Category 3

Other babies are judged to have a good chance of primary wound healing after operation, and these are placed in three grades of severity.

Grade 1—Active movement of the legs has been observed after birth. These babies need urgent operation because otherwise some of that active movement may be lost.

Grade 2—Babies in the intermediate group have certain active muscles such as hip flexors and adductors, and perhaps quadriceps. I think these are important muscles to preserve for the orthopaedic surgeon to use, and I would advocate urgent surgery.

Grade 3—At the other extreme there are babies who make no observed leg movements after birth; in these infants operation on the back will have no effect upon the muscular power in the legs. They can be treated either by a non-urgent operation (but within 24 to 48 hours to minimise infection) or by conservative means such as simple dressings.

The survivors, whether they have been operated on or not, will be offered surgery for hydrocephalus or for renal tract or orthopaedic problems to improve the quality of life or prevent deterioration.

Under no circumstances would I administer drugs to cause the death of the child. There is no doubt that those who are severely affected at birth will continue to be severely handicapped. But I conceive it to be my duty to overcome that handicap as much as possible and to achieve the maximum development of their potential in as many aspects of life as possible—physical, emotional, recreational, and vocational—and I find them very nice people. If in the *neonatal* period you are looking for opportunities to make sure they will die I am sure this attitude is likely to persist even when they are older. Some have been regarded as living completely miserable and unhappy lives.

Yet when I see them I find them happy people who can respond to concern for their personal welfare.

One final point. It has often been said by those who oppose abortion that the disregard for the life of a child within the uterus would spill over into postnatal life. This suggestion has been "pooh-poohed," yet in spina bifida there is a clear example of this. The equanimity with which the life of a 17-week-gestation spina bifida infant is terminated after the finding of a high level of α -fetoprotein in the amniotic fluid has, I think, spilled over to a similar disregard for the life of the child with spina bifida after birth.

Much of my surgical life over the past 30 years has been devoted to improving the quality of life of those who have been born with spina bifida. The attitude of mind that would eliminate all the severely handicapped reminds me of the poster issued by Christian Aid some years ago, which said "Ignore the hungry and they will go away"—to their graves. If we eliminate all the severely affected children with spina bifida there will be no more problem; but why stop at spina bifida, why not all the severely affected spastics, all those with muscular dystrophy, and all those with Down's syndrome? Why stop at the neonatal period? Our aim should be that life with spina bifida is the best possible life for that person in the family and in the community.

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Clinical Topics

Management of airway complications of burns in children

ELENA VIVORI, R E CUDMORE

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Summary

Children who have been exposed to smoke in a confined space or who have soot or burns, however minimal, on the face should be admitted to hospital. Respiratory distress may be delayed, but if it is progressive the patient should be curarised, intubated, and mechanically ventilated. Unless ventilation continues for 48 hours, followed by 24 hours' spontaneous respiration against a positive airway pressure, stridor and pulmonary oedema may recur. An endotracheal tube small enough to allow a leak between it and the oedematous mucosa must be passed to prevent laryngeal damage and subsequent subglottic stenosis. High humidity of inspired gases keeps

secretions fluid and the endotracheal tube patent. A high oxygen concentration compensates for deficient oxygen uptake and transport caused by pulmonary lesions and the presence of poisonous compounds interfering with oxygen transport. Dexamethasone to minimise cerebral oedema and antibiotics to reduce the incidence of chest infections should be given.

Introduction

It is increasingly recognised that accidents producing burns or scalds may also damage the respiratory tract, and that this damage may be much more serious than the extent of the surface injury would suggest.

A pulmonary syndrome caused by inhaling hot irritant fumes was first described in 1942¹⁻⁴ after the Cocoanut Grove Night Club fire in Boston, which killed 491 people and seriously injured many more. Only a few of the 114 victims taken to the Massachusetts General Hospital had extensive surface burns. Of the 75 who were dead on arrival, the cause appeared to be anoxia caused by inhaling irritant gases and by carbon monoxide poisoning. Only 3 of the 39 who survived long enough to receive

Alder Hey Children's Hospital, Liverpool 12

ELENA VIVORI, FFARCS, consultant paediatric anaesthetist
R E CUDMORE, FRCS, consultant paediatric surgeon

treatment had no respiratory symptoms, and all the subsequent deaths were due to pulmonary complications. From 1942 to 1962 42% of the fatally injured patients treated at the Massachusetts General Hospital died of damage to the respiratory tract after inhaling hot irritant fumes.⁵⁻⁷

Treatment of respiratory damage in adults with oxygen, humidity, and mechanical ventilation has been described,⁸⁻¹¹ but few such cases in children have been reported. In Dellwood in 1955¹² 13 newborn babies died after a fire that started by smouldering under a wood and linoleum floor in the night nursery of a small maternity home. Three presented immediately with respiratory distress and were treated with oxygen and humidity. The others appeared to be in good general condition, but after a few hours started to deteriorate rapidly, and despite oxygen, humidity, antibiotics, and steroids, all but two died within three days. Only one baby received burns, which were superficial. At necropsy the trachea and bronchi in all cases showed membranous casts, as well as mucopurulent material flecked with carbon particles and frank pulmonary oedema. Two cases of respiratory distress occurred in older children who had inhaled smoke but had no surface burns.^{13 14}

We have the impression, in Liverpool, that more children are suffering from the effects of inhaled fumes and smoke sustained in house fires, probably because of the widespread use of polyurethane materials and foam in bedding and furniture. These children have not necessarily received thermal injury to the rest of the body.

Injuries to the respiratory tract from fume inhalation are caused by various agents that may coexist and cause different damage at different levels of the respiratory tract. Inhalation of hot dry gases causes thermal injury mainly to the nasopharynx and larynx, because the temperature of the gases drops very rapidly on entering the air passages, and there is insufficient residual heat to burn the lung tissues.

Inhaled smoke causes chemical damage to the airway, whose extent depends on the composition of the smoke but which typically affects the whole of the airway and particularly the lung tissue.^{15 16} Likely substances are oxides of nitrogen, sulphurous compounds, many aldehydes, and hydrochloric acid.^{15 17} Smoke also commonly contains purely poisonous components. Carbon monoxide, although not a respiratory irritant, is extremely toxic, causing hypoxia by preventing the carriage of oxygen by binding haemoglobin to produce carboxyhaemoglobin; this then causes a shift to the left of the dissociation curve of oxyhaemoglobin, thus further reducing the oxygen available to the tissues.

Smoke from burning polyurethane foams is particularly damaging as various products are given off at different temperatures. Initially the urethanes break down to the basic isocyanates. At 500 °C these isocyanates decompose to hydrogen cyanate, ammonia, and carbon monoxide. At 800 °C cyanates and acrylonitriles are produced, while at 1000 °C hydrogen cyanide predominates.¹⁸ Some deaths attributed to smoke may well be caused by cyanide poisoning. Cyanide inhibits cytochrome B, one of the enzymes concerned in cellular oxygen transport, and is one of the most rapidly acting poisons. Even if the polyurethane foam does not ignite, the smouldering produces serious effects because of "smoke logging" and a house or factory may be filled with smoke before people have time to escape. Inhalation of these fumes causes a sensation of choking, a metallic taste in the mouth, dizziness, wheezing, and loss of balance.

The underlying lesions include laryngeal oedema and laryngotracheobronchitis with formation of sloughs and pseudomembranes, which may become detached after varying intervals, causing complete airway obstruction.¹⁹ Pulmonary lesions consist of areas of haemorrhage and oedema. Secondary infection is often superimposed on these lesions. The result of all these causes and lesions is hypoxia, which may be delayed. Patients should therefore be observed closely and intensive care measures started early.

Since 1973 we have had an increasing number of children rescued from house fires presenting with immediate or subse-

quent respiratory damage, whether or not they had surface burns. The following case histories provide the basis for our present routine management. Case 6 has already been reported.²⁰

Case reports

Case 1—An 8-year-old girl suffered 25% surface burns, including her face and neck. She was semi-conscious and violent on admission, with obstructed breathing. The carboxyhaemoglobin level was 6%. Because of her obvious hypoxia she was curarised and ventilated for 24 hours. She was then allowed to breathe spontaneously against a positive end expiratory pressure, but she rapidly developed clinical pulmonary oedema. She was therefore recurarised and ventilated for a further 24 hours, after which she was successfully extubated, remaining conscious and rational.

Case 2—A 3-year-old boy was admitted with 35% burns including face, neck, chest, and back after being found in a smoke-filled room. He was conscious and the airway appeared satisfactory, although there was slight stridor. His colour was good, and he was not restless. The carboxyhaemoglobin level was 7%. He was treated with humidity and oxygen in a head box and appeared to be progressing reasonably, when three days after admission he had a cardiac arrest during chest physiotherapy, possibly owing to detachment of a tracheal slough. He was resuscitated but suffered severe brain damage and is now decerebrate.

Case 3—A 1-year-old boy was rescued with his brother from a smoke-filled room, where furniture was burning. There were superficial burns of head and hands. Unconscious and apnoeic when removed from the room, he was resuscitated at a nearby hospital. He arrived here breathing spontaneously through an endotracheal tube, which was retained. High humidity, oxygen, and frequent endotracheal suction with physiotherapy were maintained. Endotracheal suction produced large amounts of sooty secretions. After 48 hours he appeared to be improving and was extubated. His airway condition was satisfactory, but he was kept in humidity and carefully observed. Twelve hours later he quite suddenly developed acute pulmonary oedema and had a respiratory arrest. He was promptly resuscitated and ventilated but never regained consciousness and died two days later.

Case 4—A 2-year-old brother of case 3 was in good general condition on admission, though with superficial burns (10%) of face and shoulders. About three hours later he developed stridor. Laryngoscopy showed pharyngeal and laryngeal oedema, so an endotracheal tube was passed. He was curarised and ventilated for two days while receiving dexamethasone and antibiotics. He was then allowed to breathe against a positive end expiratory pressure; clinical appearance and blood gases were satisfactory. After some hours he had an unexpected respiratory and cardiac arrest. During resuscitation the endotracheal tube was found to be completely blocked by a soot-stained slough. As more slough was seen protruding through the cords he was bronchoscoped. Large pieces of pseudomembranes were removed from the trachea before reintubating and ventilating him for a further 48 hours. He eventually made a complete recovery.

Case 5—A boy aged 11 was rescued from a house filled with smoke from burning soft furniture. He had severe burns (60%) of face and neck, and showed signs of severe inhalation of smoke and fumes. He was slightly cyanosed and very restless. He was curarised and ventilated for 48 hours, with frequent endotracheal suction that produced thick, soot-stained secretions. He received dexamethasone and antibiotics. Although recovering well from his respiratory tract damage, severe hypoxic brain damage had been sustained and for about six months he was totally unresponsive and grossly spastic, and needed total nursing care. He then began to be aware of his surroundings, and his neurological state has since slowly and steadily improved. One year after the accident he was discharged to a residential school.

Case 6—An 8-year-old boy was rescued, deeply unconscious, from a house fire. He had a respiratory and cardiac arrest in the ambulance and was being resuscitated on arrival at the Royal Liverpool Children's Hospital. He had a convulsion followed by decerebrate rigidity with transiently dilated and fixed pupils, presumably caused by cerebral hypoxia. He was promptly curarised and ventilated, using 100%-inspired oxygen for several hours, as his initial carboxyhaemoglobin level was 80%. He was given mannitol and dexamethasone and was extubated 24 hours later. Almost immediately, however, he became greatly distressed, with severe stridor and bronchospasm, progressing to pulmonary oedema and circulatory collapse. He was therefore reintubated and ventilated for 24 hours, allowed to breathe against a positive airway pressure for a further 24 hours, and then successfully extubated. He completely recovered over several weeks.²⁰

Discussion

Some children injured in house fires may already be suffering from airway obstruction on admission and will need urgent initial treatment. It is important, however, to consider the possibility of delayed onset of respiratory distress in previously seemingly unaffected children. Chiefly at risk are those who (a) have been exposed to thick smoke, especially from smouldering plastics, and (b) present with soot or burns, however minimal, on their faces. These children should be carefully observed. In the presence of restlessness due to hypoxia, increasing respiratory distress, and deterioration in the blood gases the patient should be curarised, intubated, and mechanically ventilated. This ensures an airway clear of secretions, adequate ventilation adjusted according to blood gas analysis, and guaranteed oxygenation. This is especially important in view of the deficiency of oxygen uptake and transport due to pulmonary lesions or to the presence of poisonous compounds interfering with oxygen transport. Oxygen consumption is of course decreased by the muscle relaxation induced by curarisation.

Because of laryngeal and tracheal oedema an endotracheal tube several sizes smaller than that expected for the child's age may be needed. It is vital that the endotracheal tube allows a slight leak around it to avoid any pressure on an already damaged mucosa. The inspired gases must be humidified, and physiotherapy must accompany endotracheal suction. Dislodgement of mucus and sloughs and consequent acute obstruction is an ever-present danger during the first week after injury.

Mechanical ventilation should be continued for 48 hours, followed by 24 hours of spontaneous respiration against positive airway pressure. Too early extubation may be followed by laryngeal obstruction or pulmonary oedema (cases 1, 3, and 6). In view of the late, and sometimes disastrous, onset of pulmonary oedema, which may be of a chemical origin, the positive end expiratory pressure should be reduced progressively before extubation is carried out. Small doses of morphine or promethazine should be given when necessary to ensure adequate sedation during mechanical ventilation with muscle relaxants and spontaneous respiration through the endotracheal tube. Super-added infection is likely, if not certain; so antibiotics should be given.

Dexamethasone is also used in an attempt to decrease cerebral oedema caused by hypoxia. Despite this some of our children did develop signs of cerebral irritation. Whether such signs are due entirely to hypoxic cerebral oedema or directly to poisoning is uncertain. This might explain the late and unexpected death of one patient (case 3) and the decerebrate state of another (case 2) despite immediate resuscitation after their respiratory and

cardiac arrests. Undoubtedly poisoning, both with carbon monoxide and hydrogen cyanide, does play a part. Unfortunately, carboxyhaemoglobin concentrations were not always ascertained in our series. Carboxyhaemoglobin and blood cyanide concentrations are now requested in these children on admission.

Skin burns vary in their extent and, if needed, routine intravenous fluid resuscitation will need to run concurrently with respiratory treatment.

The long-term pulmonary effects of these fumes are not clear, and survivors should be followed up. All our patients who have survived have normal chest radiographs.

An increasing number of these children will probably present themselves to casualty departments. Although flame retardant urethane foams are produced they are not widespread, and even they can be dangerous because once a fire starts they burn fiercely.²¹ Legislation is not yet contemplated as it is difficult to see how it would work. Further fire tragedies are bound to occur, but with vigorous and aggressive treatment unnecessary respiratory deaths may be prevented.

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Is there any treatment to increase sphincter tone after impairment of the muscle at haemorrhoidectomy?

Unless the haemorrhoidectomy has been completely bungled, the sphincter mechanism of the anal canal should remain anatomically intact, since the sphincter muscle is not encroached on in removing the haemorrhoids. It is true, however, that some impairment of anal continence may nevertheless follow haemorrhoidectomy—for example, Watts and his colleagues¹ in a follow-up of 100 patients six months after haemorrhoidectomy found that 13% complained of occasional leakage of flatus, 3% occasional inadvertent passage of faeces when the stools were liquid, and 7% soiling of their underclothes; one or more of the defects was found in 20% of the patients. The incidence of these disturbances is temporarily increased if the sphincters have been stretched by the surgeon at haemorrhoidectomy. Probably at least some of these cases are due more to loss of anal sensation than any impairment of sphincter mechanism, and it is simple enough, by digital examination of the anal canal, to determine whether or not the patient has adequate sphincter control. In the few cases where it is not adequate, a course of galvanic stimulation to the anal sphincter might be arranged. It is true that clumsy surgery may actually damage the

anal sphincter mechanism, rendering the patient frankly incontinent of faeces. In such cases a direct repair of the damaged sphincter muscle is technically possible.

¹ Watts, J M, et al, *British Journal of Surgery*, 1964, **51**, 88.

Is gentamicin taken with lemonade a useful adjunct in managing ulcerative colitis, or will it exacerbate symptoms? If gentamicin is given by mouth could this lead to resistant strains of colonic bacteria developing?

The cause of ulcerative colitis is not thought to be bacterial, and antibiotics given by mouth have not seemed helpful in treatment (though parenteral antibiotics are sometimes needed because of secondary septicæmia). Gentamicin is poorly absorbed, and when given by mouth acts only within the gut lumen. It is unlikely to exacerbate symptoms, though no evidence is available to suggest that it will help, either. Resistant strains of bacteria may always develop if an antibiotic is given for long enough. Nevertheless, gentamicin used for infantile *Escherichia coli* enteritis in a dose of 27 mg/kg/day for ten days has not caused clinically important antibiotic resistance.