MANAGEMENT OF CARBOXYHæMOGLOBINÆMIA

Sir,—Drs Vegfors and Lennmarken have demonstrated usefully how easy it is to be misled in the management of carbon monoxide poisoning [1]. Their case deserves further comment.

Unfortunately, there is little information on the mechanism of cellular toxicity of carbon monoxide. Carbon monoxide binds to haem-containing enzymes such as cytochrome P450 [2]. Measurements of carboxyhaemoglobin concentration are not helpful in determining the degree of tissue poisoning [3]. In particular, the likelihood of late neuropsychiatric problems, such as memory disturbance, is difficult to predict [4]. Up to 43% of survivors may have such sequelae if followed up for 3 years [5].

Vegfors and Lennmarken suggested that an elimination half-life of 2 h was sufficient. As they point out, the use of 100% oxygen at ambient pressure and at 3 atm abs would have reduced this to 1 h and to 20 min, respectively. Intracellular concentrations of carbon monoxide cannot be measured at present, but it is assumed that oxygen therapy similarly increases the elimination from the tissues and so reduces tissue damage.

Hyperbaric oxygen appears to reduce both acute symptoms and neuropsychiatric complications [2]; however, its exact role has yet to be determined. Members of the British Isles Group of Hyperbaric Therapists are planning a prospective controlled study which will look at this issue—with particular regard to long-term morbidity. In the meantime, every case of suspected carbon monoxide poisoning should be treated with 100% oxygen.

Patients with evidence of severe poisoning should be referred for hyperbaric oxygen [6]. This Hyperbaric Unit will consider a patient who has any one of the following: loss of consciousness at any stage since exposure to carbon monoxide; neuropsychological symptoms other than a mild headache; cardiac complications, including ischaemia and arrhythmias; carboxyhaemoglobin concentration greater than 20% at any time; pregnancy.

This Hyperbaric Unit may be contacted via the Duty Consultant, Intensive Therapy Unit, Whips Cross Hospital (Tel.: 081 539 5522). Other Hyperbaric Units may be contacted via the British Isles Group of Hyperbaric Therapists, Diving Diseases Research Centre, Fort Bovisand, Plymouth (Tel.: 0752 408093).

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REFERENCES


Sir,—Our claim that HbCO reads as HbO is referred to directly (reference [3] in our paper). We agree that the absorbance of HbCO at 940 nm is less than 10% of that of HbO. Nevertheless, such a value will be much smaller than that when HbO is replaced with reduced haemoglobin. As the concentration of HbCO increases, the SpO2 reads a percentage higher than the actual fraction of haemoglobin present as HbO for a given arterial partial pressure of oxygen.

Vegfors and Lennmarken stress very well that clinical evidence should guide the diagnosis of carbon monoxide poisoning, and oximetry should be used for laboratory confirmation.

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REFERENCE


Sir,—We are grateful for the interest expressed by Drs Nathanson and Hamilton-Farrell and we agree with the therapeutic points they raise. The intention of our case report was to stress and illustrate the diagnostic problems with COHb poisoning. Their points, although interesting, were beyond the aim of our case report.

Our claim that HbCO reads as HbO is referred to directly (reference [3] in our paper). We agree that the absorbance of HbCO at 940 nm is less than 10% of that of HbO. This leads to a smaller quotient which will, in the algorithm, cause an overestimation of SpO2. Therefore, it is too simple an explanation and, in fact, a misconception that HbCO reads as HbO.

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EFFECT OF PRETREATMENT WITH ORAL PYRIDOSTIGMINE

Sir,—We read with interest the article by Turner, Williams and Baker [1]. We were involved in treating service personnel injured in the Gulf conflict, and studied the response to vecuronium in those who had taken the Nerve Agent Pretreatment (NAPS) regimen—pyridostigmine 30 mg 8-hourly until medical evacuation from the Gulf 2-3 days before surgery.

Patients were anaesthetized using our usual technique comprising fenytanil, propofol, nitrous oxide and isoflurane in oxygen with vecuronium as the neuromuscular blocking agent. Vecuronium was chosen as it is now considered to be the routine agent in British Army anaesthetic practice [2]. Other admissions during this time for similar minor surgical (mainly orthopaedic) procedures were studied as controls. A Brain laryngeal mask airway was inserted and spontaneous ventilation was continued until baseline readings of the Datex relaxograph were obtained using ulnar nerve and adductor pollicis muscle electrode placement. Vecuronium approximately 0.1 mg kg⁻¹ was then administered.

As in the study by Turner, Williams and Baker, there was no consistent change in the rate of onset, degree or duration of neuromuscular block. However, the current need to achieve the