MEDICAL PRACTICE

Contemporary Themes

Manchester air disaster

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Abstract

On 22 August 1985 a fire occurred on a Boeing 737 jet airliner at take off at Manchester Airport. One hundred and thirty seven passengers and crew were on board. Fifty two passengers died on the aircraft, 85 escaped. Most survivors had minor physical injuries, but 15 required admission to hospital because of smoke inhalation and two of these had severe burns. At presentation only one survivor required ventilation but within 12 hours a further five required ventilation. Although initially patients suffering from smoke inhalation may seem relatively well, lung function may deteriorate rapidly in the first 24 hours.

Careful organisation and regular practice of procedures to deal with a major accident are essential to be able to respond adequately to such an event.

Introduction

At 7.15 am on 22 August 1985 the port engine of a Boeing 737 jet airliner exploded as the aeroplane prepared for take off at Manchester Airport. Fire spread rapidly to the rear half of the body of the aircraft, exposing the 137 people on board to the intense blaze and thick smoke. Fifty two people died; the remainder got out through the forward escape hatches; those who were last to leave were exposed to thick smoke for several minutes.

At 7.25 am this hospital knew of the fire at Manchester Airport and at 7.36 am was informed by the ambulance service that 40

casualties were in transit. The senior nursing officer on duty delayed the departure of the night shift to provide extra personnel, and the hospital switchboard activated the major accident plan.

Procedure for a major accident

The procedure for a major accident at this hospital (the major accident centre for south Manchester) was revised in the light of a Department of Health and Social Security health circular issued in 1977.¹ The procedure has been practised at regular intervals in cooperation with Manchester Airport. The response is based on a logical cascade callout list, resulting in an increasing response to the disaster. Triage, whereby immediate resuscitation is implemented and the less urgent problems of the arriving patients are deferred by senior medical staff, enables the needs of the patients to be assessed rapidly and the requirements on particular aspects of the hospital service to be determined early.

When the disaster plan was activated the accident and emergency department was cleared of non-urgent cases and the adjacent 12 bed five day ward, normally used for day case surgery, emptied of patients, who were transferred to other wards or discharged. The duty senior house officer in accident and emergency attended the first casualties, who arrived by airport coach. More seriously injured passengers arrived later by ambulance. Within 15 minutes of the activation of the accident plan the consultant in charge arrived and triage began. The intensive care unit, which had been closed for yearly maintenance, was prepared to accept patients and a 22 bed surgical ward emptied for potential admissions. The most distressed patients and those requiring medical attention were diverted to the treatment areas, whereas those with minor injuries were moved into a waiting lounge area adjacent to the five day ward.

The build up of medical and nursing personnel was complemented by the arrival of clerical staff with medical documents which are prepared for such an occasion and kept in numbered folders containing admission and treatment forms. Less seriously injured patients were soon anxious to trace and contact relatives; a telephone was made available to them in the waiting area and a choke point set up so that all patients were recorded on leaving the department, whether for admission, discharge, or x ray examination. Within 45 minutes of the arrival of the survivors at the hospital senior personnel of the local ophthalmology and regional burns unit were in attendance. Although orthopaedic and general surgeons also arrived, their services were not required. A police documentation team worked in the accident and

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emergency department with hospital staff, and a police bureau, in conjunction with social workers, correlated the list of inquiries with survivors. The hospital chaplains, helped by social workers and Women's Royal Voluntary Service staff, informed relatives of the deceased passengers.

The stand down was at 0956, less than three hours after the initial explosion.

Managing casualties

Seventy six survivors were seen at this hospital, three were taken to nearby Withington Hospital, and the flight crew remained at the scene. It became clear at the early stages of triage that the most seriously ill survivors were suffering from smoke inhalation and burns. Fifteen survivors with smoke inhalation were admitted, the criteria for this diagnosis being a history of smoke exposure, staining of the skin and clothing by smoke, production of carbonaceous sputum, conjunctivitis, hoarse voice, and wheezing on auscultation. Two patients also complained of chest tightness. Of the 15 patients admitted, seven were male and eight female (age 13-52), 14 had carbonaceous sputum and the remaining patient was unable to produce sputum, 11 were wheezy, three had a hoarse voice on arrival, two were dyspnoeic, and two had severe burns. Two had been previously diagnosed as asthmatic and none smoked. The most severely injured patient with 24% body surface area full thickness burns was in respiratory distress on arrival. His chest x ray film showed interstitial pulmonary shadowing compatible with the adult respiratory distress syndrome, and he was immediately intubated and transferred to the intensive care unit for ventilation. The remaining 14 patients were transferred to the surgical ward. Initial treatment was given with nebulised salbutamol and chloramphenicol eye ointment for conjunctivitis. The two asthmatic patients also received intravenous aminophylline (4 mg/kg six hourly) and hydrocortisone 100 mg six hourly. A 14 year old boy had in addition to smoke inhalation extensive partial thickness burns to both hands.

FIG 1—Carboxyhaemoglobin concentrations in 15 survivors admitted to hospital. Blood was drawn 45 minutes to three hours after exposure to fumes.

All 15 patients who were admitted had blood drawn for carboxyhaemoglobin estimation 45 minutes to three hours after the fire. Four samples had above normal concentrations (fig 1) (normal for non-smokers <5%), and all were considerably below expected concentrations (see Discussion). Chest x ray films of the remaining patients were normal as were blood gas estimations (the patient with the adult respiratory distress syndrome had severe hypoxia). All patients were producing large quantities (300-400 ml per 24 hours) of carbon streaked sputum, so regular two hourly physiotherapy was started. Four hours after admission a rapid deterioration occurred in one patient. Although hoarse and wheezy on admission, she developed signs of upper airways obstruction with stridor and breathlessness. She was transferred to the intensive care unit, intubated with difficulty, and ventilated. At intubation the vocal cords were balloon like with oedema. A further three

patients developed similar upper airways obstruction during the next six hours. All required intubation and ventilation, and at intubation all had laryngeal oedema.

Five hours after admission one asthmatic girl developed severe bronchospasm despite maximum bronchodilator treatment, and she was ventilated.

FIG 2—Lung function measurements showing forced expiratory volume in one second (FEV₁), forced vital capacity (FVC), gas transfer coefficient (KCO), and total lung capacity (TLC) for the less severely affected group. Serial results, where available, are connected by lines.

At intubation there were no signs of upper airways obstruction in this patient. Ten hours after admission two patients developed dyspnoea and hypoxia (oxygen pressure 8.0 kPa with 60% inspired oxygen through an MC mask). These patients were transferred to the intensive care unit and given 100% oxygen (through a Hudson mask at 15 l/min flow rate), but they were not ventilated. There were no signs of upper airways problems in these two patients clinically and on indirect laryngoscopy. Because of the glottic oedema and bronchospasm in some patients all were given intravenous steroids (200 mg hydrocortisone) within the first six hours of admission and thereafter six hourly and antibiotics (trimethoprim orally or cefuroxime intravenously) for the profuse sputum.

Recovery

The survivors now formed two groups: (a) the severe smoke exposure group in the intensive care unit, consisting of eight patients: one with the adult respiratory distress syndrome; four with laryngeal oedema and one asthmatic, of whom all were ventilated; and two with severe hypoxia despite treatment with 100% oxygen; and (b) the less severely affected patients—including the second asthmatic patient—who were observed in the surgical ward.

The following day the two patients with burns were transferred to the regional burns unit. The man with 24% full thickness burns died seven days later. The mildly exposed group improved rapidly, but all continued to experience hoarseness of the voice, episodic wheezing, and conjunctivitis. Peak expiratory flow rates, initially 60-70% of predicted values, rose to 100% within five days. Profuse bronchorrhoea continued, and regular physiotherapy was required.

Five days after the disaster all the mildly affected patients had been discharged, the more severely affected patients showing a slower recovery rate. Those with upper airways obstruction required ventilation for 24 to 48 hours and oxygen for a further 48 hours. The asthmatic patient was ventilated for 48 hours but then made a rapid recovery. The two patients

with hypoxia who did not require ventilation needed three to five days of treatment with 100% oxygen. All were given steroids, initially 800 mg hydrocortisone daily for 48 hours, thereafter prednisolone 30 mg reducing to zero and nebulised bronchodilators for seven days. A further patient who had been discharged from casualty was readmitted 72 hours after discharge complaining of shortness of breath, cough, and sputum production. He responded to physiotherapy and was discharged two days later.

phobias of travel (trains, cars, motorways), crowds, and smoky rooms had occurred often. There were no serious respiratory problems in the less severely affected group, and their symptoms had all resolved within three weeks of discharge. In the severely affected group shortness of breath and wheezing were common complaints. Episodes of painful eyes and difficulties with taste, smell, and hearing had also occurred. The respiratory symptoms of the two asthmatic patients had been more severe after the accident, and

FIG 3—Lung function showing forced expiratory volume in one second and forced vital capacity for six severely affected survivors, of whom four were ventilated.

Lung function

Lung function testing was performed as soon as patients were able to cooperate. Peak expiratory flow rates, forced expiratory volume in one second, and forced vital capacity were measured in the mildly affected group after 24 hours. Later full spirometry and gas transfer coefficients were measured. In the more severely affected patients pulmonary function was assessed as soon as assisted ventilation was discontinued. The mildly affected group showed lung function within the predicted range of normal (fig 2), but improvements in forced expiratory volume in one second and forced vital capacity occurred, especially in the asthmatic patient over the following seven days. The severely affected group showed gross impairment of both forced expiratory volume in one second and forced vital capacity (fig 3). Gas transfer was normal even when total lung volumes were reduced (fig 4). Four of the less severely affected group and three of the severely affected group had positive skin test reactions to common allergens.

they had required additional β_2 agonists and inhaled steroids. Lung function was normal in most patients (fig 5). One patient was unable to tolerate the rubber mouthpiece of the spirometer, which resulted in artificially low readings. Bronchial reactivity studies using histamine² showed appreciable hyperreactivity in the two asthmatic patients and mild reactivity in a further three patients (table).

Results of histamine reactivity in survivors three months after exposure to fire

Basal forced expiratory volume in one second (l)	% Of predicted forced expiratory volume in one second	PC ₂₀ (Concentration of histamine) (mg/ml)
4.41	100	>8
4.55	103	>8
3.97	101	>8
4.90	108	>8
3.29	106	>8
3.30	85	>8
3.85	128	>8
3.25	101	>8
3.25	81	7.4
3.41	110	6.5
2.70	112	4.8
3.30	97	0.6
3.51	103	0.2

PC₂₀=Provocative concentration (of histamine) that causes a 20% fall (in forced expiratory volume in one second).

Follow up

A review of survivors who required hospital admission was carried out 12 weeks after discharge. A mental health and respiratory symptoms questionnaire was completed for each patient. The patient who was discharged from the regional plastic burns unit was not reviewed, and a 14 year old girl (the first to require ventilation for upper airways obstruction) was not available, having had an emergency tracheostomy for subglottal stenosis shortly before review.

Psychological problems were common among all patients. Nightmares, feelings of guilt (even in those who did not lose friends or relatives), and

Discussion

The inhalation of toxic fumes or smoke is becoming more and more common in major accidents. Our experience suggests that plans for the assisted ventilation of large numbers of people in a short time need to be included in procedures for coping with major accidents. The management of the Manchester air disaster was made easier because it occurred when the cardiothoracic operating theatres were closed for yearly cleaning and thus the intensive care unit was empty. Triage enabled the rapid deployment of skills and facilities where they were most required. This was achieved as a result of careful three monthly rehearsals of the hospital personnel and departments concerned.

Inhaling smoke may result in several different injuries such as asphyxia, either from anoxia or from excessive carbon monoxide levels; upper airways obstruction; adult respiratory distress syndrome; and acute exacerbation of pre-existing airways disease.³ Each of these injuries was seen among those who survived this disaster. Despite the lack of cutaneous thermal injury, the relatively short exposure time to fumes, and the survivors having been evacuated from the aircraft within two minutes a large proportion of those who were admitted to hospital developed life threatening respiratory problems, the most common being upper airways obstruction.

FIG 4—Gas transfer coefficient and total lung capacity in six severely affected survivors, of whom four were ventilated. Serial results, where available, are connected by lines.

The inhalational injuries resulted from the toxic smoke fumes from the aircraft fire. The fabric of the inside of the aircraft would have been a mixture of polyvinyl chloride, chlorinated acrylics, urethane, isocyanates, polyacrylonitrile, polyurethane, and other flammable materials. The ignition of these materials would have released a highly toxic combination of hydrogen cyanide, hydrogen chloride, phosgene, nitrogen dioxide, isocyanates, carbon monoxide, and carbon dioxide (Civil Aviation Authority, personal communication). The toxic gases in the fumes, together with carbon particles coated by irritating aldehydes and organic acids, cause

injury of the upper and lower airways. Water soluble chemicals such as ammonia, sulphur dioxide, and chlorine combine with water in the mucous membranes to produce strong acids and alkalis that can lead to irritation, laryngospasm, bronchospasm, mucous membrane ulceration, and oedema. Gases such as hydrogen chloride, produced when polyvinyl chloride burns, can be transported on carbon particles to the lower airways, leading to bronchiolitis and alveolitis. Ciliary activity is grossly impaired, resulting in decreased mucous clearance⁴ and ultimately ventilation perfusion abnormalities.⁵

FIG 5—Lung function measurements in all survivors who were available for review at three months. FEV₁=Forced expiratory volume in one second. FVC=Forced vital capacity. KCO=Gas transfer coefficient. TLC=Total lung capacity.

Several important points are illustrated by this air disaster. Firstly, a major feature of such fires is the extremely rapid generation of large amounts of dense fumes, so that in the aircraft vision was lost within "seconds." Similarly, in the Woolworth's fire in Manchester dense smoke obscured exits in minutes (personal communication). Secondly, the frequent development of life threatening upper airways obstruction suggests that this should be anticipated and avoided by prophylactic use of endotracheal intubation and positive pressure ventilation. The survivors produced large amounts of smoke stained sputum, which was particularly troublesome and necessitated two hourly chest physiotherapy for the first 48 hours. Bronchial secretions were probably responsible for the marked V/Q abnormalities that were present. Hypoxia was temporarily relieved after physiotherapy, and we recommend regular physiotherapy for survivors of similar fires.

The carbon monoxide concentrations in our patients were considerably below those usually associated with smoke inhalation,^{6,7} being only marginally raised. These findings are contradictory to those of Clark *et al* in which a raised carboxyhaemoglobin level was considered diagnostic of smoke inhalation.⁶ The circumstances of the event we describe are not strictly comparable with those described by Clark *et al*, and therefore they cannot be compared. The use of steroids to treat all those who were seriously affected was empirical, and although the results of animal studies of the effect of steroids in the treatment of smoke inhalation suggest steroids are beneficial,⁸ it is not clear how they work.⁹ In a study of survivors of two hotel fires who were treated in four hospitals

Robinson *et al* found no benefit between those who were treated with steroids and those who were not.¹⁰ The results of further studies in patients suffering from combined burns and smoke inhalation injury suggest that giving steroids leads to increased mortality and morbidity, mainly because infection occurs more often.^{11,12} Early treatment of smoke inhalation focuses on keeping the upper airways open, and there is evidence that prophylactic upper airways intubation and continuous positive airways pressure ventilation should be carried out in anyone who gives a history of smoke exposure in an enclosed space or who on examination shows facial burns or singed nasal vibrissae or produces carbonaceous sputum.¹³ Fiberoptic bronchoscopy has been recommended as an aid to making an early diagnosis,¹⁴ but this was not performed on our patients.

The three month follow up of the survivors suggested that long term physical problems associated with exposure to these fumes are rare. The two asthmatics continued to show evidence of increased bronchial hyperreactivity and required more treatment to control symptoms than previously. A 14 year old girl who was ventilated for only 24 hours developed subglottic stenosis requiring surgical intervention but subsequently recovered. The lung function of the remaining patients was normal with no evidence of appreciable bronchial hyperreactivity. The results of follow up showed a high prevalence of psychological problems, and although we examined only those who were admitted to hospital, this may be a problem for all the survivors.¹⁵

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Personal Paper

After safe sex, safe surgery?

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"Look out for the point of the cutting diathermy!" said my boss as he handed it to sister. She drove it into the back of my hand with a nonchalant grace that would have brought tears to Eric Bristow's eyes. It would not have been so bad had it not been a patient with the acquired immune deficiency syndrome (AIDS) whose lung we were biopsying. So as we peeled off our double gloves and removed our helmets and overhoods, aprons, gowns, and overboots I ruefully rubbed my wound, wondering why we dressed up in this ridiculous garb and why on earth the orderlies were bothering to wash the walls, let alone under the sinks. We were not going to catch the human immunodeficiency virus (HIV) under the sink unless we... well it's an old joke.

The current barrage of publicity describing just exactly how to avoid catching or transmitting the virus gets the message exactly right. "Your sexual partner could be that very special person—the one that gives you AIDS." For sexual partner, read patient, or surgeon for that matter, because though probably not as risky as

intravenous drug abuse, surgery is certainly as risky as life on the sexual M25, if not necessarily in its fastest lane. The advice on safe sex does not mention wearing plastic aprons and overboots, or washing down the walls afterwards, yet still gives practical protection. It is high time that we had similar practical guidelines for the protection of both surgeons and patients.

But don't we already? No, categorically, we do not. The charade of dressing up before and scrubbing down afterwards when the patient is HIV or hepatitis B surface antigen (HBsAg) positive does not constitute safe surgery. Needled into action, I have produced some more practical guidelines for debate.

Hazards

Patients, operating theatre staff, and the operations are all potential hazards. To deal first with the patients. Should they all be screened for HIV before operation? Well why not? Hang the expense. I'd like to know. Some would argue that there is no need to screen everyone, just the high risk cases. The trouble is, you just can't tell who they are. Maynard noted that selective screening of over 6000 patients would have picked up only 10 of the 59 patients who were found to be positive for HBsAg.¹ Callender *et al* showed

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