Severe burns in children, 1964-1974

J. J. COGSWELL and A. C. CHU

From the Department of Paediatrics, Guy's Hospital, London

Cogswell, J. J., and Chu, A. C. (1976). *Archives of Disease in Childhood*, 51, 67. **Severe burns in children, 1964-1974.** 580 children were admitted to the paediatric burns unit of Guy's Hospital between 1964 and 1974, of which 97 had burns exceeding 20% of the surface area, and 33 died (34% mortality). 80% of those with burns exceeding 50% of the surface area died. Young children died after less extensive burns.

Respiratory failure, sepsis, and malnutrition were the most lethal complications. The prompt use and careful control of intravenous fluids had reduced the immediate complications associated with shock, and acute renal failure is now uncommon. Respiratory failure resulted in many deaths during the first week after injury. The need for intensive respiratory care involving paediatric, anaesthetic, and surgical staff is stressed.

Sepsis and malnutrition remain major threats to survival. Improved methods of bacteriological control by laminar air flow units and topical antibacterial agents may help to reduce infection in the future. Reduction of energy expenditure by temporary skin coverings and a high environmental temperature, combined with a high calorie intake by oral and intravenous routes, may improve the outlook for severely burned children in the next decade.

Accidents remain a major cause of death in children. Burns and scalds involving less than 10% of the total body surface area seldom cause severe illness or death, but those involving more than 10% are a medical emergency, and over the last 20 years it has been general policy to admit such injured children to specialized burns units within a paediatric ward or hospital. The purpose of this paper is to summarize experience over the last 11 years (1964-1974 inclusive) in the Paediatric Burns Unit of Guy's Hospital, London. The complications which affected outcome are analysed. A summary of the treatment given is included, but no attempt is made to review the total management of a burned child. Newer methods of management are discussed in the hope of reducing morbidity and death in the next decade.

Patients

Between January 1964 and December 1974, 580 children were admitted to the paediatric burns unit, of which 97 had burns exceeding 20% of the surface area. Hospital records of these 97 severely burned children were examined, though only in 67 patients were case

notes complete. The clinical course of the child after the injury was recorded with particular reference to complications. 33 of the 97 children admitted with severe burns died, and necropsy reports of 25 were traced.

How the burn was sustained. Of the 67 children whose case notes were complete, 50 had received flame injuries and 17 were scalded.

Flameburns. Of the 50 children who sustained severe flame burns only 7 were injured outside the home. These 7 were all over 8 years of age and received their injuries from electric power cables 2, gas explosions 4, and a bonfire accident 1. 43 children were burned by fire within the home. Clothing catching alight on unprotected gas or open fires resulted in 22 injured children, unprotected gas cooker 3, and accidents involving paraffin heaters accounted for a further 13. 9 of the 13 accidents with paraffin heaters occurred in the homes of immigrant families. In not less than 20 instances the children were alone in the house at the time of the accident.

Scalds. 17 children were admitted with scalds to their bodies exceeding 20% surface area, all sustained while in the home. In 11 the injury was sustained in the

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kitchen or living room from a spilled kettle, saucepan, or teapot. 5 children were scalded in the bath, and one child was injured by the spillage of a caustic alkali.

Age at time of injury. Of 36 children who were under the age of 5 years at the time of their accident, 15 suffered scalds and 21 flame burns. Of the 31 children of 5 years or over, 29 had sustained flame burns, while only 2 had scalds.

Management in hospital

Severely burned children admitted to the unit were managed jointly by the paediatric staff and plastic surgeons. Initial measures were designed to combat shock, control infection, and to restore physiological and psychological normality. Though there have been modifications in individual patients, an established routine for the management of burned children has not changed greatly over the last 11 years.

Procedures on admission. Assessment of vital functions, including adequacy of the airway, was carried out. The child's clothing was removed, and the child weighed. Sedation with heroin (0.1 mg/kg) was given intravenously. Tetanus toxoid (0.5 ml) was given intramuscularly, together with penicillin prophylactically. An estimate of the extent of the burn was made, and a photograph taken. A self-retaining Foley catheter was inserted, and urine sent for analysis. A blood sample was taken for baseline values for electrolytes, urea, and full blood count.

Care of burn. All burns over 20% were treated by exposure. Each child was nursed in a single cubicle in an environmental temperature of 32°C. Shortly after admission each patient received the first burn toilet under heavy sedation. Circumferential burns of chest or limbs were treated by skin splitting procedures where necessary. Bathing and debridement were further undertaken on the 10th day. Areas of deep skin loss were grafted at a later date at the discretion of the plastic surgeons.

Intravenous fluid replacement. Intravenous fluids were given to all children who had received burns involving more than 10% of the body surface. The volume and type of intravenous fluid used was based on the scheme outlined in the Table. Frequent adjustments to the rate of flow were based on observations of blood pressure, urinary output, body weight, serum electrolytes, and packed cell volume. The aim of the intravenous fluid regimen during the first 7 days after injury was to maintain urine flow at 40 ml/m² per h, and to maintain a urinary sodium between 20 and 80 mEq/l. In the serum the aim was to maintain the sodium at 130–140 mEq/l, and potassium at 3.5–5.0 mEq/l. Recently, the trend has been to use more crystalloid solutions in the acute phase of treatment, and to reserve plasma for those children in whom the serum protein concentration falls below 3 g/dl.

TABLE
Fluid regimen

The following scheme was used to assess fluid requirements during the first 48 hours after injury. Estimate of the body weight and surface area covered by the burn were required. By reference to the following chart the percent of total fluid volume in litres was estimated, assuming this to be equal to the body weight in kg.

Surface area burned (%)	% fluid volume (l)
10–15	5–7
15–20	10–15
20–30	15–20
30–50	20–30
> 50	30

For example: for 50% burn in a child weighing 10 kg, 30% of 10 l given in 48 h. This volume of fluid is given as (a) $\frac{1}{3}$ in 8 h; (b) $\frac{1}{3}$ in 16 h; (c) $\frac{1}{3}$ in 24 h. The fluid consists of alternate bottles of plasma and saline, + KCl 40 mEq/l. In periods (b) and (c) basal requirements were added to the fluid regimen. Blood was given if Hb was low.

Management of oliguria. Oliguria in the first few days after injury was deemed to be present if the child's urinary output was <300 ml/m² per day. The common cause was inadequate replacement of fluid, and such prerenal uraemia was characterized by increased urinary osmolality (>800 mOsm/kg) and a high urinary urea. Management was aimed at correcting the hypovolaemia by increasing the intravenous fluids.

Acute renal failure was suspected if oliguria (<0.5 ml/kg per h) was associated with isotonic urine (approximately 300 mOsm/kg), a high urinary sodium concentration, and a low urinary urea concentration. The urine was also examined for pigment casts to exclude a pigment nephropathy. Oliguria of renal origin was treated by promoting diuresis with 20% mannitol, or large doses of frusemide (2 mg/kg).

Respiratory care. Assessment of the respiratory state of the patient was carried out on admission. In those children with facial burns, or a history of exposure to smoke, careful attention was paid to the respiratory rate, presence of stridor, or restlessness. Acute upper airway obstruction was managed by a nasotracheal tube, or tracheostomy. Large doses of corticosteroids were given to some patients who had sustained a severe airway burn. Subsequent respiratory status was also monitored by arterial blood gas determinations. Hypoxaemia was corrected by oxygen enrichment of the inspired air. Respiratory failure was managed by intermittent positive pressure ventilation. Radiological evidence of lung consolidation was treated by antibiotics and physiotherapy.

Control of sepsis. Each child was barrier-nursed in a single cubicle. Attendants wore gowns and masks, together with sterile gloves when handling the patient. Penicillin was given to all patients as prophylaxis against streptococcal skin infection. Frequent bacteriological cultures were taken from the intact skin, the wound

site, and the nose and throat. A catheter sample of urine was sent for culture.

Pyrexia, tachycardia, and leucocytosis were not of themselves interpreted as infection, but bacteriologically proven infections of wound or blood were treated with systemic antibiotics. There was no consistent policy regarding the use of topical antimicrobial agents. Before 1970 topical antibiotic sprays (Polybactrin) were applied to the burn, but this was later abandoned because of local pain after application. Occasional use was also made of chloramphenicol dusting powder after debridement. More recently mafenide (Sulfamylon) cream and silver sulphadiazine ointment has been used in selected patients.

Psychological care. Many severely burned children exhibited some signs of cerebral dysfunction after the burn injury. Restlessness, hallucinations, delirium, or a depressed level of consciousness were indications of possible contributory causes such as hypoxia, hypovolaemia, or sepsis. In all cases an experienced medical social worker undertook the psychosocial support of the family. Feelings of fear or isolation by the injured child were kept to a minimum by daily contacts with parents, sibs, and nursing staff. Overtly disturbed children were managed by a child psychiatrist.

Results

Survival from severe burns. Of the 79 children with burns exceeding 20% surface area, 33 died (34% mortality). The more extensive the injury the worse was the prognosis (Fig. 1). 66 children had burns of less than 40% surface area, and 5 of these children died, 4 being under

FIG. 1.—Outcome related to extent of the burn in 97 children with burns exceeding 20% (57 cases from 1964-1969; 40 cases from 1970-1974).

14 months of age. 22 children were admitted with burns exceeding 50% total surface area, and of these only 4 survived. There were no survivors if the area of skin burned exceeded 80% of the total. Changes in mortality during the first and second halves of the decade are shown in Fig. 1, but numbers are too small to draw a statistical conclusion concerning the change in prognosis over the 11-year period.

Analysis of deaths. Of the 33 fatalities, a necropsy report (usually from the forensic pathology department) was traced in 25. Where no necropsy report was found the cause of death was based on the clinical assessment before death. Where septicaemia was listed as the cause of death a positive blood culture had been obtained before death.

Fatal complications in order of frequency were respiratory failure 15, septicaemia 12, renal failure 4, and cerebral death 2. Of those 15 children who died from respiratory failure, 6 sustained a major burn or blast injury to the airway, 4 died of post-traumatic respiratory insufficiency, and in 5 bronchopneumonia was the terminal event (Fig. 2). In 8 of the 12 fatal septicaemias *Pseudomonas pyocyanea* had been cultured from the blood before death.

FIG. 2.—Causes of death in 33 fatal burn injuries.

Renal failure had not been a contributory cause of death since 1968.

Time of death after admission. (Fig. 3). Acute burns or blast injuries of the airway were the major cause of death in the first 48 hours. Post-traumatic respiratory insufficiency was a major cause of death after the second day after injury.

FIG. 3.—Timing of complications after the thermal injury in 33 fatal cases.

Septicaemic deaths were at their peak during the second week after admission. After being in hospital over 4 weeks, 3 children died from septicaemia compounded by chronic malnutrition.

Analysis of complication in 67 patients. An analysis was made of the 67 cases where the burn exceeded 20% and where the case records were also complete.

Respiratory complications. Acute blast injury to the airway occurred in 7 children who had been burned in a confined building. Stridor and airway obstruction occurred within 24 hours of the injury. Despite tracheostomy and oxygen therapy, 6 of these children died. Between the second and fifth day after injury 20 children developed signs of tachypnoea and hypoxaemia. Chest x-ray showed pulmonary oedema or atelectasis. 4 of the 20 children with respiratory distress died. At necropsy the lungs showed patchy atelectasis, interstitial and intra-alveolar oedema, and intra-alveolar haemorrhage. Bronchopneumonia was diagnosed in 9 patients, 5 of whom died.

Renal complications in 67 children. Almost all the 67 children showed some oliguria in the first 24 hours after injury. In 6 children only did complete anuria due to acute renal failure occur. 3 of these were severely burned (70%) and died of respiratory complications, and 3 received peritoneal dialysis. 2 anuric children recovered renal function and made a full recovery. No case of anuria has occurred since 1968. Urinary tract infections were commonly noted. 10 of the 67 children had one or more proven urinary tract infections.

Sepsis in 67 children.

(a) **Blood stream infection.** In 10 children there was bacteriological evidence of septicaemia due to *Ps. pyocyanea*. All these children were treated with gentamicin, and all but 2 died. Septicaemia due to *Staphylococcus aureus* 3, *Proteus* 2, and yeasts 1 were also noted.

(b) **Infection of the burn wound.** Positive bacterial cultures were obtained from the burnt area in 66 of the 67 burnt children. The child whose wound remained sterile throughout his hospital course had received topical antibiotics (Polybactrin). The organisms which infected the wound were *Ps. pyocyanea* (23 infected wounds), and *Staph. aureus* (14 infected wounds). Less frequently *Protus*, *Staph. albus*, or yeasts were isolated.

Pseudomonas was cultured from the wound as frequently in the first 5-year period as in the more recent 6-year period. A positive wound culture for *Pseudomonas* occurred at any time after admission, but in at least 5 patients the organism could be cultured from the wound surface within 4 days of admission.

Pyrexia. All 67 patients had a fever starting on the first or second day after injury. This swinging pyrexia lasted for a variable time and returned to normal as the wound healed. The mean duration of fever was 28 days. The fever did not correlate with positive cultures from wound.

Discussion

Burns remain an important cause of death and disability in children. Of 67 severely burned children admitted to the unit over the last 11 years, no less than 60 accidents occurred inside the home. Though detailed social histories were not available in all instances, a high index of 'social disorganization' was usually found in the families of burnt children. 20 flame-burnt children had been left alone in the home at the time of the accident. Paraffin heaters were known to have been responsible for at least 13 of the accidents, 9% of these occurring in immigrant families. As might be expected, scald injuries predominated in babies and young children, while flame burns were commoner in older, more adventurous children. Scalds commonly occurred in the kitchen or living room, but the bathroom was also a dangerous place, especially for young children.

Pulmonary complications were associated with a high mortality. This observation has also been

made in severely burnt adults (Achauer *et al.* 1973). In this series respiratory complications were a major cause of death. 6 of the 7 children who sustained a direct burn of the airway died within the first week of admission. Airway obstruction due to oedema of the upper airway occurs within the first 24 hours after the injury and is probably due to blast injury or toxic gases. Management of the airway by endotracheal intubation, bronchoscopy to remove endotracheal slough, and assisted ventilation when necessary, remain the approach to these problems.

Twenty of the children in this series developed post-traumatic respiratory distress, and 4 of these died. The features of this syndrome have been described (Moore *et al.* 1969). Within 5 days of the injury the children developed respiratory distress with increasing hypoxaemia and patchy shadows on chest x-ray. At necropsy the lungs showed atelectasis, pulmonary oedema, and pneumonitis. The essential lesion appears to be an increase in vascular permeability to plasma and red cells. There is some experimental evidence that the lung lesion is caused by circulating endotoxin (Fine, 1973). Treatment of respiratory distress in patients after a severe burn includes correction of hypoxaemia with oxygen, and where necessary intermittent positive pressure ventilation. Intravenous fluid overload may predispose to pulmonary oedema, and monitoring of central venous pressure, or even mean pulmonary artery pressure, has been recommended (Achauer *et al.*, 1973). Though fluid overload must be avoided, maintenance of an adequate circulating blood volume after the shock phase is important (Hinton *et al.*, 1972). Once respiratory distress is established the use of a continuous distending pressure to the airway may be logical therapy, and there are claims that exchange transfusions reduce mortality (Kummer-Vago and Bettex, 1973).

Bronchopneumonia was diagnosed 5 or more days after the burn occurred in 9 children, and 5 recovered with antibiotic therapy and physiotherapy.

Renal complications were not a major cause of death. Though oliguria in the first 24 hours was very common, it was frequently due to inadequate replacement of fluid volume. Liberal use of intravenous fluid in the first day after injury, combined with an increasing awareness that fluid infusion rates must be closely controlled after the first 24 hours, has resulted in no cases of acute renal failure in the last 5-year period.

Sepsis is currently one of the commonest and most lethal of the complications in this unit. It was an important contributory factor in the deaths

of the last 6 fatally burned children. It is a particularly dangerous complication in the very young children. The organism most frequently responsible for local infection and septicaemia was the *Ps. pyocyanea*. This organism has been repeatedly isolated from the unit over the last 11 years, and it is hoped that the new unit about to open at this hospital may escape this situation.

Surgical management of burns over the last 11 years has usually been by exposure (Wallace, 1949). Recently other approaches to the dual problems of sepsis and evaporative water loss have been studied in other centres. It is reported that the rate of evaporation of water from the burnt surface can be reduced by the application of autograft or cadaver homograft skin to the burned area (Lamke *et al.*, 1971). Temporary dressings also serve to control infection until re-epithelialization can occur.

Topical applications are under continuous review in an attempt to reduce wound colonization and delayed healing. Mafenide cream diffuses through the burn eschar and is reported to be effective prophylaxis against *Pseudomonas* (Lindberg *et al.*, 1965), but it can cause acidosis. Silver sulphadiazine also has a wide spectrum of antibacterial activity without causing pain or electrolyte disturbance (Standford, Rappole, and Fox, 1969).

Another advance in the care of burnt patients is the introduction of laminar air flow isolation units, such as are planned for this paediatric department. Bacteria-free air is distributed over the patient in a uniform shower. Since the air pressure is maintained slightly positive, in respect of the external environment, airborne bacteria are excluded. Humidity and air temperature can be controlled. The use of warm air reduces the enormous energy requirements of burnt patients. This is important because it has been shown that many of the serious metabolic consequences of an extensive burn are attributable to the need to provide energy for the evaporation of water from the burnt surface (Davies and Liljedahl, 1971). The management of the child with very extensive burns is still difficult. The use of a high-air-loss bed in which the patient is supported on sterile humidified air may reduce risks of infection (Scales, 1971), but the problems of high installation cost and noise in operation have still to be overcome.

Severely burnt children who survive the acute stage after their thermal injury may die weeks later from sepsis and malnutrition (Fig. 2). Adequate nutrition is essential from an early stage even before re-epithelialization has started. A high calorie

intake is required to sustain the adaptive hyper-metabolic state, to replace protein lost by exudation, and to support synthesis of immunoglobulins and structural protein (Blocker *et al.*, 1968). Anorexia is frequently present after a burn injury, and it is often necessary to feed a fluid diet to the child through a nasogastric tube. Such a diet should supply at least 3000 cal/m² to meet the needs of energy metabolism and healing, and contain 3–5 g/kg per day of protein.

In many burnt children it is not possible to achieve a sufficiently high calorie intake orally, and in such situations supplementation by intravenous feeding is probably essential. While experience of such techniques in burnt children is limited, recent work on burnt adults has shown a marked decrease in mortality when given 6000–6500 cal/d, half the calories being given intravenously (Lamke, Liljedahl, and Wretling, 1974). A high calorie intake combined with a warm environment reduces the endogenous breakdown of protein, and hyper-osmolality is avoided. Prolonged intravenous feeding, now known to be feasible in children may help to maintain the nutrition of children with severe burns.

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- Correspondence to Dr. J. J. Cogswell, Department of Paediatrics, Poole General Hospital, Dorset.