Functional neurological symptoms.

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FUNCTIONAL NEUROLOGICAL SYMPTOMS

I was pleased to see the article by Jon Stone in the *Journal*, in which he provided an overview of functional neurological disease (Stone J. Functional neurological symptoms. *J R Coll Physicians Edinb* 2011;41:38–42.)

Having read the article, I found it very superficial with many other unequivocal signs of functional illness being omitted from the text, which I find somewhat disappointing as this is the most important area for continuing medical education.

There was absolutely no emphasis put on sensory testing, which is a very important consideration when exploring functional illness in neurology. Perhaps the most obvious area where this appears is in the examination of facial sensation, in which patients will often report change in sensation at the hairline or the angle of the jaw while the anatomical demarcation is the inter-aural plane rather than the hairline and a line from the tragus to just below the midline of the chin rather than the angle of the jaw. Similarly, a lack of midline demarcation is a clear and unequivocal sign of functional illness. Testing sensation, moving from the periphery centrally, an area of perceived change and sensation (from decreased to increased perception) should be fairly discrete and if it traverses a number of centimetres travelling along the same course on repeated testing, it is an unequivocal sign of functional illness. Similarly sensory changes that do not respect either peripheral nerves, radicular dermatomal distribution or very restricted isolated nerve distribution suggest functional illness of non-organic type but this received no mention in the article by Professor Stone.

Professor Stone rightly touched upon gait and Hoover's sign when describing functional illness but made no comment of the use of synergistic muscles when testing weakened power. A perfect example of this is testing triceps power, having fully supinated the elbow to switch off brachioradialis, and noting activity in the biceps (the antagonistic muscle), which should be completely at ease if the triceps power has been fully tested. The same applies to lower limb testing where activity of antagonistic muscles should not be noted if maximal power is being tested of the muscles under review.

I thought the table provided by Professor Stone was excellent for the differentiation of non-epileptic and epileptic seizures, although in addition might be the presence of cyanosis as this is quite uncommon in dissociative attacks and very common in convulsive epileptic seizures.

It must be said that often non-epileptic seizures are more difficult to differentiate from epileptic seizures, even with the addition of video telemetric evaluation but in general I thought that Professor Stone dealt with the question of epilepsy far better than he did with the non-epileptic presentation of non-organic disease.

What did not appear in the discussion of management of the functional neurological symptoms was the question of conversion reactions, which require both a model and secondary gain. This was basically ignored when considering treatment but it has been my personal experience that without discovering what is the secondary gain, the approach to management is far less effective. Once the secondary gain has been identified then it is possible to seek alternative methods of satisfying this need, a point largely ignored in the paper.

I fully endorse the comment by Professor Stone regarding the use of disinterested psychiatrists and the need to create rapport with a counsellor who is both attuned to the patient's needs and is prepared to put in the hard yards necessary to both create rapport with the patient and to sort out the model, secondary gain and alternative means of satisfying the patient's needs.

I also fully agree with Professor Stone on the notion that one has to be completely honest with the patient at all times and if one is so honest then one has the opportunity to establish a level of rapport which can engender trust and respect which are absolute necessities if one is to achieve benefit for the patient.

In conclusion I feel the topic of this educational paper was very important but the superficial nature of the submission made it less valuable than it might have been.

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Author's response

Thank you to Professor Beran for his letter.

I was given a strict word limit for this article which was designed as a CME module. It was chosen for publication from other CME modules. I had to cover a large topic for a general audience – the main aim being to introduce and educate non-specialists about the topic and to give some practical advice about initial management in the acute situation. Consequently a large number of potential areas for discussion had to be excluded.

Had I been given a different brief, for example, a comprehensive review of the whole topic, then it would have been much longer. Indeed I have written much longer pieces elsewhere. Hopefully Professor Beran will not feel so critical of the article's superficial nature if he realises this.

With respect to the points he makes, I attempted to describe discriminating signs with some evidence base. Unfortunately, sensory signs, while often used in practice,

do not have a good evidence base. When certain 'non-organic' sensory signs have been tested against disease controls they performed quite badly (see Stone et al[1]). This is also my own personal experience and I do not share his confidence about the sensory signs he suggests. I have been systematically looking for these 'unequivocal signs' in patients with 'organic disease' and find them to be not uncommonly present when they are supposed not to be. This could be because of functional overlay or it could be that sensory signs (in keeping with the literature on reliability of signs in general) are not that reliable since they rely on the patient's subjective report.

With respect to co-contraction of antagonists you will see that I do describe this in my review article but there was not space for it in this learning module. While I do use it, I don't think it is a helpful sign for the non-neurologist to try to learn (many of my consultant colleagues are not even sure what pyramidal weakness is) and is much harder to be certain about (or indeed show to the patient) than, for example, Hoover's sign.

With respect to other factors such as secondary gain, modelling of symptoms etc, once again the primary literature has failed to demonstrate that these issues are as clear cut as they appear to be in older textbooks of neurology and psychiatry. The problem is that clinicians do not look hard for these things in patients with organic disease where they are often present. There are no controlled studies of secondary gain in conversion disorder; it's a hard thing to study but easy to find. Studies of modelling conflict on how common this is in patients with disease (so in that case how specific is it?). I agree that all these things can be relevant in individual patients but it would be reckless to use them diagnostically and very hard for general physicians to use them fruitfully in initial treatment which is what this article was focusing on. A physician inexperienced in the area who introduces these topics with a patient at an early stage is likely to threaten the rapport that both Professor Beran and I seem to agree is important for treatment.

There are numerous other potentially relevant aetiological factors that I didn't discuss either in my short paragraph on aetiology.

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