in a 'Soap' suffering a stroke or TIA. Unfortunately (especially for those of us trying to increase public awareness), few TIAs or strokes ever seem to happen within the communities of East Enders, Neighbours or Coronation Street where cancer, trauma and heart attacks continue to dominate medical practice!

COMMENT ON

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S Aroori, N Blencowe, G Pye, R West. *Clostridium difficile*: how much do hospital staff know about it? *Ann R Coll Surg Engl* 2009; **91**: 464–9

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***Clostridium difficile* infection: more known than is realised**

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The reported results only partially agree with our previously published study, which surveyed 63 doctors and 79 nurses.¹ In our true/false questionnaire, the statement '*Clostridium difficile* can cause multiple syndromes, one of which is pseudomembranous colitis' was identified as correct by 92% and 94% of nurses and doctors, respectively. We are concerned that Aroori *et al.* stated that the only correct answer to Question 7 ('Which of the following conditions may be due to *C. difficile* infection (CDI)?') was 'toxic megacolon'. It is widely acknowledged that pseudomembranous colitis and antibiotic-associated colitis may also be due to CDI.

Clarification of Question 13 about the gold-standard test for identification of pathogenic CDI is also needed. The answer given was 'cytotoxin assay for detecting cytotoxin B'. The gold-standard test for CDI according to the Health Protection Agency is a cell culture based method,² whereby cytopathic effect (CPE) is observed after overnight culture of stool in a monkey verocell sheet assay, and neutralisation of any CPE observed with specific anti-sera. This detects both toxins A and B, and should not be confused with the most common tests (enzyme immuno assays or ELISAs) used to detect *C. difficile* toxins. There are advantages and disadvantages with both methods,² and many laboratories are now adopting a two-step testing procedure.^{2,3} At our hospital, we perform an immediate EIA and aim to issue

any reactive results within 4 h, and then proceed to confirmation by overnight detection of CPE in cell-culture.

Therefore, we take issue with the conclusion that there is a significant lack of knowledge concerning CDI among healthcare professionals, in particular nurses and consultants. Undoubtedly, as the epidemiology of CDI is changing, education of hospital staff should be continuous, and adequately updated.

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AUTHOR'S RESPONSE

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As the authors rightly pointed out, *Clostridium difficile* infection (CDI) can cause not only toxic megacolon, but also other conditions such as antibiotic-associated diarrhoea and pseudomembranous colitis. The answer to Question 7 is indeed 'all of the above' not 'toxic megacolon' alone as stated in our paper. We assure the readers of the *Annals of The Royal College of Surgeons of England* that we have taken the correct answer into account when we analysed the results. This was an error and we sincerely apologise for this mistake.

With regards to the issue raised by the authors about the gold-standard test for the diagnosis of pathogenic CDI, we acknowledge that the gold-standard test for CDI would be to test for both cytotoxin A and B, as sometimes only one of these may be positive in CDI, and apologise for being less than totally rigorous in our question. The purpose of this question was to test healthcare workers' knowledge of the principle behind the test, and in particular that this was not a microbiological 'culture and sensitivity' test, rather than exploring their understanding of detailed biochemistry. It seems unlikely that the overall response to the question would have been very different had both toxins been included in the question as perhaps they should.