

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 al¹). 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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SOUTH EAST SCOTLAND EXPERIENCE OF HIV TESTING IN PATIENTS NEWLY DIAGNOSED WITH LYMPHOMA

We read with interest the article entitled 'The impact of new national HIV testing guidelines at a district general hospital in an area of high HIV seroprevalence.' (Page 1,

Philips M, Flegg P, et al. *J R Coll Physicians Edinb* 2011;41:9–12). Haematology as a specialty is responsible for the care of patients with many of the conditions for which routine HIV testing is recommended in the British HIV Association (BHIVA) guidelines discussed in the article. Lothian, in South East Scotland, is identified as an area with high seroprevalence of HIV infection and as such all patients newly presenting to general or specialist medicine, including haematology, in this region should be considered for HIV testing according to BHIVA guidelines.

The South East Scotland Cancer Network (SCAN) was formed in 2000 with the aim of improving regional cancer service delivery. The Haematology Group includes departments within the Lothian, Fife and Borders Health Boards. Since 2005, comprehensive population-based data have been collected regarding all aspects of lymphoma diagnosis, including the number of patients undergoing HIV testing. As part of a quality initiative, specific attempts have been made to raise physician awareness of the potential for HIV testing in newly diagnosed patients with lymphoma. In 2006, across all three health boards, 7.6% of patients diagnosed with lymphoma (19 of 249) were tested for HIV. In 2009 this figure had improved to 32.4% (91 of 281 patients) but, perhaps reflecting the varying perceptions of risk, in 2009 test rates varied between 42.2% for urban residents in Lothian and 6.5% for the more rural populations covered by other health boards.

Overall testing at 32.4% of the at-risk population is still clearly short of our current 100% target but, of note, the number of positive cases identified each year has remained remarkably stable with 2/8 patients positive in 2005; 2/19 in 2006; 2/34 in 2007; 2/59 in 2008 and 3/91 in 2009. Additionally, it was a new diagnosis of HIV in only two of the 11 patients with lymphoma who were positive over this period. Thus, although it is clearly important to increase the number of patients tested for HIV, doing so may only produce a modest rise in the number of HIV-positive patients being identified directly as a result of a new lymphoma diagnosis. Any increase, however, is clinically highly relevant given that combination treatment of HIV-positive patients, with Highly Active Antiretroviral Therapy (HAART) and standard lymphoma treatments now results in survival rates approaching those of HIV-negative patients.

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THE ROUTINE USE OF FLUMAZENIL INFUSION FOLLOWING PERCUTANEOUS ENDOSCOPIC GASTROSTOMY PLACEMENT TO REDUCE EARLY POST-PROCEDURE MORTALITY

We read with great interest the article on the use of a flumazenil infusion following percutaneous endoscopic gastrostomy (PEG) placement to reduce early post-procedure mortality. (Bosanko NC, Barrett D, Emm C et al. The routine use of flumazenil infusion following percutaneous endoscopic gastrostomy placement to reduce early post-procedure mortality. *J R Coll Physicians Edinb* 2010; 40:111–14.). The authors' conclusion was that flumazenil infusion may have a role to play in patients with high risk of aspiration. This is despite not showing a difference in one month mortality post-gastrostomy placement. They advocate future randomised studies to investigate the benefits of flumazenil further. We feel that their data shows no role for flumazenil and that routine use of and reliance on this drug may worsen outcomes. They quote a 30-day mortality rate of 25.2% and a mean sedation of 4 mg of midazolam (2007–2008) and range between 1–10 mg.

Since the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report in 2004¹ we routinely audit our PEG insertions across North Tees and Hartlepool NHS Foundation Trust. We compared our most recent data to that reported in Bosanko et al's study. We identified 44 patients who underwent PEG placement in 2009. Of the total number of patients, 86% received sedation: range 1–7 mg of Midazolam, mean 1.9 mg. Of these, 18.1% received fentanyl (25–50 micrograms). The rest of the group (14%) received pharyngeal anaesthesia alone. No benzodiazepines antagonist reversal was used. During that year one-week mortality rate was 5% and one-month mortality rate was 12%.

Major complications are more likely to occur in elderly patients with multiple comorbidities. Patient selection is the most important factor to reduce mortality.² Predictors of poor outcome include advanced age, poor nutritional status, systemic infection and severe co-morbidities.³ The most common complication during endoscopy is aspiration which is usually precipitated by sedation, avoiding pharyngeal anaesthesia and ensuring oropharyngeal suction.⁴

There is clinical evidence that judicious use of sedative reduces aspiration.^{5,6} In your paper we noticed a high 30-day mortality rate post-gastrostomy insertion compared to our own results. A possible culprit was excessive sedation given to elderly patients with multiple comorbidities and longer half-life elimination, therefore potential side-effects. The paper clearly demonstrated that reversal of sedation with flumazenil did not alter the outcome for the patients undergoing PEG insertion. The British Society of Gastroenterology advised no more than

2 mg sedation of midazolam in patients above the age of 70-years-old.⁷ We should not be advocating routine use of flumazenil. Instead we should concentrate on careful patient selection, optimisation of pre-procedure nutrition and minimising sedation during the procedure.

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Authors' response

We thank Dr Porras Perez et al for their interest in our paper. As indicated in our conclusion, we accept that our data did not justify the routine use of a flumazenil infusion following percutaneous endoscopic gastrostomy (PEG) insertion. However, in line with our hypothesis, we pointed out that there may be a role in selected groups to entirely eliminate the effect of benzodiazepine sedation in the immediate post-PEG insertion period but that this could only be investigated with randomised trials. We also agree that patient selection and judicious use of sedation, in line with national guidance are key to improving outcomes. At present we also are embracing the technique of inserting PEG tubes under pharyngeal anaesthesia only for selected patients.

Our technique for PEG placement has evolved as in our paper. We have adjusted sedation doses and techniques and we continually audit and act on the results. We would argue that this is an example of the usefulness of clinical audit.

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PHYSICIAN INVOLVEMENT ENHANCES CODING ACCURACY

I would concur with Nallasivan et al (Nallasivan S, Gillott T, Kamath S et al. Physician involvement enhances coding accuracy to ensure national standards: an initiative to improve awareness among new junior trainees. *J R Coll Physicians Edinb* 2011; 41:106–8. doi:10.4997/JRCPE.2011.220) that greater physician involvement is required to enhance accuracy of coding. Coding inaccuracies seem to be particularly prevalent in interventional specialities as noted by the Audit Commission. There is also significant national variation, between 0.3 and 52% across Acute Trusts in England, with the potential for gross financial disparity.¹ We have also previously demonstrated significant inaccuracies in coding in the field of interventional pulmonology, with greater than 15% coding inaccuracy in a single centre for endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA), and even greater than 68% inaccuracy for medical thoracoscopy with estimated financial discrepancies of at least £65,000 for one procedure in one centre annually.^{2,3}

Coding inaccuracies are particularly key in EBUS-TBNA which attracts a specific tariff roughly six times the magnitude of conventional bronchoscopy.⁴ Coding inaccuracies therefore have potentially more financial consequences, with interventional procedures that attract higher tariffs. As a step towards reducing coding inaccuracy, we have altered the notification of our EBUS-TBNA reports, prospectively independently notifying all interventional procedures with the coding team and also a monthly checklist issued back by the coding team as a confirmatory double check. In the last 12 months of eighty EBUS-TBNA procedures, all have been coded correctly. Providing a safety net in this way, with greater physician engagement has made important progress in reducing coding inaccuracy and financial disparity.

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Authors' response

I fully accept the views of Dr Medford and his observations on interventional pulmonology.

As a budding rheumatologist, we have only a few interventional procedures that can be coded under different tariffs. Most procedures are done as outpatient settings alongside clinic reviews.

In specialities where intervention is fast developing, like cardiology, pulmonology, gastroenterology and renal medicine, inaccurate coding would incur a significant loss to the balance sheet for the Trust. We would also appreciate the difficulties in the coding department when the audit commission is on the roll. I would stress that concerted efforts by the concerned physician would benefit the coding and finance and also ensure patient safety.

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ERRATUM

We would like to correct an error in the article on Andrew Rae Gilchrist (1899–1995) by Derek Doyle in our June issue (*J R Coll Physicians Edin* 2011; 41:185). Rae Gilchrist's first wife was Emily Slater (Faulds was her middle name). She was the sister of Dr James K Slater, no doubt familiar to many Fellows.

Please note that opinions expressed in Letters to the Editor should not be taken as those of the Editorial team or the Royal College of Physicians of Edinburgh.