PostScript

LETTERS

Use of adrenaline by junior doctors

The survey reported by Gompels and colleagues showing the incorrect use of adrenaline in anaphylaxis by over 50% of junior doctors reveals sobering but perhaps not startling statistics.¹ Their study serves as an audit reflecting the quality of contemporary medical education in that it compares prevailing practice with established guidelines in the management of a given medical problem. Having spent 18 seamless months as a casualty officer recently and worked in two accident and emergency departments in large cities, I wish to highlight the observation that teaching on anaphylaxis was remarkably cursory in didactic sessions, as well as in the standard cardiorespiratory training workshops. When combined with the reality that moderate to severe anaphylaxis is seen infrequently, it is easy to appreciate how any superficial knowledge that exists passes into further obscurity over time.

The inability to tackle emergencies adequately results from inexperience, but the large gaps in basic medical know-how (in over 50% of graduates in this study) is a direct testament to their undergraduate and early postgraduate training. Medical curricula are ever expanding with concepts that the freshly minted doctor of the 21st century must absorb, but it appears that in this enormous amount of information the crucial elements are becoming indistinguishable.

Since this study is an audit perhaps we ought to “close the loop” by reappraising undergraduate training in earnest, especially now that many medical schools favour the submission of course work in monitoring progress at the exclusion of formal examination strategies. A specific and formal examination structure (perhaps a *viva voce* or written short answer questions) dedicated to the management of emergencies would be a useful adjunct to the traditional emphasis on the detection of signs in relatively stable patients. This arrangement would produce a preregistration doctor who is more confident and less dangerous under the onslaught of acute presentations and better primed for the senior house officer days. This plea for an improvement in our undergraduate and postgraduate education is particularly justified in the context of British medicine, where ironically, at the grassroots, the most junior doctors enter accident and emergency departments to find themselves managing (often independently) patients who are seriously unwell.

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Reference

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Authors' reply

We would like to thank Dr Gandhi for his views on undergraduate and postgraduate training, which I am sure are also a reflection of other people's views as well.

The purpose of our paper was to highlight the difficulties that are experienced in the differential diagnosis of acute anaphylaxis, and the management thereof. This study arose from the perception in our allergy practice that significant numbers of patients were referred who had inappropriate treatment. The purpose of this publication was to promote further education and debate, which hopefully it has achieved.

Chronic unexplained fatigue

I found the editorial on chronic fatigue syndrome by White both surprising and disappointing, because he used the title “Chronic unexplained fatigue” and the subtitle “A riddle wrapped in a mystery inside an enigma”, but his editorial, by ignoring very important facts about chronic fatigue syndrome, actually perpetuates that riddle, rather than helping to solve it.¹

If a puzzling and poorly manageable condition shares more than 40 features, including all of its diagnostic criteria, with a well known and easily treatable disease, this astounding clinical overlap should not be ignored, because reason not only suggests that the mysterious illness may simply be a form of the well known disease, but also hints that it is worthwhile assessing whether the classic therapy for that treatable disease could be effective for the enigmatic condition as well.

It is surprising, therefore, that in White's editorial there is not a single word about the 41 features that chronic fatigue syndrome shares with Addison's disease,² including chronic fatigue and all the physical signs and symptoms, neurocognitive dysfunctions, depressive complaints, and sleep disturbances listed in the diagnostic criteria for chronic fatigue syndrome.³ Nor is there a single word about the endocrine and adrenal abnormalities that chronic fatigue syndrome shares with Addison's disease—namely, hypocortisolism, impaired adrenal cortical function, reduced adrenal gland size, and antibodies against the adrenal gland.³

What is really mysterious about chronic fatigue syndrome is the fact that, despite its unequalled clinical overlap with Addison's disease (which, notably, does not necessarily include hyperpigmentation as a presenting feature³), no published study tried to determine whether the classic therapy for Addison's disease—that is, hydrocortisone plus fludrocortisone, could also be effective for treating chronic fatigue syndrome. Since both of these steroids, administered separately in low doses⁴ and in the proper form,⁵ have already been reported to be safe and remarkably beneficial in the treatment of chronic fatigue syndrome,³ it is even more mysterious that the effects of their combined administration on patients with the syndrome have yet to be investigated.

As someone whose chronic fatigue syndrome symptoms, after their reported dramatic resolution thanks to an old remedy for

Addison's disease,² are currently suppressed most effectively by low doses of both hydrocortisone and fludrocortisone, I cannot but suggest that chronic fatigue syndrome, far from being “a riddle wrapped in a mystery inside an enigma”, is merely a mild form of Addison's disease.

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Author's reply

I am pleased to learn that Dr Baschetti's “chronic fatigue syndrome symptoms . . . are currently suppressed most effectively by low doses of both hydrocortisone and fludrocortisone”, but I would not share his confidence in this being the answer to treating the syndrome. There is little evidence that chronic fatigue syndrome is “merely a mild form of Addison's disease”.

Two systematic reviews (published together) of blindly assessed, randomised controlled trials of these drugs found that fludrocortisone was ineffective and that there was insufficient positive evidence to recommend hydrocortisone.¹ Hydrocortisone caused serious adverse effects in some patients.

Although most studies do find a down-regulated hypothalamic-pituitary-adrenal (HPA) axis in patients with chronic fatigue syndrome, compared with healthy controls,² this could be the consequence of the relative inactivity or insomnia that occurs with chronic fatigue syndrome, rather than being a primary event.^{3,4} We should also remember that a down-regulated HPA axis is found in many conditions in medicine that have nothing to do with Addison's disease.⁴

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Homocysteine and Buerger's disease

We read with interest the case report by Courtney *et al.*¹ It demonstrates the need to consider hyperhomocysteinemia as a risk factor in vascular occlusive disease, and also the importance of reducing homocysteine levels in such patients. In addition, a meta-analysis of 10 case-control studies on patients with a raised concentration of homocysteine has been found to be a risk factor for venous thrombosis.² The young woman described in the report presented with recurrent ischaemic episodes involving the right leg initially, which required amputation. Two years later she had ischaemic left leg pain and six months later she developed small bowel infarction from which she did not recover. Histology during her last admission revealed a fresh thrombus with no evidence of organisation. However, in the discussion it is not mentioned explicitly whether the fresh clot was in the venous or the arterial mesenteric circulation. In addition, retrospectively one would wonder whether anticoagulation with warfarin would be a therapeutic option in such patients.

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Authors' reply

We thank Drs Umasankar and Huwez for their interest and comments about our paper, which concerned hyperhomocysteinemia as a risk factor for vascular occlusive disease.

They correctly point out that raised homocysteine is also established as a risk factor for venous thromboembolism and suggest warfarin as a therapeutic option. Meta-analyses have found an odds ratio of 2.5 to 2.95 for venous thromboembolic disease in patients with homocysteine levels greater than the 95th percentile of the control group.^{1,2} Recurrent venous thromboembolism has been shown to be more likely in patients with raised homocysteine after discontinuation of anticoagulation.³

However, the patient we reported did not have venous thrombosis and the fresh thrombus was reported in the arterial mesenteric circulation only. She did receive anticoagulation with warfarin but, regrettably, non-compliance was suggested by an international normalised ratio of 1.0 on admission to hospital.

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Clinical decision-making: coping with uncertainty

The uncertainty of clinical decision-making¹ is compounded by the fact that, notwithstanding its value, an inherent flaw in the medical history is its instability, due to the fact that "patients' recollections of their past symptoms, illnesses, and episodes of care are often inconsistent from one inquiry to the next".² Rather than castigate the patient for being a "poor historian", the authors proposed strategies to help clinicians recognise

and overcome obstacles to good history taking.² Clinical decision-making is also hampered by the absence of a clear survival advantage for conclusions based on higher levels of evidence.³ What the authors observed was that survival of conclusions did not differ between randomised and non-randomised studies in a selected field, namely, cirrhosis and hepatitis, even when comparison was restricted to therapeutic studies, and that, even after 1980, 25% of published randomised studies were of low methodological quality.³ In the event that these observations are applicable to the rest of medical practice, this would be indicative of a fundamental flaw in most clinical guidelines, especially those which do not specify the level of evidence on which they are based.

Finally, there is the vast uncharted territory of what could be legitimately defined as evidence based ignorance, which has come into being because a lot of clinical research has restricted its focus to problems which are amenable to "quick fix" solutions. The unfortunate consequence is a "trade off", which amounts to the acquisition of precise answers to the wrong questions instead of approximate answers to the right questions.

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