tionships. Remember most doctors have no business experience and have no consideration of the importance of a 'limited liability partnership'. No book on senior doctoring would be complete without a section on clinical governance and self-regulation and all the usual aspects are covered including again 'finding out what you can do to reduce the risk of adverse events' – 'you are not working in splendid isolation but as part of as a team' we are informed, as well as the perverse dangers of intellectual arrogance.

So, is this text likely to help one succeed as a hospital doctor? Well it is certainly not a 'how to' guide, but for those who are determined to succeed and have palpably considered taking action in such a direction, it will act as a useful guide along the way.

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letters
TO THE EDITOR

Please submit letters for the Editor's consideration within three weeks of receipt of the Journal. Letters should ideally be limited to 350 words, and sent by email to: Clinicalmedicine@rcplondon.ac.uk

Thrombolysis for ischaemic stroke

In their article dealing with thrombolysis treatment for acute ischaemic stroke, Jenkins *et al* (*Clin Med* June 2008 pp 253–8) omitted mentioning two other important flaws in the National Institute of Neurological Disorders and Stroke (NINDS) trial.[1]

Firstly, the relevant part of the trial (part 2), that provided the main impetus for this form of treatment, relied on a global test statistic as the primary measure of outcome, and not on incontrovertible hard end-points. The global statistic was itself a composite function of four inter-related neurological scores (the Barthel Index, the Modified Rankin Scale, the National Institutes of Health Stroke Scale, and the Glasgow Outcome Scale). Clinically and statistically significant differences depending on any such composite of inter-related scores must be inherently suspect.

Secondly, part 2 of the trial also required that patients in the control and active treatment groups not receive aspirin in the first 24 hours. Thus, patients treated with recombinant tissue plasminogen activator (tPA) were compared to controls in receipt of suboptimal standard therapy. To overcome the risk of serious intracranial bleeding due to combined therapy with tPA and aspirin, the investigators should have administered dummy aspirin to the active treatment group and genuine aspirin to the controls.

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Reference

1 Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. *N Engl J Med* 1995; 333:1581–7.

In response

We thank Kumana and Cheung for their comments and accept that there are often inherent weaknesses and limitations in neurological scoring systems, although the degree of potential statistical error in this case is debatable in our view. We believe the core aim of the NINDS study was to demonstrate improvement in disability following thrombolysis and, in this respect, we consider that the end-points chosen by the investigators were reasonable. We agree with Kumana and Cheung that the omission of aspirin was not ideal.

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Post-infective irritable bowel syndrome and dyspepsia

Spiller, in his interesting review (*Clin Med* August 2008 pp 417–9), tabulated nine outbreaks of gastroenteritis followed by irritable bowel syndrome (IBS). However, just as IBS is merely one component of the functional irritable gut syndrome, so some after enteritis had dyspepsia as well as IBS, a phenomenon not detectable using Rome I questionnaires.

The early two Sheffield and three Nottingham studies were uncontrolled. A general practitioner database found that 12 of 318 patients with documented bacterial enteritis developed new IBS, 11 times uninfected controls.[1] In Beijing, 329 of 450 with Shigella gastroenteritis were followed for one to two years as were 243 unaffected siblings or spouses.[2] Functional bowel disorders developed in 10% who had dysentery <1 week, 31% for 1–2 weeks, and 35% >2 weeks, significantly higher than in controls. A year after a Shigella outbreak in Seoul, incidence of IBS was 17%, but 6% in age- and sex-matched healthy volunteers.[3] Two years after a Canadian *Escherichia coli*/*Campylobacter jejuni* outbreak, IBS incidence was 24% but 10% in controls.[4]

Dyspepsia was investigated in two studies. Three months after the Nottingham *C. jejuni* infections 30 with IBS scored 9 for indigestion and 8 for diarrhoea – significantly higher than 28 'controls' (6 and 3) or 40 healthy volunteers (3 and 3).[5] A one-year follow-up study of a salmonella outbreak in 1,243 Catalan villagers gave a cumulative risk of IBS of 11.6% *v* 1.5% in controls; the risk of dyspepsia (pain, discomfort, fullness, nausea) was 17.2% *v* 3.3% in controls.[6] At 12 months there were 18 villagers who had developed only dyspepsia, 9 with only IBS and 15 with both.