SHORT REPORTS

Delayed hyperbaric oxygen treatment for acute carbon monoxide poisoning

Hyperbaric oxygen is the preferred treatment in acute carbon monoxide poisoning. Though it should be started as soon as possible, we wish to emphasise that anyone who has been exposed to carbon monoxide should be treated regardless of the delay between exposure and presentation.[1] We present our experience with delayed hyperbaric oxygen treatment for acute carbon monoxide poisoning.

Patients and results

From January 1982 to February 1983 we treated 10 cases of acute carbon monoxide poisoning. In six cases there was a delay of seven to 16 hours from the time of rescue to the beginning of treatment with hyperbaric oxygen. The duration of exposure to the environment rich in carbon monoxide varied from a few minutes (three patients caught in fires) to 14 hours. Four of the six inhaled smoke. The table gives clinical details on the six patients. They were at different levels of consciousness when rescued. Electrocardiography showed ischaemic changes in two cases, and all had metabolic acidosis. Five made full mental and physical recoveries. One (case 2) did not improve: he developed hydrocephalus with increased intracranial pressure and finally died.

Comment

Carboxyhaemoglobin concentrations correlate only roughly with clinical condition. Goldbaum *et al* explained this in their study of three groups of dogs.[2] In the study one group of dogs was given 13% carbon monoxide by inhalation. All died with carboxyhaemoglobin concentrations of 54-90%. A second group of dogs were bled until their haemoglobin concentration had been reduced by 68%: they were then reinfused with Ringer's solution and dextran and all survived. A third group of dogs were also bled until their haemoglobin concentration had been reduced by 68%, but they then received transfusions of carboxyhaemoglobin red cells. These dogs had final carboxyhaemoglobin concentrations of 57-64% and all survived. These results are explained by the exclusion of oxygen from the cytochrome oxydase a_3 enzyme by carbon monoxide. Carbon monoxide can reach the blood cells only when it is dissolved in the blood, which happens when it is breathed (as shown in the first group of dogs). The third group of dogs had no dissolved carbon monoxide in their blood, and their cytochrome oxydase a_3 activity was normal.

The carboxyhaemoglobin concentration immediately after exposure can be calculated roughly from the interval between exposure to carbon monoxide and the time of blood sampling, taking into account the half life of carboxyhaemoglobin,[3] which is 320 minutes in room air and 80 minutes when 100% oxygen is being breathed. A long delay between rescue and blood sampling results in a poor correlation between carboxyhaemoglobin concentration and clinical signs. We measured carboxyhaemoglobin concentrations in only two of our patients (cases 4 and 6), and these were 1% and 0·6% respectively. The late neurological sequelae of acute carbon monoxide poisoning are almost certainly due to oedema and damage to the blood-brain barrier caused by hypoxia. This damage can be shown by computed tomography and confirmed at necropsy.[4] Hyperbaric oxygen reduces the incidence of these phenomena, which is another reason for using this form of treatment regardless of whether there has been considerable delay.

Finally, we wish to draw attention to cases 4, 5, and 6, in which the patients suffered lengthy exposure to the toxic environment and treatment was considerably delayed. Although prolonged exposure to carbon monoxide is known to carry much greater risks than short exposure,[5] these three patients responded well to hyperbaric oxygen treatment and left the hospital free of complaints.

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2 Goldbaum RL, Ramirez RG, Karel BA. What is the mechanism of carbon monoxide toxicity? *Aviat Space Environ Med* 1975;**46**:1289-91.
3 Clark CJ, Campbell D, Reid WH. Blood carboxyhemoglobin and cyanide levels in fire survivors. *Lancet* 1981;i:1332-5.
4 Sawada Y, Sakamoto T, Nishide K, *et al*. Correlation of pathological findings with computed tomographic findings after acute carbon monoxide poisoning. *N Engl J Med* 1983;**308**:1296.
5 Kindwall EP. Carbon monoxide and cyanide poisoning. In: Davis JC, Hunt TK, eds. *Hyperbaric oxygen therapy*. Bethesda, Maryland: Undersea Medical Society, 1977:177-90.

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Israeli Naval Hyperbaric Institute, PO Box 8040, Haifa 31080, Israel
AVISHAI ZISER, MD, assistant director
AVI SHUPAK, MD, physician
PINHAS HALPERN, MD, physician
DAVID GOZAL, MD, physician
YEHUDA MELAMED, MD, director

Details of six patients on admission

Case No	Age (years)	Duration of exposure to carbon monoxide	Interval between rescue and start of treatment (hours)	Level of consciousness	Clinical signs	Blood gas tensions: Oxygen (kPa)	Blood gas tensions: Carbon dioxide (kPa)	Blood gas tensions: pH
1	2½	Few minutes	8	Stupor	Rales in both lung fields	6·9	3·1	7·32
2	3	Few minutes	7	Deep coma	Convulsions	50·1	5·1	7·37
3	5	Few minutes	7	Coma	Pulmonary oedema	9·2	6·1	7·29
4	20	8 hours	12	Fully conscious	ST segment elevation in leads V1, V2	13·0	4·3	7·35
5	44	14 hours	12	Deep coma		13·2	4·0	7·20
6	70	Overnight	16	Stupor	ST segment depression in most leads	14·9	5·2	7·39

Conversion: SI to traditional units—Oxygen and carbon dioxide tensions: 1 kPa ≈ 7·5 mm Hg.

BMX bike injuries: the latest epidemic

BMX biking is a major new craze throughout the Western world as much as skateboarding was a decade ago. To date there have been only two published reports of BMX bike injuries, both from Australia.[1 2] During one week in May 1984 our hospital admitted five children with serious injuries sustained after falling off their BMX bikes. None of these injuries were fatal but they included major trauma to the liver and spleen, a fractured skull, a partially avulsed ear, a serious soft tissue injury to the abdominal wall, and a fracture dislocation at the wrist. Subsequently we conducted a survey to measure the problem in Southampton and to see whether any safety routines are being followed.

Methods and results

During a five week period from 1 June to 7 July 1984 all patients with BMX bike injuries presenting to the hospital were registered on a protocol. Details of the patient, the mechanism of injury, and any safety equipment used were recorded. Twenty three boys were registered during this period. The mean age was 11·7 (range 2·5-15·5) and most were over 10. No girls were seen with BMX bike injuries. Ten different models of BMX bike were used by these 23 children and one bike was homemade. The injuries sustained were as follows: to the arms (12) and legs (five): contusions (six); fractures (three: distal radius, scaphoid, and metatarsal); lacerations (three); abrasions (three); and sprains (two); to the abdomen (one); and to the head and neck: lacerations of chin, face, and scalp (three) and broken teeth (two). (All lacerations needed suturing.) Only one patient was admitted to hospital; he had a subcapsular haematoma of his spleen diagnosed by ultrasound. Managed conservatively, he did not require transfusion or surgery.

Thirteen of the 23 boys had sustained their injury during what they termed