Functional symptoms in neurology: mimics and chameleons

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ABSTRACT

The mimics and chameleons of functional symptoms in neurology could be a whole textbook of neurology. Nevertheless, there are some recurring themes when things go wrong, notably diagnostic bias introduced by the presence or absence of psychiatric comorbidity or life events, neurological diseases that look 'weird' and lack of appreciation of the more unusual features of functional symptoms themselves.

INTRODUCTION

When the request came to write this review article on mimics and chameleons of functional neurological symptoms, we had to do a double take. You want us to help neurologists to avoid accidentally diagnosing functional symptoms as a disease? Are you sure you don't just mean the other way round?

Functional/psychogenic/non-organic symptoms are customarily diagnoses that figure on the differential diagnosis of other conditions. There are plenty of cautionary tales in the literature about patients with a neurological disease misdiagnosed as 'hysterical' and 'non-organic'. However, we are not aware of any previous review article that has tackled the issue of differential diagnosis from the perspective of functional symptoms.

It is gratifying, therefore, that the field has come sufficiently far to warrant a discussion of diagnostic pitfalls in the same terms as that for epilepsy and Alzheimer's disease, the ultimate aim being that neurologists might all one day say to themselves, 'It would be really embarrassing (or maybe I will get sued) if I miss the diagnosis of functional symptoms in this patient'.

Functional symptoms are, as any general neurologist knows, very common, and are the second commonest reason for a neurological outpatient consultation (in Scotland, anyway).[1] Non-epileptic attacks account for around one in seven patients in a 'first fit' clinic, and functional limb weakness is as common as multiple sclerosis.[2] They also account for a group of patients who, by self-report at least, are as physically impaired and more distressed than equivalent patients seen in neurology outpatients with disease.[3]

Most neurological symptoms can have a functional explanation. In this article, we will discuss general pitfalls in assessing and approaching patients with functional symptoms, and then discuss separately individual pitfalls of dissociative (non-epileptic) attacks, functional motor symptoms and speech/visual/cognitive symptoms.

The guiding principle of diagnosis of most functional symptoms is that there should be inconsistency during the physical examination (so-called internal inconsistency) or incongruity with recognised neurological disease. Sticking to this principle will avoid many of the pitfalls listed below. This article does not recapitulate all the positive clinical signs of inconsistency and incongruity (such as Hoover's sign for functional weakness, motor distraction tasks for functional tremor and features such as eyes closed during a generalised shaking attack), but they are available elsewhere.[4]

As in much of neurology, there are patients where there is diagnostic uncertainty; as a clinician you should always be prepared to say 'not sure'.

EIGHT SHADES OF DIAGNOSTIC CHANGE

Even when the diagnosis does appear to change over time, it is rarely as simple as, '*I thought it was functional, but actually it's multiple sclerosis*'. There are different kinds of diagnostic change with different degrees of error. As well as the most well known type of misdiagnosis—when you look back and think, '*got that wrong*'—there are other types of change that could be construed as error when in fact they are not (table 1).

Table 1 A change in diagnosis doesn't necessarily mean you got it wrong first time around (adapted from[1] with permission)

Type of diagnostic revision	Example	Degree of clinician error
1 Diagnostic error	Patient presented with symptoms that were plausibly all due to multiple sclerosis but was diagnosed with functional symptoms. The diagnosis of multiple sclerosis had not been considered and was unexpected at follow-up	Major
2 Differential diagnostic change	Patient presented with multiple symptoms. Doctor suggested chronic fatigue syndrome as most likely but considered multiple sclerosis as a possible diagnosis. Appropriate investigations and follow-up confirmed multiple sclerosis	None to minor
3 Diagnostic refinement	Doctor diagnosed epilepsy but at follow-up the diagnosis was refined to juvenile myoclonic epilepsy	Minor
4 Comorbid diagnostic change	Doctor correctly identified both epilepsy and non-epileptic seizures in the same patient. At follow-up, one of the disorders had remitted	None
5 Prodromal diagnostic change	Patient presented with an anxiety state. At 1 year follow-up, she had developed Alzheimer's disease. With hindsight, anxiety was a prodromal symptom of dementia but the diagnosis could not have been made at the initial assessment as the dementia symptoms (or findings on examination or investigation) had not developed sufficiently.	None
6 De novo development of organic disease	Patient was correctly diagnosed with chronic fatigue syndrome. During the period of follow-up, the patient developed subarachnoid haemorrhage as a completely new and unrelated condition	None
7 Disagreement between doctors—without new information at follow-up	Patient was diagnosed at baseline with chronic fatigue syndrome and at follow-up with chronic Lyme disease by a different doctor, even though there is no new information. However, if the two doctors had both met the patient at follow-up, they would still have arrived at the different diagnoses. This would be reflected in similar divided opinion among their peers	None
8 Disagreement between doctors—with new information at follow-up	Patient was diagnosed at baseline with chronic fatigue syndrome, and at follow-up with fatigue due to a Chiari malformation by a different doctor because of new information at follow-up, (in this case an MRI scan ordered at the time of the first appointment). However, the first doctor seeing the patient again at follow-up continued to diagnose chronic fatigue syndrome, believing the Chiari malformation to be an incidental finding. This would be reflected in divided opinion among their peers	None

For instance, someone presenting with functional hemiparesis who later develops motor neurone disease, may genuinely have had a functional hemiparesis, it's just that you didn't detect (and weren't able to detect) the comorbid neurological disease predisposing to it at the time. Alternatively, a patient presenting with a functional movement disorder may, 1 year later, have a stroke, but it still doesn't account for their functional movement disorder. Diagnostic disagreements, patients where the diagnosis of functional symptoms is initially in the differential but then drops out, also form part of the list of ways in which diagnoses may change over time, without there necessarily having been a 'howler'.

GENERAL CLINICAL FEATURES OF MIMICS AND CHAMELEONS

Table 2 lists some factors that we often come across in patients who have been erroneously labelled as having a disease when they actually have functional symptoms, and vice versa.

'This patient is anxious/recently stressed/has a personality disorder', so, must have functional symptoms

Probably the commonest source of diagnostic error is when the clinician pays too much attention to the patient's psychosocial history and not enough to the presenting symptom. A generation and more of doctors have been taught via psychiatric diagnostic criteria that functional neurological symptoms are a form of 'conversion disorder' and, as such, represent the conversion of recent stress into a physical symptom.

In fact, the evidence on life events is not conclusive. Some studies have found an excess of recent and more distant life events[5] compared with disease controls, but some have not.[6 7] It is clear that there are many patients with no recent life events, and many others in whom life events or stress cannot be easily related to their symptom.

Likewise, the data on comorbid psychiatric disorder (such as anxiety, panic and depression) and personality disorder (such as avoidant or borderline personality) suggest that these things are more common than in disease controls. However, many patients with functional symptoms are psychiatrically 'normal', and around a third of patients with defined neurological disease have comorbid psychiatric symptoms. The upshot of this is that it is dangerous to base your diagnosis on the psychosocial history, however tempting the narrative may appear. Just as you wouldn't make a diagnosis of stroke because someone smokes and has high blood pressure, these features should be regarded as supporting risk factors but not diagnostic in their

Table 2 Functional symptoms; general factors relevant to spotting mimics and chameleons

Mimics: features of neurological disease that can lead erroneously to a diagnosis of functional symptoms	Diagnostic clues/how not to mess up
The presence of psychiatric disorder, especially personality disorder	Detecting psychiatric comorbidity may be useful in treating the patient but should be ignored in making the diagnosis. Focus on the nature of the attack/the physical examination. Are the physical features typical of functional symptoms?
Presence of schizophrenia or other psychotic illness	Such patients seldom have functional symptoms.
The patient's presenting complaint is of new onset mood or behavioural disturbance.	Patients with functional symptoms rarely complain of significant psychiatric or behavioural symptoms, for example, panic, as their primary, subjective complaint, even if it is clearly present
The presence of an obvious life event or stressor	Ignore the presence of recent stress in making the diagnosis, even if this may be relevant for treatment
Failure to consider that the patient may have functional symptoms AND a neurological disease	Remember that neurological disease is one of the most powerful risk factors for developing functional symptoms. (eg, epilepsy/non-epileptic attacks, multiple sclerosis/functional weakness, idiopathic intracranial hypertension/ functional visual symptoms)
Failure to consider that the patient may have functional symptoms AND a progressive neurological disease, which may be too early for you to diagnose (yet)	As above, but in some cases, especially where neuroimaging doesn't help, the disease may only become apparent on follow up (eg, motor neurone disease, Wilson's disease, Alzheimer's disease, myopathy)
'La belle indifférence'—apparent indifference to disability	This 'sexy' French concept is wedded to conversion disorder and is of no diagnostic value, probably occurring more frequently in neurological disease, especially with frontal lobe involvement[11]
Normal neuroimaging	Many neurological diseases, for instance, epilepsy, motor neurone disease, myopathy, spinocerebellar ataxia have normal brain and spine imaging. Don't rely on it alone to exclude disease
Chameleons: features of patients with functional symptoms that can wrongly put you off the diagnosis	
The patient is 'normal'/'nice'/'stoical'/'like me'	Nice people get functional symptoms too
The patient has no 'form' that is, previous functional symptoms	Patients can present with dramatic neurological functional symptoms with no prior history
The patient has not been stressed	Between 1 in 3 and 1 in 4 patients have no evidence of recent stress
The patient is not tired/ only has one symptom	Lack of fatigue or other symptoms should make you think twice about a diagnosis of functional symptoms but monosymptomatic presentations do occur
The symptoms came on after injury, minor pathological disease	Commonplace in functional symptoms[12]
The patient suggests a psychological causation	Around 1 in 4 patients with functional symptoms do think that psychological factors are relevant
The patient has an established diagnosis of 'known epilepsy', 'known MS'	Always question other people's diagnoses (and your own!)
The patient is too old	Older patients with functional symptoms often have health anxiety and comorbid disease and are probably under-recognised[8]
Incidental abnormalities on MRI (eg, enlarged perivascular space, Chiari malformation), EEG, serology or other tests	Do not assume that all structural abnormalities are relevant

own right. Be particularly careful of the patient who thinks their symptoms *are* stress related, as patients with disease are more likely to present with psychosocial attributions than patients with functional symptoms.[2]

'The patient is too normal/nice/stoical/male/old/'like me'', so, must have a neurological disease

The converse and quite common pitfall is the patient with functional symptoms who has the misfortune to share the same social and demographic features of the doctor attempting to make the diagnosis. In line with the discussion above, middle-aged males, people who are 'normal', 'nice' or 'seem genuine enough' can all develop functional symptoms, even dramatic ones. Studies on older patients with non-epileptic attacks show that they have an equal sex ratio, and often suffer from potentially life-threatening disease (such as ischaemic heart disease or severe asthma), triggering health anxiety that links to the attacks themselves.[8]

'I've made a diagnosis, there is no need for another one'; the problem of comorbidity

The children's card game 'Top Trumps' involves comparing features of cars or superheroes, like 'top speed' or 'agility' to see which one is best. In the

Case 1 La belle indifférence, dysphonia and drooling

A 38-year-old woman presented to general medical services with a whispering dysphonia that was, even with the benefit of hindsight, correctly identified as a functional symptom. She then developed progressive mobility symptoms with unsteadiness, slowness and a change in personality.

Medical staff commented on her 'belle indifférence' to her neurological symptoms and attributed her executive impairment on neuropsychological testing to possible conversion disorder.

One year after initial presentation, she had drooling, dysarthria, ataxia, parkinsonism and had become virtually mute.

See Answers at the end of the article for the conclusion to this case

Case 2 A businesswoman whose 'funny face' was not funny

A 41-year-old successful businesswoman presented with an acute onset of facial asymmetry, initially noticed by her 10-year-old daughter who said it looked 'funny'. The symptom progressed over 1–2 h, with tingling and a pulling feeling on the right side of her face, which prevented her from speaking properly. She also developed a heavy feeling in the right arm and leg over 2 h, which worsened on arrival at hospital.

She had no risk factors for stroke, other neurological disease and no recent life events other than being busy running her business. She had no history of depression, anxiety, fatigue or any other functional symptoms.

The medical clerking recorded lower right facial weakness along with right-sided arm and leg weakness, as well as dense numbness of the right hand.

An MR brain scan, including diffusion weighted imaging and MR angiogram, was normal. The neurology team was called.

See Answers at the end of the article for the conclusion to this case

neurological 'Top Trumps' of the outpatient clinic, functional symptoms are usually a card that neither doctor nor patient wishes to hold, since it would always be beaten on criteria of public understanding and popularity by a disease diagnosis (figure 1).

The presence of any disease, however small, therefore, tends to 'trump' the presence of functional symptoms; but the reality is that the experience of bodily dysfunction caused by neurological disease is one of the most powerful risk factors for developing functional symptoms. Many patients have two diagnoses, for instance, epilepsy and dissociative (non-epileptic) attacks; multiple sclerosis and functional limb weakness; idiopathic intracranial hypertension and functional visual symptoms. It is easy for the presence of disease to obscure the presence of functional symptoms. Conversely, recognising the functional symptom, diagnosis can assist the patient's treatment as it will often have more potential for reversibility than the underlying disease. In our own Scottish Study of 2467 outpatients with neurological disease, around 12% also had a diagnosis of a functional symptom. In these 12% of patients, no single disease category was more common than another. In other words, patients with, for example, multiple sclerosis or Parkinson's disease, do not appear to be more prone to functional symptoms than people with epilepsy or muscle disease.[9]

Comorbidity can present concurrently, for example, a patient who presents acutely with a mild episode of demyelination in whom the clinical features of the weakness are predominantly functional. Or functional symptoms can develop as a later complication of neurological disease; with health anxiety often having a key aetiological role.

More problematically, some patients, especially those with degenerative and slowly progressive conditions, may present with functional symptoms years before the clear onset of their neurological disease. In some cases, a definite functional diagnosis, such as functional paralysis can present as part of the commonly encountered 'psychiatric prodrome' in dementias. In others, the experience of having a very mild ataxia, for instance, in the very early stages of spinocerebellar ataxia, seems enough to trigger the functional symptom.

The answer is, don't play neurological top trumps! Always look for comorbid disease, even if the diagnosis of functional symptoms is clear-cut, and make two diagnoses if necessary. If there is no disease, consider whether there are features that deserve longer-term follow-up that might indicate the patient is in the early stage of a slowly progressive disease. Finally, accept that you will sometimes get it wrong, or fail to anticipate the development of a disease, however careful you are. Studies of functional symptoms from the 1970s onwards coalesce around frequencies of misdiagnosis of about 5% after 5 years.[10] This is the same rate of misdiagnosis for most neurological and psychiatric disorders, and probably at least as common as misdiagnosis of functional symptoms as disease.

DISSOCIATIVE (NON-EPILEPTIC) ATTACKS

A previous article in this series discussed mimics and chameleons of epilepsy, including dissociative (non-epileptic) attacks.[13] Looking at the problem from the other end of the telescope, several features come into view which can lead to confusion both in terms of mistakenly calling attacks non-epileptic when they are

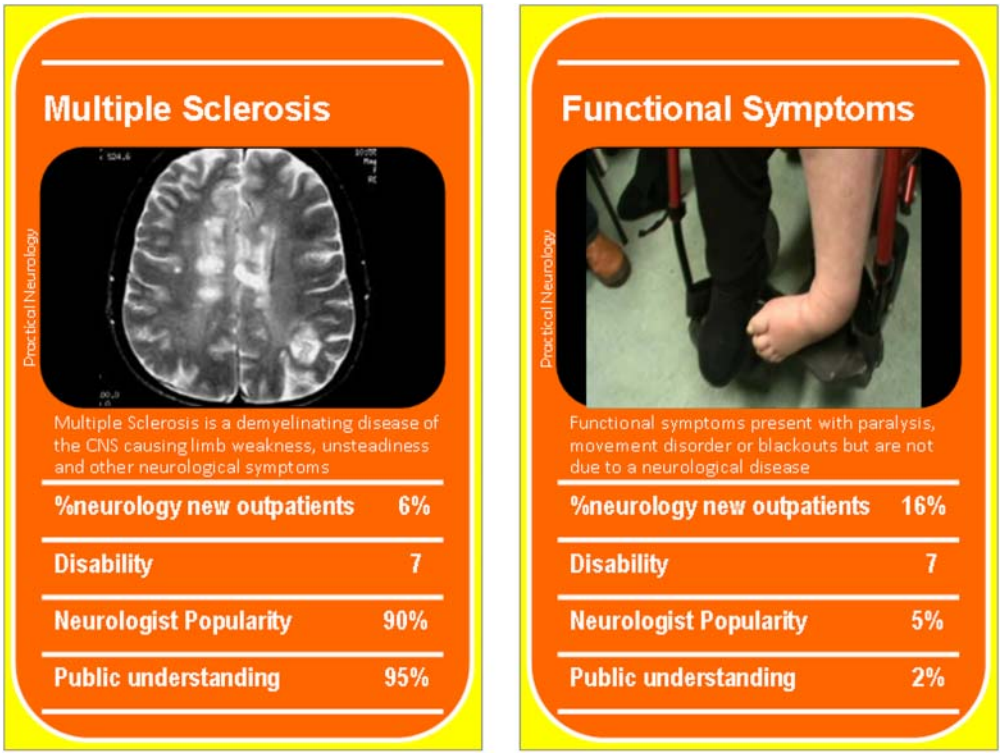

Figure 1 Neurological diseases tend to trump functional symptoms for both neurologists and patients, but try not to play this game when making a diagnosis in clinic. Instead, be willing to make two diagnoses if necessary.

not, and missing the diagnosis of non-epileptic attacks when it is right in front of you (table 3).

The diagnosis of non-epileptic attacks should be made on the basis of objective signs, such as eyes closed, resistance to eye opening, ictal or postictal weeping and prolonged attacks.[14] The problem is that the evidence for many of these signs comes from videotelemetry studies: in the real world, witnesses, including medical ones, can be very unreliable. For example, reports of eye closure from witnesses may be close to useless when compared with video EEG evidence.[15] Therefore, the patient's subjective seizure experience is also important in giving additional clues. Simple questions, such as whether they 'remember the shaking' can be helpful,[14] but there is also evidence that there are conversational features of seizure description typical of non-epileptic attacks, including reluctance to describe seizure symptoms, or giving a poorly detailed description.[16] Instead, patients with dissociative attacks prefer to focus on the situations in which their attacks occur and the consequences of attacks.

The main theme of the non-epileptic attack mimics is that there are types of epilepsy that look a bit weird. For example, frontal lobe seizures can be associated with retained awareness or pelvic movements that can lead to assumptions that the patient may be 'acting out' abuse.[17] It is particularly important to remember that in temporal lobe epilepsy there can be quite a long prodrome lasting minutes in which the patient may have fear and dissociative symptoms similar to a patient having a dissociative (non-epileptic) attack.[18] Although ictal fear is usually distinguishable from a panic attack by the shorter duration, associated temporal lobe features and impaired awareness,[19] this is not such an easy distinction to make with non-epileptic attacks which may have all these features.

The chameleons are mostly those things that people think are quite specific to epilepsy, but in reality are not, such as injury[20] (and especially report of injury), olfactory hallucinations and going blue.

In clinical practice, it is not always possible to be sure what a patient's attack disorder is due to, even with all this information. For this reason, the careful neurologist strikes a balance between making confident diagnoses where possible, but saying 'not sure' where appropriate. In any patient, it is important not to close the book completely on the diagnosis, checking seizure descriptions at each visit, and watching out for the combination of both epilepsy and non-epileptic attacks. Trials of antiepileptic drugs should be avoided because they rarely deliver convincing answers, often increase diagnostic confusion, and may have iatrogenic effects.

FUNCTIONAL MOTOR SYMPTOMS

The diagnosis of functional motor symptoms should always be based on positive evidence on the examination of internal inconsistency (eg, Hoover's sign for

Table 3 Dissociative (non-epileptic) attacks: mimics and chameleons

Mimics: conditions that can look like dissociative (non-epileptic) attacks	Diagnostic clues/how not to mess up
Generalised tonic–clonic seizure	Include: ictal guttural 'cry' (not weeping) typically at onset, stertorous breathing, eyes open
Frontal lobe seizures	Short duration (less than 30 s) Retained awareness during seizures Shouting, truncal or cycling leg movements Onset often/mostly from sleep
Temporal lobe seizures with ictal fear	Progression to generalised convulsion. Structural cause. Many temporal lobe features (eg, olfactory hallucinations, macropsia) can appear in a dissociative non-epileptic attack
Self-induced seizures	Some patients with epilepsy can induce their own seizures, or may manipulate their medication to do so
Autoimmune limbic encephalitis (eg, Anti-NMDA, Anti-VGKC)	Patients may present with psychiatric symptoms, unusual behaviour and focal seizures
Stress induced seizures or syncope	Some epileptic seizures and cardiac syncope (eg, long QT-related) can be triggered by emotional stress
Chameleons: features of genuine dissociative (non-epileptic) attacks that can wrongly put you off the diagnosis	
Olfactory hallucinations	Reports of 'burning rubber'/ 'faeces'/'chemical smell' appear quite commonly in dissociative attacks
Dissociative experiences	Depersonalisation, visual and perceptual changes in dissociative attacks can sound like temporal lobe epilepsy
Eyes open	Although 'eyes closed' is a good clue, some patients with dissociative attacks do open their eyes (with rolling) during attacks
Cyanosis/breath holding	Including low oxygen saturations
Injury	Including bitten tongue (sometimes visibly), broken teeth, (recurrent) shoulder dislocation and falls on stairs all occur in dissociative attacks. Reports of injury may be more common than actual injury
Incontinence	Urinary incontinence is common and faecal incontinence does happen in dissociative attacks
Seizures arising from sleep/when alone	Occurs in dissociative attacks
Response to 'trial of antiepileptics'/relapse of attacks when antiepileptics withdrawn	Patients with dissociative attacks may experience both strong placebo effect when antiepileptics are started and nocebo effect when they are stopped
The patient in intensive care who several non-neurologist physicians and anaesthetists are convinced is in status epilepticus	Prolonged events/seizure 'status' is more common in patients with dissociative attacks than in patients with epilepsy. Up to 50% of patients attending hospital in apparent refractory status have this diagnosis

paralysis or a tremor that stops or entrains during contralateral cued rhythmical movement).[4] However, there can be difficulties in overinterpretation of these 'positive signs', and it would be unreasonable to expect them always to perform, especially in isolation. The presence of pain in a limb, inattention or neglect, or simple failure to understand the examiner's instructions, are all reasons why these signs may be falsely positive.

As with epilepsy, things that look weird, like stiff person syndrome or generalised dystonia, particularly if they are inherently somewhat variable, can fool the unwary into a diagnosis of functional symptoms. The list in table 4 is obviously not comprehensive. Orthostatic tremor (a movement disorder only present on standing), alien limb phenomena in corticobasal degeneration, and the aura of paroxysmal kinesigenic dyskinesia, are just some of the reasons why the diagnosis of functional neurological symptoms should usually be made by a neurologist who is familiar with the breadth of unusual presentation that neurological disease has to offer.

Conversely, in patients who do have functional symptoms, there can be surprising findings. Just as reflexes can be brisk in people who are anxious, we have seen patients with unilaterally increased reflexes as a transient phenomenon. Such reflex asymmetry was well reported in the older literature.[21] Occasionally, patients with unilateral motor symptoms also develop something that looks very similar to ankle clonus, but is variable between assessments. It is not unusual for plantars to be mute on the same side as functional hemisensory loss.

Functional facial symptoms, typically with contraction of orbicularis oculis, orbicularis oris and platysma and sometimes with jaw deviation are clinically quite common (figure 2). They were well described in the older literature, and have recently been described

Table 4 Functional motor symptoms: mimics and chameleons

Mimics: conditions that can look like functional motor symptoms	Diagnostic clues/how not to mess up
Higher cortical gait disturbance	Don't rely on an 'odd gait' to make the diagnosis
Acute parietal stroke/pathology	May have Hoover's sign/MRI brain
Stiff person syndrome	Electrophysiology findings and antiglutamic acid decarboxylase (anti-GAD) antibodies
Dystonia (geste antagoniste), better walking backwards or running)	Familiarity with clinical presentation of organic movement disorders
Myasthenia (variability, give-way weakness)	Avoid putting diagnostic weight on edrophonium (Tensilon) test, which can be false positive (even when blinded) in patients with chronic fatigue syndrome. Strong placebo response to corticosteroids may also occur in patients with functional symptoms
Pain with weakness in limbs	Place less reliability on positive signs of functional weakness in presence of pain. Ask patient if they think pain is the reason their limb is weak
Paroxysmal dyskinesia especially with aura and urge to move	Familiarity with clinical presentation of organic movement disorders
Tics/Tourette's syndrome	Ability to suppress with rebound movements, (may be distractible)

Chameleons: Features of functional motor symptoms that can wrongly put you off the diagnosis*	
Variable ankle clonus	Happens
Facial symptoms	Common
Slightly asymmetrical reflexes /mute plantar	Happens
Contractures in fixed dystonia	Happens
Migraine at onset	Migraine can trigger functional symptoms
Tremor unaffected by distraction	In chronic functional tremor motor distraction tasks sometimes no longer visibly affect the tremor. Tremorgraphy or video recording may be helpful.
Urinary retention	Appears quite common in patients with acute back pain and leg weakness in the absence of structural changes.[26] Also occurs with opiate overuse
Axial 'propriospinal' myoclonus	Usually functional[27]
Convergent spasm leading to apparent sixth nerve palsy	Look for variability over the assessment or resolution with a more distant target
Lack of pain or sensory disturbance	This should make you think twice about a diagnosis of functional motor symptoms but can occur

again in more detail.[22] Since these facial symptoms lead to an appearance of weakness (even though they are due to muscle overactivity), this can lead to erroneous diagnoses of stroke if the presentation is acute.

Slightly better known, although still commonly missed, is convergence spasm, which is relatively common in patients with functional motor symptoms.[23] You can usually bring this out by asking the patient to converge on a near target for 10 s. In convergence spasm, the convergence persists long enough to produce the appearance of impaired abduction, which can be mistaken for a sixth nerve palsy (figure 3). One way around this is to go back and test eye movements without convergence, and using a more distant target at a different point in the assessment, or just to observe eye movement during the consultation, to show the inconsistency.

Contractures can cause concern about a diagnosis of functional or fixed dystonia, but these do occur in patients who have been immobile for a long time, albeit they are rare and should at least prompt some reconsideration of the diagnosis. They can be demonstrated under anaesthesia.

There is insufficient space here to rehearse all the debates about the overlap between functional symptoms and complex regional pain syndrome. It is indisputably the case, however, that the functional motor symptoms seen in complex regional pain syndrome are clinically identical to those functional motor symptoms seen in patients without pain.[24 25] The debate would be a lot more interesting if advocates of the 'biological model' were not so ready to dismiss patients with 'psychogenic' symptoms as having symptoms of doubtful authenticity.

OTHER MIMICS AND CHAMELEONS

- *Laryngeal dystonia* (as seen in multiple system atrophy) can be confused with functional dysphonia when the otolaryngological assessment suggests that there is nothing wrong with the vocal cords.
- *Cortical blindness* (eg, from a stroke). Beware the patient who reports being blind but has normal pupillary reflexes and preserved optokinetic nystagmus. These patients should always earn an MRI brain scan as, occasionally, this is due to bilateral occipital pathology—cortical blindness. A subset of patients with cortical blindness think they can see even though they cannot (Anton's syndrome).
- *Brain injury* (diagnosed on the basis of a poor cognitive score) can be a functional chameleon. Poor concentration

Figure 2 Asymmetrical platysma contraction can arise as a functional symptom, causing facial asymmetry and diagnostic confusion. Reproduced from Fasano *et al*[22] with permission.

and memory symptoms are common as part of the 'postconcussion syndrome' after minor head injury. Although this is sometimes partly attributable to minor injury (at least for a period of weeks), there is evidence that the 'postconcussion syndrome' arises just as commonly after trauma to other parts of the body.[28] Postconcussion syndrome is predicted by psychological factors more than by the severity of the head injury,[29] and in many cases is best seen as a generic functional response to trauma, rather than a consequence of brain injury. Some patients who present late after physical injury find their way into neuropsychological assessment. If the assessor is unwary (which may include a neuropsychologist) they may interpret poor cognitive scores as unequivocal evidence of brain injury. This is especially the case if the patient scores normally on simple screening tests of anxiety and depression. Contrary to popular belief, a low score on these does not 'filter out' patients with functional symptoms. For the neurologist, it is particularly important not to be put off a diagnosis of functional cognitive symptoms by a low score on cognitive tests such as the Addenbrooke's Cognitive Examination (ACE). Tests like this are largely validated against healthy controls, and not against patients with functional disorders. Low scores on tests of attention, memory and fluency are

Figure 3 Convergent spasm can look like a sixth nerve palsy. Reproduced from Fekete *et al*,[23] with permission.

typical. The patient with a low ACE score who attends on their own is another clue that the symptoms may be functional. Even detailed psychometric examination has a very poor predictive validity in such patients, particularly in the absence of effort testing. The key message is that brain injury should always be diagnosed with regard to standard peri-injury clinical features, such as presence of loss of consciousness or amnesia, which often gets lost as time passes.

PSYCHIATRIC DISORDERS

Our bias in this article was to disentangle functional symptoms from neurological disease, but there are occasions when other psychiatric disorders need to be considered.

- *Panic attacks*. Patients with panic disorder not infrequently find their way to a neurology clinic with paraesthesia or intermittent dizziness. Panic symptoms can present as unilateral paraesthesia. Some patients have predominantly cardiorespiratory symptoms (chest tightness, palpitations and shortness of breath), whereas, others may have a more autonomic presentation (nausea, sweating, chills). Dissociation is often described as 'dizziness'.[30] Typically, the patient in the neurology clinic regards the intense fear as a reaction to the physical symptoms rather than a cause of the symptoms, but a subset of patients do not experience severe fear, so-called 'panic without fear'.[31]
- *Anxiety*. As with panic disorder, it is possible to have all the somatic symptoms of anxiety, fatigue, restlessness, irritability, poor concentration and insomnia, but not necessarily feel that anxious. Hyperventilation may also be unassociated with feelings of panic (and may cause unilateral paraesthesia and tetany).
- *Depression* is a frequent complication of neurological disease, and often is associated with poorer than expected physical function, diffuse aches and pains and marked fatigue.
- *Body dysmorphic disorder* presents with an excessive preoccupation with a bodily part that usually works normally. It is not a common presentation in neurological settings. The conviction of the concern about the affected body part is usually very noticeable.
- *Psychosis* is rarely confused with functional symptoms. Where somatic hallucinations are present, they are usually accompanied by bizarre and complex delusional explanations.
- *Mania* presents with increased motor activity accompanied by grandiosity and loosening of associations in language function. It is rarely confused with functional symptoms, but can be mistaken for encephalitis.

- *Alcohol misuse* (frequently covert) can present as a recurrent 'attack' disorder, with individual episodes of intoxication, and with a range of neurological complications[32]

FACTITIOUS DISORDER AND MALINGERING

Finally, there is one last area of diagnostic overlap with functional symptoms: the patient who is deliberately assuming symptoms in order to gain medical care (factitious disorder) or some material/financial benefit (malingering).

This is something that neurologists often worry about[33] (probably more than misdiagnosis of disease). The truth is that the only way to know if someone's symptoms are consciously produced, as opposed to genuinely experienced, is to obtain either:

- a confession, or
- evidence of a major discrepancy between reported function (what the patient tells you) and observed function (what you see the patient doing when unobserved).

In reality, such evidence rarely appears. This leaves open the possibility of exaggeration, as for any subjectively reported symptom, such as pain and depression. Evidence of lying in the past, evidence of inconsistent reporting of the history between different doctors, multiple name changes and attending multiple hospitals are clues to factitious disorder and malingering.

Some things argue against the idea that malingering is endemic—for instance, the way that patients consistently describe their subjective symptoms, have attacks when suggested during EEG, develop wear marks on their shoes, and retain disability and distress at follow-up. An important study of actigraphy in functional tremor showed that even patients who know they are being monitored are hopeless at guessing how bad their symptoms are. In this study, the 10 patients with functional tremor thought, on average, that their symptoms were present 83% of the time, when in fact they were only present 4% of the time (compared with 58% reported vs 24% observed in organic tremor).[34] In other words, the patients with functional tremor in this study probably only had the symptom when they thought about it, but this gives the illusion of the symptom being there all the time. This is a bit like assuming that the light is always on in the fridge because it appears to be so whenever you open the door!

Clinicians are sometimes asked to comment explicitly on these issues in a court setting. In personal injury settings, functional symptoms/possible functional overlay is commonplace. It is appropriate to make such diagnoses to the court, but remember that in any given individual, functional symptoms cannot be distinguished from deliberate exaggeration by clinical examination alone. Strong support of an individual's claim and testimony of their honesty in the absence of clear objective supporting evidence is a quick route to later professional embarrassment. In the context of criminal cases, diagnoses of functional symptoms are likely to only apply to fugue states and psychogenic non-epileptic attacks. Many perpetrators of violent crime report no memory of the events, but in the overwhelming majority of cases, medical explanations are not relevant. Functional diagnoses should probably not be made in this situation, and then, if they are, only where there is clear objective supporting evidence, and the diagnosis is not incompatible with the nature of the crime.

CONCLUSION

The mimics and chameleons of functional symptoms could be a whole textbook of neurology, but when things go wrong, there are some recurring themes, notably: diagnostic bias introduced by the presence or absence of psychiatric comorbidity or by life events; neurological diseases that look 'weird'; and a lack of appreciation of the more unusual features of functional symptoms themselves.

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Answer to case 1 La belle indifférence, dysphonia and drooling

This patient had a low serum caeruloplasmin, abnormal liver function tests and Kayser–Fleischer rings, confirming the diagnosis of Wilson's disease.

Even with hindsight, the dysphonia was functional. It improved with copper chelation, illustrating that patients in the early stages of degenerative diseases may present with functional symptoms (Wilson's disease possibly especially so).

'La belle indifférence' noted by her doctors was in fact executive dysfunction caused by her brain disease; it is an example of why this is such a poor 'clinical sign'. In fact, when patients with functional symptoms appear indifferent, it is commonly just the patient 'putting up a brave face' on things to avoid being labelled as mentally ill.

Drooling is a particularly characteristic feature of Wilson's disease.

Answer to case 2 A businesswoman whose 'funny face' was not funny

Examination by a neurologist found that there was variable overactivity of facial muscles on the right side of the face with intermittent jaw deviation to the right combined with platysma overactivity on that side, giving an appearance of weakness. This was in keeping with functional facial movement disorder.

The weakness in the right arm was global and 'giveway' in distribution with dense sensory loss below the elbow to all sensory modalities. There was a positive Hoover's and hip abductor sign in the right leg.

The diagnosis of functional motor and sensory symptoms was made to the patient's relief. The patient subsequently felt she had been under more stress than she recognised at the time and also identified dissociative symptoms at onset. Physiotherapy and time has led to gradual improvement

Readers in the UK can hear this patient telling her own story on BBC Radio 4 Inside Health 12th Oct 2012 along with a commentary by the authors of this article at ww.bbc.co.uk/programmes/b01n65zl.