LETTERS TO
THE EDITOR

General Medical Council registration

EDITOR,—In August last year, we experienced difficulties in obtaining General Medical Council (GMC) registration for several doctors in their first senior house officer post at the Lister Hospital, Stevenage.

In month of the job and emergency (A&E) department this meant that the doctors involved could not treat and discharge patients. The confirmed registrations were available late on the second day of the doctors’ jobs. No locums were required. However obtaining confirmation involved considerable time and caused unnecessary stress to the doctors, departments, and medical staffing officers concerned.

To assess the extent of the problem a sample of hospitals’ medical staffing departments in North Thames was contacted by telephone. A standard set of questions was asked regarding problems with GMC registrations, any locums required, and the much medical staffing time was spent in dealing with the situation.

Replies were received from 23 hospitals. Over half of them had telephoned the GMC on 4 August to confirm registrations. In our sample were hospitals who were up to 10 days behind in ensuring that medical work in A&E and lack of 24 hour senior supervision in many departments, which means that senior house officers without full GMC registration are in practice unable to make any useful contribution to the care of patients. We would be interested to know if colleagues have had similar difficulties and would welcome suggestions for dealing with this problem.

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Carbon monoxide poisoning and hyperbaric oxygen

EDITOR,—Unfortunately, by restricting their analyses to hydrogen ion concentration alone, the authors of this paper may not have demonstrated that metabolic acidosis occurred in the patients they describe.1 Raised hydrogen ion concentration is indicative of acidemia. Without data on other acid-base variables the aetiology must remain elusive. Repeating the study looking at actual or standard base deficit may well solve this problem. Unconsciousness leading to airway compromise is one example of how the acidemia described could be respiratory in origin.

I agree with the authors that the most significant effect of carbon monoxide poisoning is the development of severe hypoxia and hence intracellular acidosis. It is, however, somewhat simplistic to assume that plasma hydrogen ion concentration alone correlates with the degree of cellular carbon monoxide damage. The buffering capacity of the blood can vary widely under a variety of clinical conditions.

These patients also have other possible aetiologies for acid-base derangement. Salicylate poisoning is well described as evolving both metabolic acidosis and respiratory alkalosis. This does not appear to be corrected for in the analysis. Other poisons common to suicide attempts such as ethyl alcohol may also provoke metabolic acidosis. The authors make no comment on other possible confounding causes of acid-base abnormality.

I agree with the authors that measurement of carboxyhaemoglobin (COHb) concentration is well described as evolving both metabolic acidosis and respiratory alkalos. A series of intensive care patients treated for carbon monoxide poisoning at the same hospital in 1996/7 actually demonstrated a negative association between initial COHb concentration and survival. Those who died or suffered permanent neurological impairment had an initial mean COHb of 31.45% and patients who fully recovered had an initial mean COHb of 46.48%.

This paper is useful as it draws attention to the falsehood that the initial COHb is helpful in determining the severity of carbon monoxide poisoning. A reliable measure of intracellular acidosis is required. Presently, probably the best that can be done is to use semiquantitative blood gas measures of metabolic acidosis (of which hydrogen ion concentration alone is not one) and possibly serum lactate estimation. Neither of these is specific to carbon monoxide poisoning. Severe derangement of either or both of the above will, at least, identify the most unwell.

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2 Thomas R. Hyperbaric treatment of severe carbon monoxide poisoning in intensive care patients: the first eight months experience from the new hyperbaric hospital at Royal Hospital Haslar. Presented at Tri-service Anaesthetic Meeting, Royal College of Anaesthetists, November 1997.

The authors reply

The potential for respiratory acidosis and the influence of salicylates have been addressed in paragraph 3 of our results.1 We reported that there was no significant difference between the mean partial pressures of carbon dioxide in the two groups, confirming that any differences were due to metabolic, rather than respiratory, acidosis. In addition we reported the salicylate levels in the multiple treatment group. The hydrogen ion activity is, by definition, the cardinal feature of acidosis. The actual and standard base deficits are figures derived from the hydrogen ion concentration and partial pressure of carbon dioxide. We have therefore reported the primary measurements rather than derived figures.

Direct measures of intracellular acidosis would indeed be very interesting but are far beyond being clinically feasible in the accident and emergency department. Hydrogen ion activity in blood is a universally and rapidly available investigation, indicative of a patient’s carbon acid-base status.

The data Dr Thomas mentions are potentially interesting and clearly support our contention that COHb is an unreliable measure of severity of poisoning. The observation in his series that the patients who died or suffered permanent neurological impairment had a lower mean initial COHb than those who recovered could be explained by a delay in making initial COHb measurements. The type of exposure may also have differed; a long exposure to a low concentration of carbon monoxide would lead to a lower COHb but a higher tissue burden, than a short exposure to a higher concentration. As reported in our review article Dr Martin Hamilton-Farrell has observed a correlation of external clinical data (including neurological abnormality in those patients suffering long duration or multiple exposures.1 The numbers of patients studied and a statistical analysis to ascertain the significance of the difference in the mean COHb levels is clearly fundamental. The methodology by which patients were assessed after treatment is also important as subtle neurological abnormality may be

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