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Medication in Functional Neurological Disorder: Harm, Help or Adjunct to Psychotherapy?

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Abstract

People with functional neurological disorder (FND) are prescribed many drugs, yet treatment reviews focus on psychotherapy and physiotherapy and give medication little attention. This narrative review, based on a structured search of PubMed and Embase, asks three questions from a psychiatric perspective: when medication harms people with FND, what it offers for comorbid psychiatric conditions or functional symptoms, and how it should sit alongside psychotherapy. Findings are synthesised narratively; no formal risk-of-bias assessment or certainty grading was performed.

Harm is the best-documented effect of medication in FND. Antiseizure drugs are often given for misdiagnosed functional seizures, and each failed drug trial is associated with a longer delay to the correct diagnosis. Benzodiazepine prescribing is common and tends to continue after diagnosis. In emergencies, prolonged functional seizures treated as status epilepticus are associated with escalating doses, respiratory depression and intubation. In case reports, some drugs, including levetiracetam and interferon, were temporally associated with the onset or worsening of functional symptoms. In small studies, withdrawal of antiseizure drugs after a confident diagnosis appeared safe and may itself have reduced attacks.

Evidence of benefit is weak. Antidepressants appear to help mainly when depression or anxiety drives the presentation, and there is insufficient controlled evidence to show whether any drug adds to psychotherapy for the functional symptoms themselves. Benzodiazepines have performed no better than psychological approaches in acute settings, and placebo responses are large. Evidence for antipsychotics, drug-assisted interviews, electroconvulsive therapy, ketamine and psychedelics comes from small or uncontrolled studies.

There is no good evidence that medication directly treats FND, and insufficient evidence to show whether adding it to psychotherapy improves functional symptoms. Its main roles are as a source of harm to be reviewed and withdrawn, and as support in treating comorbid depression, anxiety and post-traumatic stress disorder alongside psychological and physiotherapy-based treatment. We propose a stepwise approach: confirm the diagnosis, review and withdraw unnecessary drugs, plan emergency care, treat comorbidity, and keep psychotherapy and physiotherapy central. Randomised trials of antidepressants added to psychotherapy, and of deprescribing beyond antiseizure drugs, are research priorities.

Categories: Neurology, Psychiatry, Psychology
Keywords: antidepressants, benzodiazepines, conversion disorder, deprescribing, functional neurological disorder, functional seizures, psychotherapy

Introduction And Background

Functional neurological disorder (FND) is a common cause of disability in neurology and liaison psychiatry. In a Scottish study of 3,781 new neurology outpatients, functional and psychological symptoms were the second most common diagnostic category, at 16% [1]. FND describes genuine neurological symptoms, such as seizures, weakness or abnormal movements, that arise from a disorder of nervous system functioning rather than from structural disease. Its main presentations are functional (dissociative) seizures, functional movement disorders, functional weakness, functional sensory and cognitive symptoms and functional dizziness. Functional seizures can closely resemble epileptic seizures, and the two can coexist in the same person. Diagnosis rests on positive clinical features that show the symptoms are incompatible with recognised neurological disease, such as Hoover's sign in functional weakness, and, for seizures, on the recorded attack, ideally with video-EEG. Since the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), diagnosis no longer requires an identifiable psychological stressor [2]. Psychotherapy and specialist physiotherapy are the treatments with the best support. The 2026 American Academy of Neurology (AAN) guideline puts psychotherapy at the centre of care for functional seizures [3], and a 2023 scoping review found that most treatment research concerns psychological and physical therapies [4].

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Medication sits awkwardly in this picture. People with FND are prescribed many drugs. In 322 people with motor FND in South London, those on medication took a mean of 4.7 agents, compared with 2.9 in psychiatric controls [5]. Much of this prescribing follows a mistaken diagnosis of epilepsy, and some of it may cause harm through adverse effects, dependence, emergency escalation and delay to the correct diagnosis and treatment. Yet depression, anxiety and post-traumatic stress disorder (PTSD) are common in FND, and treating them is a routine part of psychiatric care. Medication therefore matters for three reasons: inappropriate drugs can cause harm, comorbid psychiatric disorders often need drug treatment, and it is unclear whether medication helps, hinders or has no effect on psychological treatment.

Recent overviews give medication little space. A 2026 review of FND treatment across subtypes devoted two paragraphs to pharmacology [6], and the scoping review by Varley and colleagues listed medication studies without synthesising harms or withdrawal [4]. Large trials of cognitive behavioural therapy (CBT) and physiotherapy have reported mixed primary outcomes [7,8], and only one randomised trial has tested medication alongside psychotherapy [9].

The objective of this narrative review is to summarise the evidence on medication in FND for psychiatrists and neurologists, covering all FND phenotypes but noting where evidence is limited to functional seizures. It asks three questions from a psychiatric perspective. First, when does medication harm people with FND, and how can unnecessary drugs be withdrawn safely? Second, what is the evidence for drugs that treat comorbid psychiatric conditions or the functional symptoms themselves? Third, how should medication sit alongside psychotherapy? We end with a stepwise approach to prescribing and a set of research priorities.

Review

Search approach

This is a narrative review informed by a structured literature search; it is not a systematic review. We searched PubMed and Embase on 22 October 2026, and the full search strategy is given in the Appendices. The search combined two concept blocks: FND under its current and older names, and medication in its broadest sense, including drug classes, named drugs, polypharmacy, withdrawal and intubation. No date or age limits were applied, so older and paediatric studies remained visible, and records were limited to English. The first author reviewed the retrieved titles and abstracts, removed duplicates between databases and read the full texts of relevant studies, and two further authors read the selected studies and checked the extracted findings against the original papers. Studies were chosen for their relevance to the review's three questions rather than through formal eligibility criteria, and guidelines and earlier reviews were used as background. Reference lists of key reviews were checked for further studies. The review did not use a registered protocol, formal quality grading or meta-analysis, and it is not reported to PRISMA standards.

We defined the scope of the review as follows. The population was people of any age with FND, including functional seizures, functional movement disorders, functional weakness, functional sensory and cognitive symptoms, and persistent postural-perceptual dizziness (PPPD), a functional vestibular disorder that was included in the search terms from the outset because of its clinical overlap with FND. Older diagnostic labels, such as conversion disorder, hysteria and pseudoseizures, were accepted. Interventions and exposures were any medication, drug-assisted procedure, drug withdrawal or emergency drug treatment, with any comparator or none. Outcomes were core functional symptoms (for example, attack frequency, motor severity and disability), psychiatric symptoms, adverse events and harms from healthcare. We included randomised and non-randomised trials, cohort studies, case series and case reports; case reports were used mainly to describe harms or drugs that had not been studied in any other way. Records were excluded if they did not concern FND, did not report on medication, were conference abstracts or were not in English.

Duplicate records between the two databases were removed by matching titles, authors and years. Some cited studies came from the same research groups, such as the two Glasgow studies of antiseizure drug withdrawal and the two sertraline trials described below; each reported a separate sample, and they are treated as separate studies.

We used a narrative synthesis because the studies varied too widely in phenotype, intervention, design, outcome and follow-up to be pooled. Studies were grouped by the review's three questions and then by clinical theme. The design of each study is stated in the text, and where controlled studies exist, they are described before uncontrolled ones and given more weight; no statistical weighting or meta-analysis was performed.

Throughout, we use "functional symptoms" to mean the core functional outcomes listed above, and we distinguish these from improvement in depression or anxiety and from short-term relief of an acute episode. In keeping with the narrative design, no formal risk-of-bias assessment or certainty grading was performed. Most evidence concerns functional seizures, so the phenotype of each study is stated below, and findings are not assumed to apply to other phenotypes. Because diagnostic standards have changed, the year and original diagnostic label of each controlled study are given below, and older or historical evidence is identified as such in the text. In the Clinical implications section below, we indicate which recommendations go beyond the available studies and reflect expert opinion.

Artificial intelligence was used to support the work. The authors used Claude (Anthropic, San Francisco, CA, USA), a large language model, to help screen the titles and abstracts of exported database records, to extract study details into a spreadsheet and to draft and edit the text. Only published bibliographic records and articles were processed, and no patient data were used. Screening decisions and extracted data were recorded in the spreadsheet. The first author checked the screening decisions, and every study finding, figure and reference cited in this review was checked by the first author against the original abstract, full text or publisher record; claims that could not be verified were removed. Two further authors then read the selected studies and checked the extracted findings against the original papers. They did not independently screen the database records. The conclusions were written by the authors.

The selected studies fell into four themes, which shape the sections that follow. The themes were derived from the review's three questions, with placebo, suggestion and expectation treated as a separate theme because a distinct group of retrieved studies addressed it. Each selected study was assigned to one main theme in the screening spreadsheet, and these assignments were checked by the first author. The largest group described harm from medication and its withdrawal, mostly in functional seizures. A second group tested drugs as treatments, either for the functional symptoms or for comorbid psychiatric conditions. A third compared drugs with psychological treatment or combined the two, and a fourth examined placebo, suggestion and expectation. Because much of this literature predates current terminology, we use "functional seizures" and other current terms in our own text and give the original diagnostic label when describing a study that used an older one.

Antiseizure drugs and delayed diagnosis

The most common route to harm is a mistaken diagnosis of epilepsy. A 2003 review estimated that three-quarters of people with functional seizures and no epilepsy are first treated with antiseizure drugs [10]. In 313 consecutive patients, diagnosis came a mean of 7.2 years after onset, and antiseizure treatment was linked to a longer delay [11]. In a later cohort of 297 patients, each failed antiseizure drug trial added at least a third of a year to the delay [12]. In one US functional seizure clinic, 67% of patients had been prescribed an antiseizure drug, with a median of two drug trials, although 65% had no comorbid epilepsy [13].

The problem is not confined to adults. Of 223 children admitted to a tertiary epilepsy centre with difficult paroxysmal events, 86% were taking antiseizure drugs on admission, yet 87 (39%) were found not to have epilepsy. Most had other non-epileptic events, 12 had functional seizures, and a quarter had their medication tapered before or during admission [14]. People with learning disability are at particular risk. In a consecutive series, patients with functional seizures and learning disability were more likely than those without to be taking antiseizure drugs at diagnosis, to have coexisting epilepsy and to have had previous prolonged functional attacks [15].

The drugs can also appear to work. In one cohort, 22 patients with functional seizures reported complete or partial remission while taking antiseizure drugs, which the authors attributed to a placebo response that may delay the correct diagnosis [16]. An apparent response therefore does not confirm epilepsy, and it can keep a patient on an unnecessary drug for years. These studies are observational, and patients with more frequent or severe attacks are more likely both to be given antiseizure drugs and to wait longer for a diagnosis, so the association with delay does not prove that the drugs cause it. For psychiatrists, the practical lesson is that a history of several failed antiseizure drugs is itself a reason to question the diagnosis.

Polypharmacy and benzodiazepine exposure

People with FND take more medicines overall. In 322 people with motor FND in South London, antidepressants, antiepileptics and analgesics were all prescribed more often than in psychiatric controls [5]. People with functional seizures used more medications than people with epilepsy and were more likely to use narcotics and benzodiazepines [17]. An older cohort found that people with functional seizures (reported as non-epileptic seizures) took fewer antiseizure drugs than people with epilepsy but more other drugs (a mean of 2.3 vs 1.4), and reported more drug allergies [18].

Benzodiazepine exposure is common and persistent. Of 20,848 US adults newly diagnosed with functional seizures, 33.1% received a benzodiazepine in the year before diagnosis. Of those taking one in the month before diagnosis, 62.9% were still taking it in the month after [19]. Diagnosis therefore does not by itself lead to review of the prescription. Each additional drug brings its own adverse effects, interactions and risk of dependence.

Emergency treatment of prolonged functional seizures

Prolonged functional seizures are often treated as status epilepticus, with escalating doses of intravenous drugs. In two large status epilepticus trials, 79 of 980 enrolled patients aged eight or older had prolonged functional seizures. Adverse events occurred in 26% of these patients in one trial, and 33% in the other, and about one in five were admitted to intensive care [20]. In a series of 80 emergency admissions with video-EEG-confirmed functional attacks, 12 (15%) were intubated. Intubated patients had received more

benzodiazepines (a median of 30 mg vs 10 mg diazepam equivalents) and had more complications and longer stays [21]. Patients with functional status also received higher benzodiazepine doses than those with refractory convulsive status epilepticus, up to seizure termination or respiratory failure [22].

Prolonged attacks are common in specialist settings. In one long-term monitoring unit, nine of 49 patients with functional seizures (18.2%) had prolonged attacks mimicking status epilepticus, compared with eight of 154 patients with epilepsy (5.2%) who had true status epilepticus. Emergency admission and having taken fewer than three antiseizure drugs independently predicted prolonged functional attacks [23]. Children are exposed too. Of 10 children with functional seizures seen in a paediatric emergency department, all but one had invasive procedures or laboratory tests, and six were given anticonvulsants [24].

Older case series show the same pattern. In 20 patients with intractable functional seizures, attacks persisted despite intravenous diazepam and therapeutic phenytoin and phenobarbital levels, and 11 patients had respiratory arrest or elective intubation [25]. Failure to recognise postoperative functional status can lead anaesthetists to give inappropriate and potentially harmful treatment [26]. These associations come from observational studies. Patients with longer or more dramatic attacks are more likely both to receive high doses and to be intubated, so higher doses may partly reflect illness severity rather than cause harm on their own.

Practical measures may help. One is to avoid intubation as a reflex response to a prolonged attack [27]. This does not mean withholding emergency treatment. Every prolonged attack still requires airway assessment and support, glucose testing, a search for reversible causes, the local status epilepticus protocol when epileptic status cannot be excluded, early reassessment, urgent EEG where available and neurological advice when the diagnosis is uncertain.

Another measure is an electronic record alert. In 178 adults with an alert for functional seizures, the proportion with at least one intensive care admission fell from 21 (12%) in the year before the alert to two (1%) in the year after, intubations fell from six (3%) to one, and rescue benzodiazepine use fell from 54 (31%) to 10 (6%) [28]. This was a before-and-after comparison without a control group, and the authors noted that the diagnosis itself and the COVID-19 pandemic may have contributed to the fall. Alerts also carry a risk of diagnostic overshadowing: 38% of the patients in that study had coexisting epilepsy, and an alert should prompt careful assessment rather than replace it.

Pregnancy adds a further risk. Five women with functional seizures continued taking antiseizure drugs through pregnancy, obtained from several sources, despite knowing their diagnosis and the risks to the fetus [29].

Drugs temporally associated with functional symptoms

Some case reports describe functional symptoms that began or worsened soon after a drug was started. Two patients with epilepsy developed functional seizures after starting levetiracetam, which stopped or decreased markedly once the drug was withdrawn [30]; this temporal pattern does not establish that the drug caused the attacks or show a specific mechanism. A functional movement disorder worsened during interferon-alpha treatment [31]. A functional disorder (reported as conversion disorder) in an adolescent taking sertraline and erythromycin was first managed as serotonin syndrome in intensive care [32]. In a drug trial, one participant developed multiple functional symptoms within hours of the first dose, and unblinding showed she was on the standard treatment arm [33].

Functional symptoms have also been reported around anaesthesia. Five patients developed seizures after a general anaesthetic and were later shown on video-EEG or ictal EEG to have functional seizures, two of them with concurrent epilepsy [34]. In another case, a woman developed seizure-like activity, rigidity and altered breathing immediately after intravenous lidocaine given before induction. Surgery was postponed, conversion disorder was diagnosed, and she improved with an antidepressant and supportive psychotherapy before uneventful surgery 40 days later [35]. Invasive procedures carry their own risks: more than one-fifth of 54 children with functional movement disorders had unnecessary surgery [36].

Taken together, these studies suggest that harm comes less from any single drug than from a pattern, although almost all of the evidence is observational or anecdotal. A mistaken diagnosis is associated with treatment, treatment with delay to the correct diagnosis, and emergency escalation with acute harm. Symptoms that begin soon after a new drug or anaesthesia are best described as temporally associated with it.

Withdrawing antiseizure drugs

The AAN guideline recommends against antiseizure drugs and benzodiazepines for functional seizures unless there is another indication, such as coexisting epilepsy [3]. The evidence on withdrawal is limited but stronger than for most interventions in FND. In a prospective Glasgow cohort, 78 patients met standard criteria for functional seizures without epilepsy and had their antiseizure drugs withdrawn, 64 of them as outpatients. There were no serious adverse events over 6 to 12 months, and attack frequency fell across the

group, although eight patients had a transient rise [37]. The same group then ran an exploratory randomised trial in 25 patients, a separate sample. Attack frequency fell significantly from baseline to nine months with immediate withdrawal but not with withdrawal delayed by nine months (p=0.028). The immediate withdrawal group also reduced rescue medication use more (p=0.002) and gained more internal locus of control (p=0.005) [38]. In these small studies, withdrawal after careful diagnosis appeared safe and may itself have helped. These findings come from selected patients in specialist services in whom epilepsy had been carefully excluded, and they should not be generalised to all patients with functional seizures. Antiseizure drugs remain appropriate where epilepsy coexists or cannot be confidently excluded, and each withdrawal decision should be made for the individual patient.

Follow-up studies point the same way. Between 50% and 65% of patients had stopped antiseizure drugs at follow-up [39,40], and 83% of clinicians in a US survey reported tapering them after diagnosis [41]. In one management series of 50 patients, 46% of those taking anticonvulsants stopped or reduced them. In that series, 80% of acute cases became attack-free, compared with 28% of chronic cases [42]. In a series of patients with intractable functional seizures, those diagnosed with conversion disorder improved gradually once anticonvulsants were stopped [25]. Withdrawal is not always sustained. In a Dutch follow-up of patients with video-EEG-confirmed functional seizures, eight of 28 respondents were taking antiseizure drugs again 23 to 67 months after diagnosis [43].

The main risk of withdrawal is missing coexisting epilepsy. Three patients in the Glasgow cohort later showed complex partial seizures [37]. In 14 people with both kinds of attack, the first event recorded on video-EEG was functional in 12 [44]. Among people with drug-resistant seizures, functional attacks were three times more likely to coexist with epilepsy than to occur alone [45]. Withdrawal should therefore follow a confident diagnosis, based on review of the attack semiology and, ideally, video-EEG, with specialist neurological input [46]. The taper should be planned for the individual, with written safety-netting that explains what to do if attacks change, a defined follow-up plan and a route to urgent reassessment if new or different events occur. Where epilepsy and functional seizures coexist, reducing antiseizure drugs to the lowest effective dose has been reported to help [47].

Withdrawing benzodiazepines and other drugs

There is little direct evidence on withdrawing benzodiazepines, opioids or other drugs in FND. Given how often benzodiazepines continue after diagnosis [19], a gradual taper under standard benzodiazepine guidance is a reasonable default. Analgesic and opioid use deserves the same review, since analgesics are prescribed more often in motor FND than in psychiatric controls [5] and people with functional seizures are more likely than people with epilepsy to use narcotics [17]. Rescue benzodiazepines for prolonged attacks are a particular target, because they are associated with much of the emergency harm described above [21,22] and their use fell after an electronic record alert flagged the diagnosis [28].

Deprescribing should not extend to drugs that treat a genuine comorbid condition, because stopping an effective antidepressant risks relapse of depression or anxiety. Each drug should be judged against its original indication rather than withdrawn wholesale. Stopping a drug that a patient has taken for years can also feel like a loss of care, so withdrawal is best presented as part of explaining the diagnosis, with a clear plan, a named clinician and continued contact. The Dutch follow-up, in which some patients returned to antiseizure drugs [43], suggests that withdrawal without ongoing treatment of the functional disorder may not last.

Antidepressants for comorbid depression and anxiety

Having considered how medication can cause harm and how unnecessary drugs can be withdrawn, we turn to where it may help, beginning with comorbid psychiatric conditions. Psychiatric comorbidity is common in FND. In 23 outpatients with functional movement disorder referred for psychiatric assessment, 18 (78%) had a psychiatric disorder in addition to the functional diagnosis [48], and in a children's rehabilitation programme the figure was 84.8% [49]. Both figures come from selected specialist samples and may not apply to people with FND in general. In 50 children with functional seizures, separation anxiety disorder, school phobia and mood disorders were the common comorbid diagnoses [50]. In adults with functional seizures, dissociative disorders and PTSD were more common in those without epilepsy than in those with both conditions, as was psychotropic treatment [51]. Liaison psychiatrists recommended antidepressants for 40% of inpatients with somatoform disorders, anxiolytics for 18% and antipsychotics for 10% [52]. The question is less whether to treat comorbidity than whether doing so also helps the functional symptoms, so the studies below are described in terms of which outcomes improved. The studies that have tested a drug or drug-assisted procedure are summarised in Table *1*. The clearest signal comes from Voon and Lang. Fifteen people with chronic functional movement disorder were treated with citalopram or paroxetine, with a switch to venlafaxine if there was no response at four weeks. Of the 10 whose primary diagnosis was conversion disorder, eight had marked improvement in motor and global ratings and seven remitted, and all had current or past depression or anxiety; depression scores also improved across the treated group. None of the five with somatisation disorder, hypochondriasis or probable factitious disorder improved [48]. The study was open-label and small, without a control group, so improvement may also reflect expectation, attention from the clinical team or the natural course of the disorder. It is consistent with antidepressants helping when a

mood or anxiety disorder is part of the presentation, but it cannot separate a direct effect on the functional symptoms from an effect mediated through mood. In functional seizures, the only placebo-controlled trial randomised 38 patients to sertraline, increased to a maximum of 200 mg daily, or placebo for 12 weeks. Seizures fell 45% from baseline on sertraline (p=0.03) and rose 8% on placebo, but the difference between groups was not significant once baseline differences were adjusted for [53]. In an open-label study of venlafaxine in 19 patients with functional seizures and anxiety or depressive disorders, 15 of the 17 who completed follow-up had their seizures more than halved, but there was no control group [54]. A review of randomised trials in somatoform disorders found antidepressants effective in four of five trials, but few involved conversion disorder and the author judged the evidence preliminary [55]. In PPPD, a Cochrane review found no eligible placebo-controlled trials of selective serotonin or serotonin-noradrenaline reuptake inhibitors [56].

Study	Population (original label)	Design	Participants	Drug or procedure; dose and duration	Comparator	Follow-up	Main finding
LaFrance et al., 2010 [53]	Functional seizures	Pilot RCT, placebo-controlled	38 randomised	Sertraline, up to 200 mg daily for 12 weeks	Placebo	12 weeks	45% fall vs 8% rise from baseline; between-group difference not significant after adjustment
LaFrance et al., 2014 [9]	Functional seizures	Pilot RCT, four arms	38 randomised, 34 analysed	Flexible-dose sertraline, alone or with CBT-informed psychotherapy	Psychotherapy alone; usual care	16 weeks	Seizures fell 59.3% (p=0.008) with combined treatment and 51.4% (p=0.01) with psychotherapy alone; sertraline alone not significant (p=0.08)
Ghanbarizadeh et al., 2018 [57]	Acute conversion disorder, ED	RCT, single-blind	144	Oral quetiapine 50 mg	Intravenous haloperidol 5 mg	30 minutes and 24 hours	Symptom relief at 30 minutes in 91.6% vs 90.4% (p=0.81); extrapyramidal effects at 30 minutes in 0 of 71 vs 7 of 73 (p=0.013), not significant at 24 hours
Jafari et al., 2018 [58]	Acute conversion disorder, ED	RCT, double-blind	140	Intravenous haloperidol 2.5 mg	Intravenous midazolam 2.5 mg	One hour to one week	Symptom resolution in 64 of 70 (91.5%) vs 45 of 70 (64.3%) (p<0.001); early side effects in 12 of 70 vs 3 of 70 (p=0.026)
Dreissen et al., 2019 [59]	Jerky or tremulous functional movement disorders	RCT, double-blind	48 treated	Botulinum toxin, two injection sessions three months apart	Saline placebo	Four months	Improvement in 16 of 25 (64.0%) vs 13 of 23 (56.5%); no significant difference
Mousavi et al., 2008 [60]	Acute conversion disorder	RCT, four arms, unblinded	80	Intravenous diazepam	Muscle relaxation, suggestion, hypnosis	One month	All groups improved; recovery faster with relaxation than diazepam; no difference in relapse
Ataoglu et al., 2003 [61]	Functional seizures (conversion disorder), ED	Controlled comparison, allocation not described as random	30	Diazepam	Paradoxical intention	Six weeks	Response in 9 of 15 (60%) vs 14 of 15 (93%); paradoxical intention better for conversion symptoms (p=0.034)
Jain et al., 2020 [62]	Conversion disorder	Comparative study	Not stated in abstract	SSRI with CBT	SSRI alone	Not stated in abstract	Combined treatment better than SSRI alone
Voon and Lang, 2005 [48]	Chronic functional (psychogenic) movement disorder	Open-label series	15 treated	Citalopram or paroxetine, switched to venlafaxine if no response at four weeks	None	Not stated in abstract	Eight of 10 with primary conversion disorder improved; none of five with other somatoform diagnoses
Pintor et al., 2010 [54]	Functional seizures with anxiety or depression	Open-label study	19	Venlafaxine	None	Not stated in abstract	Seizures reduced by more than 50% in 15 of 17 completers
Blumer et al.,	Severe chronic functional	Case series	18 (15	ECT	None	Not stated	Marked improvement in 11 of 15

2009 [63]	seizures		completed)			in abstract	
Poole et al., 2010 [64]	Conversion and dissociative disorder	Systematic review of uncontrolled studies	55 papers	Drug-assisted interview	None	Varied	Suggestion and catharsis linked to recovery; no controlled studies

TABLE 1: Studies testing a drug or drug-assisted procedure in FND

Studies are ordered by design: randomised trials first, then non-randomised controlled, open-label and case-series evidence, then a review. Diagnostic labels are those used in the original studies. Doses and follow-up are given where reported.

RCT: randomised controlled trial; CBT: cognitive behavioural therapy; ED: emergency department; SSRI: selective serotonin reuptake inhibitor; ECT: electroconvulsive therapy; FND: functional neurological disorder

Antipsychotics and other drug classes

Outside the acute trials discussed below, evidence for other drug classes is limited to case reports. Low-dose amisulpride produced a durable response in a woman with functional paralysis who had not responded to escitalopram and lorazepam [65], and adjunctive brexpiprazole has been reported in treatment-resistant functional seizures [66]. These reports describe single patients selected because they responded, so they cannot show how often such drugs help, and antipsychotics carry metabolic and extrapyramidal risks that would weigh against routine use.

A review of functional movement disorders described comorbid depression or anxiety as a favourable prognostic sign [67]. This finding points the same way as the antidepressant studies. Treating a mood or anxiety disorder is justified in its own right and may help the functional symptoms when that disorder is part of what maintains them. It does not support adding drugs from other classes when no comorbid condition is present.

Medication combined with psychotherapy

Only one randomised trial has directly compared medication, psychotherapy and their combination in FND. In a multicentre pilot trial in functional seizures with 34 patients analysed, seizures fell 59.3% with CBT-informed psychotherapy plus sertraline and 51.4% with psychotherapy alone, and both changes were significant. Sertraline alone (26.5%) and usual care were not [9]. Each arm was compared only with its own baseline, so the trial cannot show that adding sertraline improves on psychotherapy. It does show that psychotherapy reduced seizures with or without the drug, while sertraline alone did not. An earlier open trial of manualised CBT for functional seizures reported similar gains. Of 17 patients who completed 12 weekly sessions, 11 had no seizures by the final session, and depression, anxiety, somatic symptoms and quality of life improved [68]. Psychotherapy can therefore improve the mood and anxiety symptoms that medication is often prescribed to treat.

Other comparisons are weaker. An Indian study of patients diagnosed with conversion disorder found CBT plus a selective serotonin reuptake inhibitor (SSRI) better than the SSRI alone [62]. In a single-blind trial of psychodynamic psychotherapy for functional movement disorders, movement scores, depression and anxiety improved in 10 patients, with psychiatric medication used as an adjunct where indicated [69]. In a Czech cohort of 56 people with functional seizures treated with psychopharmacotherapy and/or psychotherapy, about a third were seizure-free at two years, a third had at least a 50% reduction, and a third did not respond [70]. In the Dutch follow-up, six of 13 patients who received psychotherapy became seizure-free, whereas all seven who were referred but not treated continued to have seizures [43]. None of these studies separates the effect of the drug from that of the therapy.

The large psychotherapy and physiotherapy trials set the context. The CODES trial (n=368) found no significant difference in seizure frequency with CBT, but better quality of life, less distress and better functioning [7]. The Physio4FMD trial (241 patients analysed) found no significant difference in physical function at 12 months with specialist physiotherapy [8]. Cochrane reviews have found little reliable evidence for any treatment. The review of psychological treatments for functional seizures found 12 studies, only four of them randomised, and could not pool their results [71]. The review of psychosocial interventions in conversion and dissociative disorders found 17 randomised trials with 894 participants, including comparisons with diazepam, and rated all the evidence as low or very low certainty [72]. In somatoform disorders more broadly, CBT was effective in 11 of 13 randomised trials [55].

Psychotherapy therefore has the stronger and larger evidence base, although its effects on core symptoms are modest. There is insufficient evidence to show whether medication adds to psychotherapy for functional symptoms. This is not the same as evidence that it adds nothing: the small trials available do not

demonstrate an additive effect, but they were not powered to detect or exclude one, and a modest direct effect of some drugs on functional symptoms remains possible. Its best-supported role is to treat comorbid depression, anxiety or PTSD, which may in turn make engagement in psychotherapy possible.

Acute drug treatment in the emergency department

Four trials have tested drugs for acute functional symptoms (reported as conversion disorder), all in emergency or acute settings and three of them in Iran (Table *1*). Benzodiazepines did not outperform psychological approaches. Intravenous diazepam brought slower recovery than muscle relaxation in an 80-patient, four-arm trial, with no difference in relapse [60]. Diazepam produced a lower response than paradoxical intention (60% vs 93%) in 30 emergency patients with functional seizures [61]. In contrast, 2.5 mg of intravenous haloperidol resolved symptoms more often than the same dose of intravenous midazolam (64 of 70 vs 45 of 70 patients), although haloperidol caused more early side effects [58]. Oral quetiapine 50 mg matched 5 mg of intravenous haloperidol at 30 minutes, with fewer extrapyramidal effects at 30 minutes but not at 24 hours [57].

These trials have important limits. They measured symptom relief over minutes to hours, or at most a week, and do not show any effect on the longer-term course of FND. Rapid resolution of an acute episode is not the same as treatment of the disorder: acute functional symptoms often settle on their own, and sedation, suggestion, the expectation created by an injection and the attention that accompanies treatment may all contribute. Three of the four were conducted in one country, and blinding was absent or unclear in some. Given the harms of escalating benzodiazepines described above, they mainly support avoiding benzodiazepines rather than adopting antipsychotics, and they do not justify giving any sedative drug to a patient whose functional attack is not causing harm.

Drug-assisted interviews and electroconvulsive therapy (ECT)

Drug-assisted interviews (abreaction) have a long history. A systematic review covering the literature from 1920 found 55 papers but no controlled study. Suggestion and emotional catharsis during the interview were linked to recovery, while combining two drugs and comorbid psychiatric disorder were linked to worse outcomes [64]. In a small series, intravenous diazepam was used to interview eight diagnostically difficult patients and was reported to be safe and to help clarify diagnoses that included conversion disorder, depression and dissociative states [73]. The evidence is poor and uncontrolled. The technique may help some acute or treatment-resistant presentations, but these reports cannot separate any drug effect from suggestion, which appears to contribute substantially to recovery.

In 18 patients with severe chronic functional seizures, 15 tolerated ECT, and 11 of them improved markedly [63]. These patients also had depressive symptoms and pain, so the effect may reflect treatment of comorbid depression. There is no good evidence that ECT treats FND itself, but it should not be withheld when severe depression coexists, and its use should follow the usual indications for depression rather than the functional symptoms.

Botulinum toxin, psychedelics and ketamine

The only placebo-controlled trial of botulinum toxin in functional movement disorders randomised 48 patients with jerky or tremulous movements. Improvement at four months was similar with botulinum toxin and saline (64.0% vs 56.5%), and the large improvement in both groups points to a strong placebo response [59]. The trial matters beyond botulinum toxin, because it shows how misleading uncontrolled reports of improvement can be when placebo responses are this large.

A systematic review of psychedelics found nine studies from 1954 to 1967, with 26 patients in total; 18 were reported to have recovered to some degree, but the criteria were subjective, and the studies were of low quality [74]. A 2026 systematic review of ketamine and esketamine found 13 studies, mostly case reports and small series, with some improvement in motor symptoms and functional seizures but insufficient evidence to establish efficacy [75]. Interest in drugs that might make psychotherapy more effective is growing, but this remains experimental, and these drugs should be used only within research protocols.

Placebo, suggestion and expectation

Suggestion runs through all these findings. Saline infusion with verbal suggestion induced attacks in 41 of 66 patients; at 4.5 years, seven of 16 with a positive test were attack-free, compared with five of 15 with a negative test [76]. In an outpatient EEG laboratory, suggestion or saline infusion produced non-epileptic attacks in 52 of 72 referred patients, and the authors estimated that about half of suspected cases could avoid long-term video-EEG monitoring [77]. Open placebo therapy, given with the patient's knowledge and consent, has been used in a functional movement disorder [78].

The use of placebo is contested. Supporters of provocative testing argue that it can turn an inconclusive assessment into a diagnostic one and that leaving the wrong diagnosis in place is the greater harm [79]. Opponents argue that deceptive induction risks the patient's trust and later engagement with treatment,

and that methods without deception should be used instead [80]. We favour transparent, non-deceptive methods, such as suggestion-based induction explained to the patient in advance, because they protect consent and trust. Provocation testing of any kind can support but cannot replace video-EEG evaluation, and a negative or positive result does not by itself exclude epilepsy. For prescribers, the wider point is that any drug may act partly through expectation, for good or ill, as the apparent response to antiseizure drugs shows [16]. How a drug is introduced, and what the patient is told it will do, can shape both benefit and adverse effects.

Clinical implications

The evidence supports a simple order of priorities: stop what harms, treat what coexists, and keep psychotherapy and physiotherapy at the centre. Table *2* sets this out as a stepwise approach for psychiatrists and neurologists.

Step	What to do	Main evidence
Step 1: Confirm the diagnosis and review every medicine	Confirm FND with positive clinical signs and, for seizures, video-EEG where possible. Record each drug, its original indication and who started it.	[3,10-13,44,45]
Step 2: Withdraw drugs without an indication	After a confident diagnosis, with review of attack semiology, EEG assessment and specialist neurological input, taper antiseizure drugs on an individual schedule. Give written safety-netting, arrange follow-up and agree a route to urgent reassessment if new or different events occur. Taper benzodiazepines gradually. Review opioids and other analgesics.	[3,19,37-40,46]
Step 3: Plan emergency care	Write an individual plan for prolonged attacks that avoids escalating benzodiazepines and reflex intubation, while keeping airway support, glucose testing, a search for reversible causes, the status epilepticus protocol where epileptic status cannot be excluded, urgent EEG and neurological advice. Flag the diagnosis in the health record without allowing it to replace assessment.	[20-22,27,28]
Step 4: Assess and treat comorbidity	Screen for depression, anxiety, PTSD, pain and sleep problems, and treat them on their usual indications.	[48,49,53-55]
Step 5: Keep psychotherapy and physiotherapy central	Offer medication alongside, never instead of, psychotherapy or physiotherapy, and explain what each drug is for.	[7-9,62]
Step 6: Review regularly	Check benefits, adverse effects and expected effects, and stop drugs that are not helping.	[16,17,59,76]

TABLE 2: A stepwise approach to medication in FND

Steps 1 and 2 rest mainly on observational studies and one small randomised trial; Steps 3 to 6 draw substantially on expert opinion and general clinical practice.

FND: functional neurological disorder; EEG: electroencephalography; PTSD: post-traumatic stress disorder

The first two steps rest on the strongest evidence in this review. A confident diagnosis, with video-EEG where seizures are involved, should come before any change, because missing coexisting epilepsy is the main risk of withdrawal [37,44,45]. In selected patients, withdrawal of antiseizure drugs then appears safe and may itself reduce attacks, although this rests on small studies and should not be generalised to every patient with functional seizures [37,38]. An emergency care plan matters as much as the outpatient prescription, since most acute harm comes from escalating intravenous treatment [20-22,28].

The later steps rely on weaker evidence and largely reflect expert opinion and general psychiatric practice. Antidepressants should be prescribed for depression or anxiety on their usual indications, with an honest message that they are being used to treat the mood or anxiety disorder, and that there is not yet enough evidence to say whether they also improve the functional symptoms directly [9,48,53]. Framing may matter: a patient told that a drug will stop the functional symptoms may lose confidence in the diagnosis when it does not. Each prescription should have a stated purpose and a review date, so that drugs which do not help are stopped rather than added to.

Future research directions

The most pressing question is whether medication adds anything to psychotherapy, and for whom. The sertraline pilot trials [9,53] and the antidepressant series by Voon and Lang [48] suggest a benefit confined to patients with depression or anxiety. A definitive trial should randomise patients to an antidepressant or placebo alongside standardised psychotherapy, stratify by comorbid mood and anxiety disorders, and include functional motor disorder as well as functional seizures, since almost all controlled drug data come from functional seizures. Such trials should also look for predictors of response, such as symptom duration, comorbidity and illness beliefs.

Deprescribing deserves trials of its own. The only randomised evidence concerns antiseizure drugs [38], yet benzodiazepines often continue after diagnosis [19], and analgesics are prescribed more often in FND than in psychiatric controls [5]. Trials of supported benzodiazepine and opioid withdrawal, measuring attack frequency, function and emergency attendance, would turn the harm data in this review into practice. Emergency care needs the same attention. An electronic record alert reduced intubation and rescue benzodiazepine use in one centre [28], and multicentre studies should test whether such pathways work more widely.

Mechanistic research could explain why drugs help some patients and harm others. Current models describe functional symptoms as the product of attention and strongly held expectations that shape perception and movement [81]. This account predicts large placebo and nocebo effects, as seen in the botulinum toxin trial [59], in apparent responses to antiseizure drugs [16] and in symptoms that begin soon after a new drug is started [30,33]. Studies that measure patients' expectations before prescribing, and test honest ways of explaining what a drug is for, could make prescribing safer and more effective. The same questions apply to ketamine and psychedelics, which have been proposed as aids to psychotherapy but are supported so far only by uncontrolled reports [74,75].

Finally, comparison between studies is hampered by inconsistent outcomes. A systematic review found that no single measure suited the full range of FND symptoms and recommended consistent use of validated measures covering symptoms, functioning and quality of life [82]. Drug trials in FND should adopt these recommendations so that their results can be compared and pooled.

Limitations

This review has the limits of a narrative design. We searched only PubMed and Embase, without PsycINFO, Cochrane CENTRAL, Scopus, trial registries, grey literature or systematic citation tracking, so relevant studies, particularly psychological and rehabilitation studies and unpublished trials, may have been missed. Titles and abstracts were screened by a single author, and data were extracted once and then checked by two further authors rather than extracted independently in duplicate. The review did not use a registered protocol, a flow diagram or a record of reasons for full-text exclusion. No formal risk-of-bias assessment or certainty grading was performed, so the strength of each study was described rather than graded, and our conclusions should be read in the light of a small, heterogeneous and low-certainty evidence base. Several study details, including some doses and follow-up periods, were available only from abstracts. Much of the evidence comes from older studies that used terms such as hysteria, conversion reaction and pseudoseizures, with diagnostic standards that would not meet current criteria, and many did not report whether video-EEG or positive clinical signs were used to confirm the diagnosis. Some participants in these studies may therefore have been misclassified.

The evidence itself is thin. Most drug studies are case reports, case series or open-label cohorts. The few controlled trials are small and short, and several are unclear about randomisation and blinding. Functional seizures dominate the literature; there are few drug studies in functional motor disorder and almost none in functional sensory or cognitive presentations. Most treatment studies measured attack frequency or short-term symptom relief rather than function, quality of life or long-term outcome. Harm is better documented than benefit, but mostly through retrospective cohorts that show association rather than cause.

Conclusions

There is no good evidence that medication directly treats FND, and insufficient evidence to show whether adding medication to psychotherapy improves functional symptoms. In some cases, medication can actually cause harm, particularly when people with dissociative or functional seizures are incorrectly treated with antiseizure medications or benzodiazepines. This can delay the correct diagnosis and, in acute situations, may lead to unnecessary interventions such as respiratory support or intubation. Once FND has been confidently diagnosed, and coexisting epilepsy has been excluded, withdrawing antiseizure medication appears safe in selected patients and may even reduce seizure frequency. That said, medication still has an important supporting role in managing co-occurring conditions such as depression, anxiety, and PTSD alongside psychological and physiotherapy-based interventions. For psychiatrists, this highlights the importance of regularly reviewing medication, discontinuing treatments that are no longer indicated, ensuring clear emergency care plans are in place, and treating comorbid mental health difficulties on their own merits rather than attributing everything to FND.

Appendices

Database	Platform	Date searched	FND terms	Medication terms	Limits	Records
PubMed	NLM	22 October 2026	Conversion Disorder (MeSH); functional neurological; conversion disorder; psychogenic non-epileptic and nonepileptic; non-epileptic seizure and attack; PNES; functional, dissociative seizure; functional, psychogenic movement and motor disorder; functional tremor, dystonia, weakness; persistent postural-perceptual dizziness; functional cognitive disorder	Psychotropic Drugs, Antidepressive Agents, Anticonvulsants, Benzodiazepines, Analgesics, Opioid, Antipsychotic Agents, Deprescriptions, Polypharmacy (MeSH); drug therapy (subheading); medication; pharmacotherapy; antidepressant; SSRI; sertraline; antiepileptic; antiseizure; benzodiazepine; opioid; antipsychotic; haloperidol; quetiapine; propranolol; prazosin; clonidine; ketamine; esketamine; psychedelic; deprescribing; taper; intubation	English	1,138
Embase	Ovid	22 October 2026 (database 1974 to 20 October 2026)	Conversion disorder and psychogenic nonepileptic seizure (Emtree); the same free-text terms as PubMed, with functional terms placed next to movement, motor, tremor, dystonia, weakness, paresis or gait	Psychotropic agent, antidepressant agent, anticonvulsive agent, benzodiazepine derivative, opiate, neuroleptic agent, deprescription, polypharmacy, drug withdrawal (Emtree); the same free-text terms as PubMed	Human, English; conference abstracts excluded	2,574

TABLE 3: Literature search strategy

Terms were combined with OR within each column and the two columns with AND.

FND: functional neurological disorder; MeSH: Medical Subject Headings; Emtree: Embase subject headings; NLM: National Library of Medicine; PNES: psychogenic non-epileptic seizures; SSRI: selective serotonin reuptake inhibitor

Additional Information

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All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

AI use statement: The authors used Claude Opus 5.5 (Anthropic, San Francisco, CA, USA; accessed October 2026) as follows: language polishing, structure or formatting, methodological or statistical suggestions, literature comparison, drafting summaries, data cleaning or exploratory analysis, extensive copy editing or writing support, screening of database search results (titles and abstracts) and extraction of study data into a spreadsheet in Abstract, Introduction and Background, and Review (including the search approach and Tables 1 to 3), References, and the screening log and data extraction spreadsheet. The Conclusions were written by the authors without AI assistance. The authors directed the scope, search strategy, and structure of the review. AI-assisted screening decisions were reviewed by the first author, and the selected studies and

their key findings were reviewed by two further authors. Every reference was checked against its source record (DOI, publisher page or PubMed), and all figures, sample sizes and statistics were checked against the original abstracts or full texts; claims that could not be verified were removed or reworded. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work.

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