I n this article we offer an approach to management of functional symptoms based on our own experience and on the evidence from other specialities (because the evidence from neurology is so slim). We also tackle some of the most difficult questions in this area. What causes functional symptoms? Does treatment really work? What about malingering?

We give two example cases adapted from real patients to illustrate our approach.

**WHAT CAUSES FUNCTIONAL SYMPTOMS?**

Table 1 is not comprehensive but it summarises a large literature on the suggested causes of functional symptoms. This is a question that has been approached from many angles—biological, cognitive, psychoanalytic, psychological, social, and historical. The factors shown have been found to be more common in patients with functional symptoms than in patients with similar symptoms clearly associated with disease pathology. Tables like this can help you to make a *formulation* of the aetiology of the patient’s symptoms rather than just a diagnosis. An important feature of the table is the recognition of biological as well as psychological and social factors in the production and persistence of functional symptoms.

Most of the factors in table 1 have also been associated with other types of functional somatic symptoms such as irritable bowel syndrome and chronic pain as well as with depression or anxiety. Consequently they should be regarded more as vulnerability factors for developing symptoms, than as specific explanations for why some patients develop certain symptoms such as unilateral paralysis and others have attacks that look like epilepsy. Recent functional imaging studies of patients with functional motor and sensory symptoms are beginning to offer biological clues (fig 1); they also challenge the idea that such symptoms are “all in the mind”—they are in the brain too.  

We will discuss the factors listed in table 1 later when considering an approached treatment. A fuller discussion of aetiology can be found elsewhere.

**EXPLAINING THE DIAGNOSIS**

In the first of these two articles we described our own approach to history taking and examination designed to be an efficient way of assessing the problem. The findings of this assessment will help you to *tailor* the explanation you give to individual patients. There is no “one size fits all” solution, but certain ways of saying things seem to work better than others.

Most people who develop symptoms want to know what is causing them. Explaining the diagnosis in a clear, logical, transparent, and non-offensive way is the key to management by the neurologist. It may sometimes be sufficient to produce improvement.

There are several reasons, both pragmatic and scientific, why we prefer the word “functional” in diagnoses such as “functional weakness” or “functional sensory disturbance”. It is a diagnosis that: (1) replaces an erroneous physical versus psychological debate, allowing for a more productive functional/reversible versus structural/irreversible dichotomy; (2) provides a rationale for any treatment designed to improve the functioning of the nervous system—in particular, it allows the use of both physical and psychological strategies; (3) avoids offence (fig 2) and thus can be used transparently with the patient. We do acknowledge, however, that all diagnostic terms have limitations. The usefulness of the term with patients would diminish if “functional” became seen as a euphemism for psychogenic (as it is by some doctors now).

The word you use is probably not as important as the way that you use it. We probably underestimate our patients’ ability to detect an unconvincing explanation (or one that the doctor does not really believe).

At a first encounter, our explanation of the diagnosis to the patient includes the following key points:

- **Explain what they do have**—For example, “You have ‘functional weakness’—this is a common problem. Your nervous system is not damaged but it is not working properly. That is why you cannot move your arm”.

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A composite scan of four patients with functional hemimotor and sensory symptoms compared to recovery. There was hypoactivation of the contralateral thalamus, caudate, and putamen during the symptomatic state. Activations on a scan do not tell us how the symptom came to be there (or even if it was fabricated or not), but along with studies of endocrine and immunological abnormalities challenge a purely “psychogenic” view of the problem. Reproduced from Vuilliemier et al, with permission from Oxford University Press.

### Table 1 A scheme for thinking about the aetiology of functional symptoms in neurology

<table>
<thead>
<tr>
<th>Factors</th>
<th>Biological</th>
<th>Psychological</th>
<th>Social</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposing</td>
<td>Genetic factors affecting personality</td>
<td>Poor “attachment” to parents and others</td>
<td>Childhood neglect/abuse</td>
</tr>
<tr>
<td></td>
<td>Biological vulnerabilities in nervous system?</td>
<td>Personality/coping style</td>
<td>Poor family functioning</td>
</tr>
<tr>
<td>Precipitating</td>
<td>Abnormal physiological event or state (e.g. hyperventilation, sleep deprivation, *sleep paralysis)</td>
<td>Perception of life event as negative, unexpected</td>
<td>Symptom modelling (via media or personal contact)</td>
</tr>
<tr>
<td></td>
<td>Physical injury/pain</td>
<td>Depression/ anxiety</td>
<td>Life events and difficulties</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute dissociative episode/panic attack</td>
<td>Fear/avoidance of work or family responsibilities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Perception of symptoms as being outwith personal control due to disease</td>
<td>The presence of a welfare system</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anxiety/catastrophisation about cause of symptoms</td>
<td></td>
</tr>
<tr>
<td>Perpetuating</td>
<td>Plasticity in CNS motor and sensory (including pain) pathways</td>
<td>Not being believed</td>
<td>Social benefits of being ill</td>
</tr>
<tr>
<td></td>
<td>Deconditioning (e.g. lack of physical fitness in chronic fatigue, deconditioning of vestibular responsiveness in patients with dizziness who hold their head still)</td>
<td>Avoidance of symptom provocation (e.g. exercise in fatigue)</td>
<td>Availability of legal compensation</td>
</tr>
<tr>
<td></td>
<td>Neuroendocrine and immunological abnormalities similar to those seen in depression and anxiety</td>
<td></td>
<td>Stigma of “mental illness” in society and from medical profession</td>
</tr>
</tbody>
</table>

—We pay particular attention to overcoming the patient’s fear that you do not believe them or think they are mad, imagining or putting on their symptoms. If you have found that this is something they might be concerned about, simply saying “I don’t think you’re mad, imagining or putting on your symptoms” can be very effective.

—For example, “You do not have multiple sclerosis, epilepsy, etc.”

—For example, “I see lots of patients with similar symptoms”.

—For example, “Because there is no damage you have the potential to get better.”

—For example, “I know you didn’t bring this on but there are things you can do to help it get better”.

—Metaphors and comparisons may be useful—For example, “The hardware is alright but there’s a software problem”; “It’s like a car/piano that’s out of tune, all the parts are there, they just aren’t working right together”; “It’s like a short circuit of the nervous system” (non-epileptic attacks); “It’s like the opposite of phantom limb—they feel a limb that is not there, you cannot feel a limb that is (functional weakness)”.

—Show the patient their positive signs—For example, a patient (and their family) can be shown their own Hoover’s sign or talked through a video of their non-epileptic attack. Explain how this confirms the diagnosis that the nervous system is working at some times but not at others.

—Introducing the role of depression/anxiety—Use your judgement about whether this will be helpful or harmful at an early stage. For example, “If you have been feeling low/worried that will tend to make the symptoms even worse”.

—Use written information—Using this approach makes it much easier to send the patient their clinic letter. This in turn can do a lot to persuade them of your point of view. Leaflets perform a similar function (fig 3).

—Talk to the family and friends—Reinforce the diagnosis with family or friends.

—Making a psychiatric referral—For example, “Dr X has a lot of experience and interest in helping people manage and overcome symptoms like this. Referring you to him does not mean that I think you are mad”.

—For example, “…ah now I know that your husband is having an affair, that explains you weak leg”) but instead emphasise how important they may be as a factor making the symptoms worse.

The patient may want to know why this has happened to them. An honest answer might be that you do not know or that the reasons are probably complicated. But just because...
you do not know why something happens does not mean you cannot diagnose it, or treat it. You can explain to the patient that you would have just as much trouble trying to provide a cause of their illness if they had multiple sclerosis, migraine, or Parkinson's disease.

For patients with mild symptoms, explanation and reassurance with encouragement to resume normal activity may be sufficient. In those with more resistant symptoms one or more of the following treatments may be helpful.

**PHYSICAL REHABILITATION**

Patients with physical problems often need physical treatments. Some of our best treatment successes have been accomplished by experienced neuro-physiotherapists who are able to combine hands on, physical treatments with explanation and encouragement. There have been encouraging results from studies of physical rehabilitation in patients with functional disability, but none of them has been randomised and few report long term outcome. In patients with chronic fatigue there is systematic review evidence that graded exercise is helpful overall.

**COGNITIVE BEHAVIOURAL THERAPY: HELPING THE PATIENT THINK AND BEHAVE DIFFERENTLY**

Many neurologists regard cognitive behavioural therapy (CBT) as a mysterious treatment. It is not. Essentially it is an extension of the explanation, a way of helping the patient to become aware of, examine, and if appropriate revise the way they think, respond emotionally and behave in response to symptoms. The aim is to maximise function and reduce symptoms—but not necessarily to abolish them. In formal CBT the patient meets a therapist every one or two weeks and practices new ways of thinking about and responding to their symptoms between these sessions. In common with most neurological services we do not have ready access to specialist CBT therapists. We therefore try to incorporate the principles of CBT into medical care. Before you dismiss this suggestion as unrealistic you should be aware that it was well described by neurologists practising 100 years ago. They called it “rational persuasion” or “re-education”.

CBT emphasises the interaction of cognitive, behavioural, emotional, and physiological factors in perpetuating symptoms (fig 4). The patient’s cognitive interpretation of bodily symptoms is key. This will depend on their knowledge and experience of disease. For example, it is hardly surprising if someone with paralysis misinterprets their symptoms as multiple sclerosis or a stroke and as evidence of irreversible damage to their nervous system. In keeping with these beliefs the patient may behave in a way that seems sensible but which actually make the problem worse—for example, by avoiding behaviours or situations that exacerbate the symptom (such as exercise) or endlessly seeking a medical diagnosis before considering treatment. These factors have been demonstrated to be relevant to both the aetiology and treatment of chronic pain and chronic fatigue.

Evidence exists at systematic review level that CBT is effective for a wide range of functional somatic symptoms. Its use has also been described (although not properly tested) in patients with non-epileptic attacks, motor symptoms, and severe and multiple functional symptoms. How can you do it? Think back to the 35 year old patient with disabling unilateral weakness, fatigue, and pain (as well as anger about being told that her symptoms are “psychological”) described
at the start of our first article. The following are some of the elements of CBT that you could incorporate into the consultation:

- Accept all symptoms at face value and give a positive explanation.
- Persuade the patient that change is possible, they are not “damaged”, and they do have the potential to recover.
- Give the patient a rationale for treatment—for example, exercise will help recondition your muscles and “tune up” your nervous system.
- Encourage activity, warning the patient that they may feel temporarily worse afterwards but that there will be benefits in the long term. Warn against “over doing it” on good days as much as “under doing it” on bad days. Explain that symptoms tend to be cyclical anyway and predict days when they will feel “back to square one”. Start with small goals such as walking around the garden once and build up.
- Establish a sleep routine. Give simple advice such as avoiding sleep during the day, getting up at a specified time, and getting out of bed for 15 minutes rather than lying worrying awake at night.
- Encourage the patient to reconsider unhelpful and negative thoughts. For example, patients with pain commonly think “If I go for a walk I’ll feel much worse afterwards and I’ll make the ‘wear and tear’ that my GP told me about worse”. Instead the patient should be encouraged to consider and test out an alternative possibility such as “If I go for that walk, I will be sore afterwards for a bit but in fact it will be strengthening my bones and muscles not making them worse and it will help build my confidence”.
- Look for modifiable physiological/dissociative trigger factors. If you can find evidence of dissociative or autonomic symptoms before symptom onset this may provide an additional extra rationale for this type of treatment. For example, if a patient with non-epileptic

**Figure 4** A model of perpetuating factors in functional symptoms (in this example, fatigue) illustrating some targets for treatment with cognitive behavioural therapy and antidepressants.
A 32 year old man was admitted for investigation of an abnormal foot posture. Twelve months earlier he had been in a car accident when he had twisted his right ankle. Although there was no head injury he only has a hazy memory of the accident and remembers feeling spaced out and shocked that he had crashed. There was no bony injury but instead of making a normal recovery his ankle gradually inverted until it remained in a fixed position (fig 6). In addition he complained of a lot of back pain and had gradually developed weakness of the whole leg. On questioning he said he felt constantly exhausted and had trouble sleeping. There was a history of recurrent groin pain which had not been diagnosed but had resolved. He was taking dihydrocodeine which only “took the edge off” his back pain. He had been working long hours as a van driver and said that he really enjoyed his job which he had been unable to do since the accident. He said he was not depressed but he found it hard to describe anything he enjoyed doing. His family had seen a big change in his personality. They said his ankle remained like that when he was asleep. There was no compensation case.

On examination he looked miserable. He had collapsing weakness of all the muscles in his right arm and leg and Hoover’s sign was strongly positive. The ankle was in a fixed inverted position. Attempts to straighten it produced irregular shaking in the whole leg. Vibration sense and light touch were altered over the whole of the right side of his body including his face. Magnetic resonance imaging, neurophysiology, and other tests were normal.

What would you do? How would you explain the diagnosis?
What kinds of treatment might help?
Read the outcome of case 1 at the end of the article

attacks discloses such symptoms a CBT approach similar to that used for panic (in which the patient is encouraged to be aware of “catastrophic” interpretations of symptoms and to consider more benign ones) can be used. In our experience, patients with paralysis, who also often report these symptoms just before onset and can have a relapsing course, can be helped by explaining the link between these “warning symptoms” and their weakness.

Looking at obstacles to recovery. Rather than focus on possible “causes” from the past such as an unhappy childhood, focus on obstacles to recovery. These may include the hated job which they will have to return to, the legal case that may not settle for five years, and the benefit trap. It still surprises us how often these issues can discussed openly and frankly and the patient encouraged to actively address them once you have won the patient’s trust.

Provide information about more detailed written material. There is so far little available but a self-help book on fatigue can help the patient get started by themselves. If you think they’ll accept it there are plenty of CBT based self help books for depression and anxiety.

Further elements of treatment could involve spending more time discussing these explanations with key family members, discussing with the patient how to explain their

improvement to other people, and negotiating a phased return to work.

As a final stage it may be possible to start making links between symptoms and stressful events—for example, “When you had that terrible day what were you thinking about—do you think that was relevant?”. This final stage is often the one that many doctors think should be at the “front end” of treatment, not appreciating that it can be one of the most difficult to pull off and, in our experience, not always essential in order to make a recovery.

Reading this you may think “This is impossible to fit in to my busy neurology clinic”. But if you are in the habit of providing constructive explanations for symptoms and some pointers about what to do about them, you are probably already practising a CBT approach.

ANTIDEPRESSANTS AND OTHER DRUGS
Antidepressants can help many patients with functional symptoms, even those who are not depressed. A recent systematic review of antidepressant treatment in patients with a range of functional symptoms found a number needed to treat of only three (comparing favourably with many treatments in neurology). In practice, their use may be difficult because of: (1) psychiatric stigma; (2) a perception that they are addictive or harmful; and (3) side effects. All these therefore need to be discussed before prescribing—for example, saying to the patient: “So-called antidepressants often help these symptoms even in patients who are not feeling depressed. They have wider actions than treating depression—for example on pain, sleep, and appetite—and can reverse the abnormalities in brain function we have talked about”.

The patient is best warned that they will probably experience side effects but that these will tend to wear off eventually so the treatment should be persisted with for at
Case 2: Uncontrollable shaking

A 24 year old auxiliary nurse working in a care home for the learning disabled was admitted with a three month history of shaking attacks. These were occurring up to five times a day and lasting between 2–10 minutes (fig 7). She initially did not report any prodromal symptoms but on direct questioning admitted to strong and frightening depersonalisation and a warm feeling in the minute or so before some attacks. During some of the attacks she reported being able to hear people around her but being unable to respond. She had been getting the feeling that ward staff thought she was ‘‘making it up’’. She sometimes slept after the attack, but could be tearful. Otherwise she reported exhaustion, poor sleep, and poor concentration. She had a history of chronic pelvic pain and laparoscopic surgery. She was on anticonvulsants from a different hospital and had been told that she had epilepsy. Her case notes documented a history of childhood sexual abuse and previous contact with psychiatrists for depression. She angrily denied feeling low or anxious or any recent life stresses. She said that although her job could be challenging it was also very rewarding and she was upset to be off work. She had just left the parental home to live with her boyfriend. Examination was normal. During a witnessed attack, her eyes were observed to be shut, she had an increasingly fast respiratory rate, and limb movements were asynchronous. EEG during one of the attacks was normal.

How are you going to explain the diagnosis? How are you going to handle the issue of her history of sexual abuse? What would you do to help?

Read the outcome of case 2 at the end of the article

least four weeks. They should also be warned not to expect any benefit until this time.

There is not much evidence to guide us in the choice of antidepressant agent. Tricyclic antidepressants are particularly helpful in patients with insomnia and pain but can cause unacceptable drowsiness and a dry mouth. Selective serotonin reuptake inhibitors (SSRIs) and other more selective drugs have similar efficacy and are probably preferable in the medically unwell and elderly, but also have side effects such as nausea. They also may not be so good at treating pain. Other drugs used in chronic pain such as gabapentin should also be considered. Antidepressants do not help the symptom of depersonalisation. We generally explain to patients that they can get better without the tablets—which is true—but that they are worth trying if they want to ‘‘explore every therapeutic avenue’’.

OTHER MANAGEMENT TECHNIQUES

Hypnosis and intravenous sedation

There is some evidence for the use of hypnosis in patients with functional motor symptoms. Alternatively, examination under sedation can be used therapeutically to demonstrate to the neurologist, and by means of video to the patient, that an apparently paralysed limb can move or a fixed dystonic foot is not fixed. These methods merit more systematic study.

Psychodynamic and other types of psychotherapy

In classical psychodynamic theory, conversion disorder implies that distress resulting from intolerable mental conflict is converted in to a somatic symptom with consequent relief of distress. Another facet of this theory is symbolism—for example, where a symptom like a pseudo-seizure may be said to represent a symbolic re-enactment of childhood sexual abuse. Symbolism is hard to test but the evidence overall is not consistent with ‘‘conversion’’. For example, the more somatic symptoms a patient has the more emotional symptoms they will also tend to have. In addition, in our experience, when a patient looks as if they are not distressed, they often are—they just do not want to tell you about it.

More recent psychodynamic theory has moved on from the ideas above and instead highlights the importance of early relationships and their effect on the relationships people form as adults. For example, it is plausible that poor parenting could produce interpersonal dependency in adulthood. If this dependency and excessive ‘‘attachment behaviour’’ was to a doctor or a family member only interested in physical problems you can begin to see how a tendency to repeatedly present somatic complaints might develop. Adverse experience in childhood may also influence the person’s tendency to develop certain symptoms. For example, chronic pelvic pain complaints are more common in women who have been sexually abused.

In non-specialist practice awareness of these factors may help to make patients’ otherwise inexplicable symptoms more understandable. But they are hard to make use of in treatment. For some patients a more in depth psychotherapy that helps the patient to make links with these antecedents of symptoms may be of value, but has not been evaluated in randomised trials.

WHICH PATIENTS SHOULD BE REFERRED TO A PSYCHIATRIST?

Remember that if you sent everyone you see with functional symptoms to a psychiatrist, you will be sending one third of your neurology clinic to them. Patients with mild symptoms, symptoms that respond very positively to the initial explanation, or those with a good GP will probably not need (or accept) the complication of another referral. However, a patient who is unimproved after a receiving a careful explanation, a trial of antidepressants and psychotherapy should probably be referred. A patient who has previously not been helped by experienced psychiatric intervention is
probably unlikely to benefit. If you do need to refer to a psychiatrist it is often easier to do this at a second appointment. Ideally, the psychiatrist will have been interested and experienced in managing patients with somatic symptoms. A liaison or neuropsychiatrist, if available, is often best.

DIFFICULT MANAGEMENT ISSUES

Litigation
There is no doubt that simulation in order to make money (fig 5) does occur (as discussed in more detail below). Furthermore, very few of us would refuse the opportunity to make some money if it was suggested we might be entitled to it. Leaving aside the financial motivation, compensation is a potent obstacle to recovery for all patients regardless of whether their symptoms are functional or not. Seeking compensation implicitly involves a commitment to: (1) the idea that there is physical injury/damage; (2) the notion that someone else is to blame for the symptoms now and in the past; and (3) a desire on the patients part to “prove” that they really do have the symptom. Many patients with functional symptoms who are not seeking compensation share these feelings about their symptoms, often because they feel no-one believes them and they are being accused of imagining their symptoms. It is hardly surprising then that in the context of an injury many should get involved with a legal process that is prepared to back them up and get them some money in the process. These complex issues and the negative role that lawyers and doctors can play in this process are discussed in a highly readable if polemical book, Whiplash and other useful illnesses.

Disability and incapacity benefits
Should a patient with functional symptoms receive disability benefits? Such benefits can be substantial, be more than previous earnings, and can lead to a situation where a patient will lose money if they get better. This dilemma may be usefully discussed openly with the patient. Remember that there is little research to support the idea that secondary gain is a greater factor in patients with functional symptoms than in those with disease.

Aids and appliances
A similar dilemma arises when thinking about aids such as sticks and wheelchairs. They can be both helpful, in improving independence and confidence, and harmful, leading to dependence on them and decreased activity. Each case must be evaluated on its merits.

THE PATIENT WHO DOES NOT GET BETTER

Many patients with functional symptoms are hard to treat and follow a lifelong course of symptoms, disability, and medical consultations. It is important to have realistic expectations about who can be helped and to accept that you may have to treat several to make a big difference to one. If you have made an effort with a particular patient but it is clear that they do not really believe that things can change (or you do not have any more resources to help) then this may be the point at which to ask the GP to take over their long term chronic care. Rather then ending contact on a negative note you may wish to tell the patients that they are coping well with a difficult illness and that you are sorry you have not been able to help more.

If the patient has a history of repeated presentation to secondary care with the associated risk of iatrogenic harm, a positive plan to “contain” the patient in primary care may help—for example, by the GP making regular monthly appointments regardless of whether there are new symptoms or not. This in itself may reduce the number of new symptoms and will enable more optimal management of things like recurrent depression, but the GP may still need to ask you to review the patient from time to time.

PROGNOSIS

Symptomatic recovery and other measures of outcome
The natural history of functional symptoms in neurology has not been well described. In outpatients, a third to a half of patients can be expected to be unchanged or worse a year after diagnosis. Symptom persistence is more likely for those with motor symptoms or pseudoseizures than just sensory symptoms. Some patients will develop other functional symptoms and attend multiple medical specialities; iatrogenic harm from unwarranted surgery and drugs is a major problem. Known predictors of poor outcome are long symptom duration and personality disorder.

Misdiagnosis
Since an influential paper by Slater in the 1960s, many doctors have been worried that a high proportion of patients with functional symptoms, like paralysis and non-epileptic attacks, will go on to develop disease that with hindsight explains their symptoms. In fact, a number of recent studies have reported rates of misdiagnosis of around 5% in regional and tertiary neurological centres. This misdiagnosis rate is similar to that of other neurological disorders. In practice this means that occasionally you will get the diagnosis wrong and so you should be willing to re-evaluate the patient. When misdiagnosis does occur, it is most common in gait and movement disorder and where the clinician has placed too much emphasis on a bizarre or “psychiatric” presentation.

MALINGERING AND FACTITIOUS DISORDER: IS THE PATIENT MAKING IT UP?

Discriminating between consciously produced and unconsciously produced functional symptoms is difficult, if not impossible. Patients’ awareness of control over a symptom like paralysis is probably not “all or nothing” but rather on a continuum. It may also vary over time so that a patient may begin an illness with little awareness about what is happening but gradually gain a degree of conscious control with time (or vice versa).

Doctors are almost certainly worse at detecting deception by a patient than we would like to think. A recent study where examiners were blind to whether subjects were feigning paralysis or genuinely experiencing it during a hypnotic state showed no greater than chance performance. As Miller put it, the detection of malingering is “nothing more infallible than one man’s assessment of what is probably going on in another man’s mind”. The only investigations that reliably tell you that someone is malingering are covert surveillance demonstrating a major discrepancy in function or a direct confession, but these are rarely obtained outside medicolegal scenarios. Functional imaging of the brain is opening up new possibilities of detecting differences in intention and action that are not visible clinically, but these remain experimental and may never be reliable. When conscious intention is discovered or confessed, a distinction must be made between those patients generating symptoms or behaviour merely to gain “medical
What would you do? How would you explain the diagnosis?
The clinical features were consistent with a functional dystonia associated with back pain, fatigue, and functional right sided weakness. However, the dystonia portion of the diagnosis was left as “probably functional” initially. We were also concerned about the possibility of a contracture. We opted to treat the other aspects of his illness first to see what would happen. He was given a positive explanation for the symptoms and shown a video of another patient with the same problem. We explained the role of physiotherapy and of antidepressants (even though we agreed that he was not feeling depressed). As a result of this he fairly quickly felt confident about the diagnosis and his back pain, fatigue, and weakness began to slowly improve. He started to make plans for the future and discussed changing his job if and when he could recover. He admitted that he had been feeling very frustrated and low in recent months. His foot, however, remained stubbornly inverted.

What kinds of treatment would you attempt?
Six months later, in the absence of any improvement in his ankle posture, he was brought in for videotaped sedation. In a highly clinical environment he was given a titrated dose of propofol so that he was awake but drowsy. In this state he was able to move his ankle normally for the first time in one and a half years. He was also able to watch the video afterwards. A few days later there was a “breakthrough” in his treatment and his foot straightened and started moving normally. He now is back at work and walking normally. Although he is not completely asymptomatic he is very grateful for his recovery. Asked what had made the difference he said he was terrified that something in his medial ankle would “snap” if it ever came back to a normal position. We made a diagnosis of definite functional dystonia.

Case 1: Outcome

Case 2: Outcome

How are you going to explain the diagnosis?
The history and investigations are typical for non-epileptic attacks. This is exactly the kind of patient in whom it can be counterproductive to wade in with a psychological explanation. She has angrily denied any emotional symptoms and has already been given a diagnosis of epilepsy, a valid diagnosis that may be difficult to take away. Non-epileptic attacks can be explained as a common and treatable temporary “short circuit” of the nervous system but without the abnormal electrical activity of epilepsy. Repeated reminders that you do not think she is making the symptoms up will probably be necessary. Written information including a leaflet and a copy of your letter will help confirm to the patient and her family that this is a real diagnosis and that you are not just waffling.

How are you going to handle the issue of childhood sexual abuse?
It is usually advisable to make connections between remote events such as this and current symptoms unless you have established a good relationship with the patient over a period of time. Even if you think there is a connection between her abuse, her pelvic pain, and the non-epileptic attacks—and there may be—the patient is unlikely to want to hear your thoughts on the matter or be helped by them, at least for some time.

What would you do to help?
The most important first step is to undo the diagnosis of epilepsy and make a clear and unequivocal diagnosis of non-epileptic attacks. Her anticonvulsants should be stopped. The presence of prodromal symptoms is helpful. Explain that depersonalisation is common and not life threatening. Ask her to try rebreathing into a paper bag over her mouth and nose the next time she feels it coming on. Explain how patients can learn to stop attacks like these in time with practice. Make a careful referral to a liaison psychiatrist explaining you don’t think she’s crazy, but Dr X has special expertise in these kinds of symptoms. If you manage to get her to turn up for the appointment the psychiatrist ought to be able to engage her, begin to explore psychological aspects, and monitor progress. Ideally the neurologist should make sure the patient believes/understands the diagnosis at follow up as well.

CONCLUSION
Early management of functional symptoms involves demonstrating to the patient that you believe them and that you recognise their symptoms as being common and potentially reversible. A lot more research is needed in to the optimum approach but our experience is that using the “functional model” of symptom generation allows a transparent explanation and interaction with the patient that can facilitate later physical and psychological treatments. Much of the core of a cognitive behavioural approach to treatment is in fact simple advice about exercise, sleep, and ways about thinking about symptoms that can be given effectively by a neurologist. While it is unreasonable to expect everyone to get better, it is also a mistake to think that a neurologist cannot make a difference, even in a limited time.

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