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 the accident 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 how 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, no locums were required but a considerable amount of time was spent by medical staffing departments and the doctors themselves in obtaining this confirmation. If representative this has major manpower implications nationwide. The factors causing delays were: (i) incorrect doctors' addresses; (ii) cheques not clearing; and (iii) GMC workload (as one medical staffing worker stated "They said they were having a hectic day and their computer had crashed").

Those hospitals affected arranged for doctors without appropriate registration to either "shadow" other doctors in their departments until confirmation was received or attend induction courses.

We should like to raise the following points:

- Why are certificates of satisfactory completion of house jobs only obtainable in the last month of the job?
- Do these certificates need to go back to the medical school before going to the GMC? (Provisional registration implies having completed a medical school degree!)
- Could GMC registrations be phased throughout the year to avoid the rush for the August deadline? A house officer having satisfactorily completed a first post and the first two to three months of the second, could reasonably "set the wheels in motion" for full registration, pending completion of the second post.

As the GMC has approximately 4000 applications for provisional and 4000 for full registration every year[1] some delays are inevitable. However, as the volume of the workload is predictable, there must be a way to ease the pressure on the GMC, medical staffing departments, and junior doctors in the month of August each year.

Recent correspondence with the head of Operations of the Registration Directorate at the GMC informs us that a review of the service provided last summer is being conducted, and improvements will be implemented where possible.

This is a problem which commonly affects A&E departments because of the high turnover of junior staff and the fact that it is, for many doctors, their first senior house officer post. This is compounded by the nature of the 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 patient care.

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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1 General Medical Council. *General Medical Council annual review.* London: GMC, 1998: 13–16.

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 acidaemia. 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 acidaemia described could be respiratory in origin.

I agree with the authors that the most significant effect of carbon monoxide poisoning is the development of a cytotoxic hypoxia and hence intracellular acidosis. It is, however, somewhat simplistic to assume that plasma hydrogen ion concentration alone correlates with the degree of intracellular 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 evoking 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 also may 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 useful only as an indication that carbon monoxide poisoning has occurred. 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 outcome[2]: patients who died or suffered permanent neurological impairment had a mean initial COHb of 31.45% and patients who fully recovered had a mean initial 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 recognised 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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1 Turner M, Esaw M, Clark R. Carbon monoxide poisoning treated with hyperbaric oxygen: metabolic acidosis as a predictor of treatment requirements. *J Accid Emerg Med* 1999;16:96–8.
2 Thomas R. Hyperbaric oxygen treatment of severe carbon monoxide poisoning in intensive care patients: the first eight months experience of the new hyperbaric facility 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 from being clinically available in the accident and emergency department. Hydrogen ion activity in blood is a universally and rapidly available investigation, indicative of a patient's overall 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 measurement or differences in first aid treatment (for example 100% oxygen) before COHb measurement. The types 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 higher incidence of residual neurological abnormality in those patients suffering long duration or multiple exposures.[2] 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