Algorithm for managing injury from smoke inhalation

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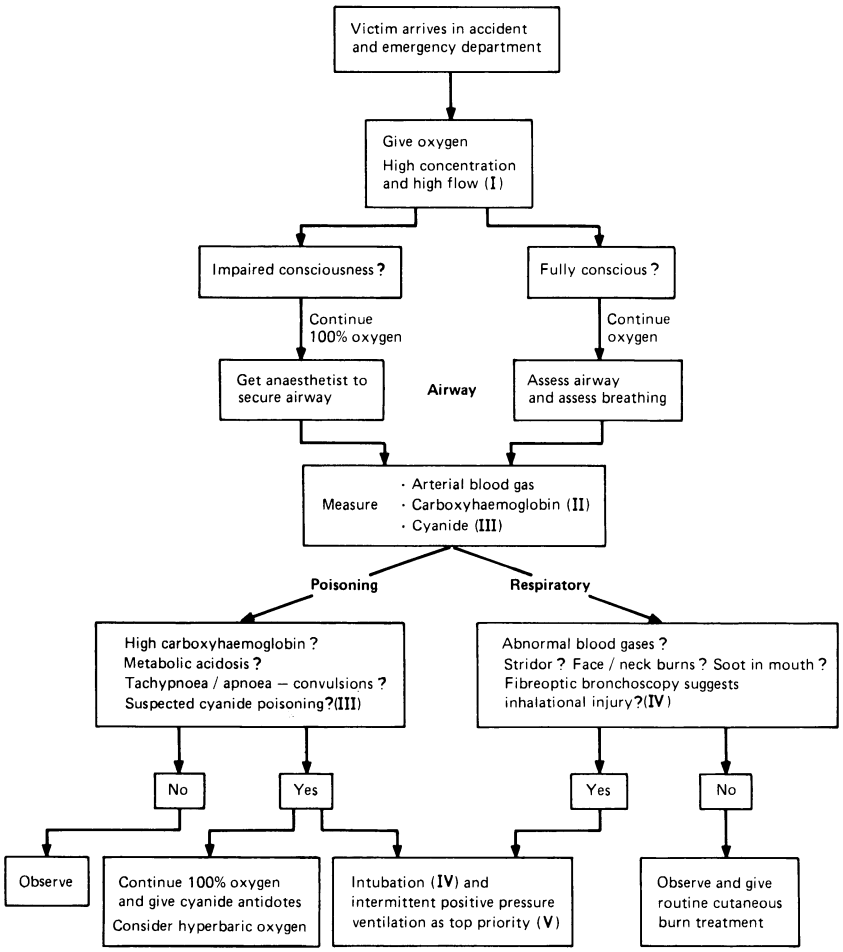
Thermal injury accounts for 14 000 casualties a year in the United Kingdom. With improvements in managing cutaneous burns mortality has steadily reduced so that inhalational injury from smoke and hot, toxic gases is increasingly important in outcome.[1,2] When overlooked, smoke inhalation can lead to early or late deterioration in the patient's condition due to damage to the airways or lungs. In addition, poisoning may occur by inhalation of gases such as hydrogen cyanide or carbon monoxide. These problems were experienced in the fire at King's Cross in 1987, in which a fifth of the victims were found at postmortem examination to have high concentrations of carboxyhaemoglobin. Furthermore, despite controversy over the forensic method used there was evidence that cyanide poisoning may have had a contributory role in some of the deaths. In this unusual fire, however, the heat blast led rapidly to asphyxiation, thus reducing the duration of exposure to toxic inhalation.

This disaster, supported by previous experience with inhalational burns in this unit, suggested the need for a protocol to guide clinicians in the early management of these patients. We have proposed an immediate care plan in the form of an algorithm. This aims to promote early recognition and treatment of the airway problems and the poisoning that result from hot, toxic fumes. The flow chart (figure) was designed to give direction to the admitting clinicians and paramedical staff. Points needing explanation are labelled on the chart (I to V) and elaborated in the text.



Algorithm of early management of inhalational injury in fire victim. (Roman numerals indicate relevant accompanying text)

Oxygen administration and measurement (I)

In the initial assessment of a burnt patient evidence of underlying hypoxaemia and poisoning may be hidden by smoke stains or burns. Confused or aggressive behaviour may be due to cerebral anoxia and should not be attributed only to pain. To avoid the immediate and long term cerebral damage caused by hypoxia, upper airway obstruction, and carbon monoxide poisoning[3] early administration of high concentrations of inspired oxygen is essential. Most of the commercially available plastic masks (MC, Hudson, etc) achieve variable and unpredictable oxygen concentrations that depend on the patient's respiratory rate, tidal volume, and rate of inspiratory airflow. The best choice is one of the high airflow masks with oxygen enrichment, such as the Venti-mask,[4] which can achieve 60% inspired concentration. When 100% oxygen is necessary a tight fitting oronasal mask connected to a breathing system with an expiratory valve and reservoir bag should be used.

Alternatively, the patient can be intubated and ventilated with 100% oxygen. A non-rebreathing system should be used in cases of suspected carbon monoxide or cyanide poisoning to avoid reabsorption. Measuring and interpreting the blood concentration of oxygen in these patients is difficult. A conventional blood gas analyser with electrodes measures the tension of oxygen dissolved in the plasma. In healthy patients the oxygen tension determines the percentage of haemoglobin that binds oxygen to form oxyhaemoglobin. Such analysers calculate this functional oxygen saturation on the assumption that only normal haemoglobin (capable of binding oxygen) is present. Blood from a fire victim, however, may well contain high carboxyhaemoglobin concentrations with a dangerously low concentration of oxyhaemoglobin (despite high measured oxygen tension and high derived calculated saturation). In these circumstances only a blood oximeter that uses spectrophotometry actually measures the concentrations of oxyhaemoglobin, reduced haemoglobin, carboxyhaemoglobin, and methaemoglobin in the sample.

The oxyhaemoglobin can then be used to calculate the oxygen content of the blood, which may be considerably lower than the calculated oxygen saturation would suggest. In this way the blood oximeter will confirm a presumptive diagnosis of carbon monoxide poisoning and show any deficiency in oxygen delivery to the tissues. Given their current popularity, it should be noted that pulse oximeters are unreliable in the presence of carboxyhaemoglobin and methaemoglobin[5] as they too cannot measure each type of haemoglobin independently.

Carbon monoxide poisoning (II)

Carbon monoxide combines strongly with haemoglobin, causing impairment of oxygen transport. In addition, there is evidence of direct tissue toxicity

caused by unbound carbon monoxide dissolved in the plasma.[6] Several workers consider that it is this tissue toxicity that produces the symptoms and the cerebral damage and suggest that carboxyhaemoglobin concentrations correlate poorly with clinical condition and prognosis.[7] Others point out a good association between carboxyhaemoglobin concentrations and clinical state and describe concentrations of >40% causing coma and >60% collapse. Carboxyhaemoglobin may also be measured as a diagnostic test: concentrations over 15% on admission suggest smoke inhalation. Back calculation with a nomogram has been recommended as a means of estimating concentrations at the time of exposure.[8]

Carbon monoxide poisoning presents with symptoms and signs ranging from nausea and headache to coma and convulsions. In a burnt patient any alteration in the neuropsychiatric state or consciousness should be viewed with suspicion. Laboratory bench tests or blood oximeters measure carboxyhaemoglobin by spectrophotometry. Carbon monoxide meters (such as the Bedfont Micro Smokerlyzer) are available to measure exhaled carbon monoxide. Metabolic acidosis may be distinct.

The immediate treatment of carbon monoxide poisoning is with high concentrations of oxygen. Several workers recommend giving hyperbaric oxygen,[9,10] which increases the amount of oxygen dissolved in plasma, thereby improving oxygen delivery to the tissues. This also combats the directly toxic effects of carbon monoxide in tissues. Hyperbaric oxygen is especially recommended in (*a*) patients who are or have been unconscious; (*b*) patients with neurological symptoms; (*c*) patients with cardiac complications; (*d*) patients with carboxyhaemoglobin concentrations >40%; and (*e*) pregnant women. Comatose patients should be transferred early, but late referral for hyperbaric oxygen may be worth while. For the nearest hyperbaric chamber telephone the Royal Navy, Portsmouth (0705) 822351 ext 24875/41769/22008.

Cyanide poisoning (III)

Fumes from smouldering plastics such as polyurethane, nylon, and Acrilan contain hydrogen cyanide gas.[11] Inhaling these fumes results in rapid systemic absorption and cyanide poisoning. Cyanide paralyses mitochondrial respiration by binding reversibly with enzymes containing ferric ions. In particular, cytochrome oxidase is affected and oxidative phosphorylation stops.

Clinical signs of cyanide poisoning are produced by cellular hypoxia and therefore affect several systems. Commonly, abnormalities of the central nervous system occur with headaches, dizziness, and fits. In the early stages the hypoxia causes stimulation of the respiratory centre with tachypnoea and dyspnoea later followed by bradypnoea and apnoea. Cardiovascular signs include hypertension and tachycardia leading eventually to hypotension and cardiovascular collapse.

Diagnosis—Unlike with industrial exposure or attempted suicide by ingestion the evidence for cyanide poisoning in a fire is circumstantial. Measuring blood cyanide concentrations is difficult as cyanide disappears rapidly from stored blood specimens. Furthermore, the actual process of measurement takes several hours and is usually not available locally. Nevertheless, a sample of heparinised whole blood should be sent immediately to the regional poisons unit labelled with the time of sampling and the time since the poisoning occurred. Concentrations of ≤13 μmol/l are commonly seen in smokers. Concentrations of >50 μmol/l are associated with altered consciousness, and concentrations of >100 μmol/l are lethal.[12] Because of the difficulty in measuring cyanide, thiocyanate concentrations are considered useful by some[13] but not all workers.[14] Burns units may find a simple hydrogen cyanide gas detector (such as the Drager tube) useful as an early diagnostic aid. Findings supporting cyanide poisoning are a normal arterial oxygen tension, a decrease in measured (as opposed to calculated) arterial oxygen saturation due to cyanhaemoglobin, an increased mixed venous oxygen saturation (>75%), and a decrease in the arteriovenous difference in oxygen content (normally 5 ml/100 ml). Additionally, a severe metabolic acidosis, high lactate concentration, and increased anion gap (>12 μmol) may suggest cellular hypoxia. The usefulness of high carboxyhaemoglobin concentration as a marker of cyanide inhalation is controversial.[15]

Treatment of cyanide poisoning (III)

Unlike cyanide poisoning by ingestion systemic absorption of further cyanide stops on removal of the fire victim from the scene. At this stage the cyanide is already present in the blood and tissues. Emergency treatment starts with transfer to hospital and giving oxygen. The urgent objectives are to reduce blood cyanide concentrations and promote detachment of cyanide from cytochrome oxidase in the tissues. There are three principal methods.

Conversion of haemoglobin to methaemoglobin by the antidote promotes the binding of cyanide to the three ferric haem groups in each methaemoglobin molecule. The percentage of methaemoglobin achieved plus the percentage of carboxyhaemoglobin should be measured and not allowed to exceed 40%, thus avoiding excessive reduction of oxygen carrying capacity. Therefore, in the case of fire victims caution should be exercised until the concentration of carboxyhaemoglobin is known. Antidotes used to produce methaemoglobin are amyl nitrite, sodium nitrite, and 4-dimethylaminophenol. It should be noted that sodium nitrite can induce distinct vasodilatation and hypotension.

Augmentation of the endogenous cyanide detoxification system—Normally cyanide is converted slowly to the essentially non-toxic thiocyanate in the bloodstream and by enzymes such as rhodanase in the mitochondria. Giving sodium thiosulphate and thus providing an extra source of sulphur will accelerate this process.

Chelation of the cyanide—Dicobalt edetate combines with cyanide to form an inert complex, cobalticyanide. A potentially serious consequence of its use, however, is cobalt toxicity. This is more likely in the absence of cyanide. Although dicobalt edetate has been the agent of choice in the United Kingdom, it should be used only in cases of severe poisoning, whether suspected or proved. When the patient is still conscious some workers propose that the less toxic antidotes should be used.[16]

The protocol for the treatment of cyanide poisoning has recently been the subject of a joint study in Geneva, under the auspices of the World Health Organisation, by the International Programme on Chemical Safety and the Commission of the European Communities.[17] The following section summarises the recommendations of their document *IPCS/CEC Evaluation of Antidotes in Poisoning by Cyanide*.

FIRST AID MEASURES

These measures are to be undertaken only in cases of unequivocally severe poisonings.

Trained staff (wearing protective clothing and breathing apparatus if hydrogen cyanide or liquid cyanide preparations are involved should

- stop further exposure

- start artificial ventilation with 100% oxygen by a non-rebreathing system
- give 0·2-0·4 ml amyl nitrite through an Ambu bag.

If a doctor is present immediately on the scene he or she should

- start artificial ventilation with 100% oxygen by a non-rebreathing system
- give 0·2-0·4 ml amyl nitrite through an Ambu bag.

HOSPITAL TREATMENT

Hospital doctors must establish whether specific antidotal treatment was given at the time of the incident before further doses are given, especially in the case of agents that form methaemoglobin.

In cases of severe poisoning when the patient is in deep coma with dilated, non-reactive pupils and deteriorating cardiorespiratory function (blood cyanide concentrations 115-154 μmol/l (3-4 mg/l))

- start artificial ventilation with 100% oxygen
- start cardiorespiratory support

Then give

- either 10 ml of 3% sodium nitrite solution (300 mg) intravenously over 5-20 minutes
or 5 ml 5% dimethylaminophenol solution (250 mg or 3-4 mg/kg) intravenously over one minute
or 20 ml 1·5% dicobalt edetate solution (300 mg) intravenously over one minute
or 10 ml 40% hydroxocobalamin solution (4 g) intravenously over 20 minutes
- and 50 ml 25% sodium thiosulphate solution (12·5 g) intravenously over 10 minutes.

In cases of moderately severe poisoning when the patients have suffered a short period of unconsciousness, convulsions, vomiting, or cyanosis (blood cyanide concentrations 77-115 μmol/l (2-3 mg/l))

- give 100% oxygen but for no longer than 12-24 hours
- observe in intensive care
- give 50 ml 25% sodium thiosulphate solution (12·5 g) intravenously over 10 minutes.

In cases of mild poisoning when patients have nausea, dizziness, and drowsiness (blood cyanide concentrations <77 μmol/l (<2 mg/l))

- give oxygen
- reassurance
- prescribe bed rest.

The above scheme was developed to deal with all modes of cyanide poisoning. In smoke inhalation, however, there may be doubt initially about the presence of cyanide poisoning. In these circumstances, therefore, we recommend using the fairly safe antidotes amyl nitrite and sodium thiosulphate. Thereafter, only when concentrations of carboxyhaemoglobin and methaemoglobin are known and the patient's clinical progress has been reviewed should the other agents be used.

Severely poisoned patients may on occasion fail to respond to the initial dose of a specific antidote. Although repeated doses of hydroxocobalamin or sodium thiosulphate, or both, are unlikely to be associated with toxicity, expert advice should be sought before a repeated dose of any other specific antidote is given. Intensive supportive treatment is of paramount importance in these circumstances. Finally, the above scheme raises the question of whether amyl nitrite should be made available to ambulance crews and paramedical staff at the scene of the accident.

Indication for intubation (IV)

When airway obstruction is suspected indirect laryngoscopy or fibreoptic laryngoscopy or bronchoscopy is an invaluable investigation in the hands of experts. Indications for intubation[20] are (*a*) unconsciousness or failure to maintain an airway; (*b*) evidence of severe carbon monoxide or cyanide poisoning; (*c*) stridor; (*d*) facial burns and depression of the central nervous system; (*e*) facial burns and circumferential burns of the neck; (*f*) full thickness burns of the nose or lips; and (*g*) visible oedema of the pharynx and larynx. Nasotracheal tubes are more comfortable and require less sedation. This will allow spontaneous respiration with continuous positive airway pressure (or intermittent positive pressure ventilation as appropriate).

Respiratory failure (V)

The onset of respiratory failure may be recognised clinically with increasing respiratory rates and dyspnoea. Criteria for respiratory support are (*a*) respiration rate >30/min; (*b*) vital capacity <12-15 ml/kg; (*c*) arterial oxygen tension <11·0 kPa when breathing 40% oxygen; (*d*) arterial oxygen tension <6·0 kPa when breathing air; and (*e*) arterial carbon dioxide tension high enough to cause fall in pH to 7·2.

When severe hypoxaemia occurs with good ventilatory response (arterial carbon dioxide <6·0 kPa) continuous positive airway pressure by mask can be effective.[17] Otherwise intermittent positive pressure ventilation through a nasotracheal tube will allow satisfactory treatment of respiratory failure with easy sedation and management of the patient. Early venous access for fluids and monitoring of central venous pressure (under scrupulous asepsis) is recommended before dressings, ointments, and oedema render the task too difficult. The place of tracheostomy is controversial, but modern attitudes are less critical[18] and it may lead to less long term laryngeal scarring.[19]

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The algorithm is available in poster form from the authors.

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Lesson of the Week

Communicating with Asian patients

Kathryn A Stevens, R F Fletcher

Patients from the Asian subcontinent who do not speak or read English need the help of a qualified interpreter or written information or videos in their own language

Patients cannot hope to benefit fully from the advice given in a consultation or be expected to comply with treatment if they cannot understand the information that is given to them. Many of our patients are of Asian ethnic origin, and some have obvious difficulties with written and spoken communication. Though a previous report recommended providing teaching materials in Asian languages,[1] there is doubt about the usefulness of such written material because of uncertainty about the level of literacy among patients. To help us develop suitable material in the dietetic department we carried out a survey of our patients to assess the possible value of different ways of communicating with them.

Patients, methods, and results

Patients of Asian ethnic origin from the Indian subcontinent who attended the general outpatients department at this hospital over seven days in May 1988 were invited to answer a short questionnaire. Only one patient refused, and 150 patients were interviewed by an Asian interpreter. Relatives were not asked to interpret.

The patient's ability to read English and an Asian language was assessed by asking the patient to read aloud a short, simple sentence in the appropriate language. Each sentence was different to reduce the possibility of the patient guessing the content.

The patients' ages ranged from 13 to 74; 96 were women. Eighty five could read an Asian language: Punjabi (43), Urdu (22), Hindi (10), Bengali (5), and Gujarati (5).

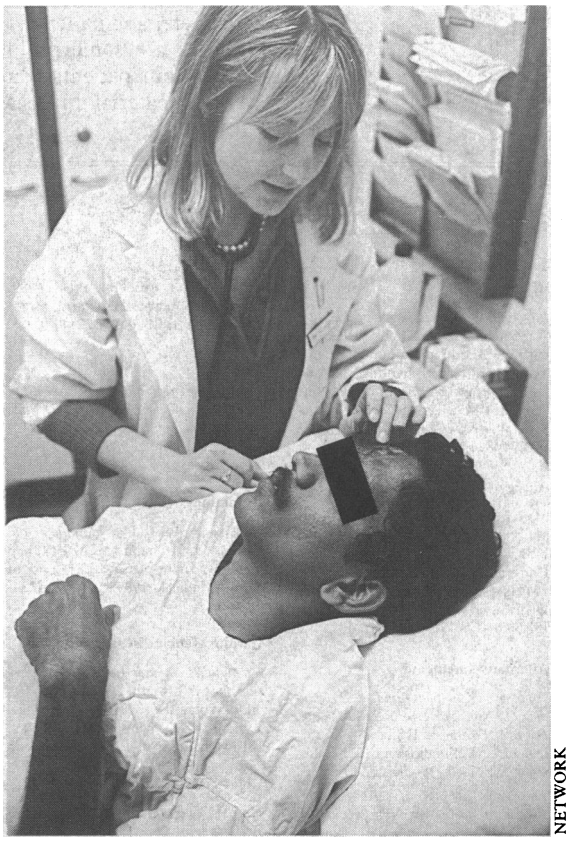
A total of 76 of the 150 patients could speak English, and 53 could read it as well (table I). All of the men and two thirds of the women aged 13 to 28 could speak English but the proportion fell to under a fifth of the men and one quarter of the women aged 61 to 74 (table II). Fifteen patients said that they could read, but they were unable to do so when tested.

TABLE I—*Literacy among 150 patients of Asian origin seen in outpatient clinics*

Literacy	No of patients
Read English only	20
Read English and an Asian language	33
Read an Asian language only	52
Read no language	45

TABLE II—*Numbers of men and women of Asian origin who could speak English according to age*

Age (years)	Men		Women	
	No	No speaking English	No	No speaking English
13-28	11	11	32	21
29-60	31	22	56	18
61-74	12	2	8	2



NETWORK

A doctor examines a patient in the casualty department after a road accident

Discussion

Our patients fell into three groups of roughly equal sizes. One third read English, and often an Asian language as well, one third read an Asian language only, and one third could not read. Among those speaking Asian languages, about half spoke Punjabi, but four other languages were also spoken. Thus the needs of our patients for help in understanding information about their health care are highly variable. One hundred and twenty five of our patients wanted

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