic and physician technique brought about sustained cure or major improvement in five out of a case series of 11 patients with functional neurological symptoms who had a median symptom duration of 14 months [86].

Conclusions

Functional neurological disorders cause significant distress and disability to patients, but unfortunately they are often not addressed well in the current medical system. In the last few years, a better understanding of new phenotypes, underlying concepts and treatment approaches towards these symptoms from clinical studies, experiments and imaging has led to new directions within the field. The

models that we use to think about the mechanism and aetiology of these problems should now incorporate biological as well as psychological factors. The emphasis in classification on making a diagnosis using positive diagnostic criteria highlights the central role of the neurologist in providing transparent explanation, information and triage of treatment. Data from treatment studies should provide encouragement to neurologists, psychiatrists, physiotherapists and others to work together, perhaps sometimes in new ways, to improve the health and prognosis of patients with functional neurological disorders.

Compliance with ethical standards

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