INHALATIONAL BURNS IN CHILDREN

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1. INTRODUCTION

This study began in 1990 in the Burn Unit of The Red Cross War Memorial Children's Hospital (RCWMCH) in Capetown. It came to our attention that children in the Burn Unit developed respiratory problems. These were complications of fireburns, smoke inhalation, explosions and even hot water scalds. They presented with a wide and confusing array of symptoms and many failed to improve with the symptomatic treatment given. Greater understanding of the pathology was needed in order to investigate and manage these problems correctly. The ultimate aim of this study was to establish a treatment protocol that could be followed by junior staff.

1.1 Historical Perspective

The problem of inhalational burns first reached public attention in 1942 when 491 people died after being trapped in a fire at the Coconut Grove Nightclub in Boston (Saffle,'93). 144 patients arrived at the Massachusetts General Hospital (Aub,'43;Cope,'43), and were noted to have 4 major modes of presentation. The first group arrived in extremis with upper airway damage and severe hypoxia. They died within a few hours of admission. The second group developed respiratory distress within a few hours. This resembled pulmonary oedema with cyanosis, tachypnoeia and frothy secretions. A third group became symptomatic after 24 hours with airway obstruction due to laryngeal oedema. The fourth group survived the initial resuscitation but developed bronchospasm, atelectasis and pneumonia after 48 hours.

The work of Joseph Moylan and his anatomical description of inhalational injury (Moylan,'80) did much to further our understanding of these confusing presentations and established the use of specific investigations to determine the anatomical site of injury (See **Figure 1.1**). Thus fibreoptic bronchoscopy soon became an essential tool in the management of laryngeal and major airway burns (Moylan,'75;Mellins,'75;Fry,'77) while Xenon 133 lung scan proved useful in confirming lung parenchymal damage.

Meanwhile, progress was being made regarding the cause of the damage. It was established that hot dry gases caused damage to the upper airway, while steam could

contains noxious solids, liquids and gases which can cause chemical damage to the entire airway and lung parenchyma (Zikria,'72). More recent studies have further elucidated the effects of individual products of combustion, as well as the effects of breath-holding while in the fire, and the effects of toxins on breathing patterns (Cohen,'83).

This brings us to the present. Current burn management involves aggressive fluid resuscitation which has virtually eliminated hypovolaemia as a cause of burn death. The revised local treatment of burns, with early escharectomy, topical antibiotics and augmented skin cover has considerably improved the mortality in burns. Inhalation has now emerged as the major determinant of mortality in burns (Thompson,'86). Thompson found that burns mortality increased with age, skin surface burned, and the presence of inhalation.

1.2. *Epidemiology*

RCWMCH is a centre for secondary and tertiary care of patients under the age of 13 years. It receives referrals from throughout the Western Cape, a population at the time of the study of about 3 666 000 people (Bridgman,'92). The Cape Town metropolitan population was estimated at 3 101 000, of whom between 415 000 and 488 000 lived in "informal settlements" which are essentially shanty towns. In addition there was an ongoing process of urban influx of poor rural black people estimated at a rate of 5000 per month which had been taking place since the 1970's even before the abolition of influx control in 1986. These new arrivals were largely absorbed into the shanty towns.

The RCWMCH saw approximately 11 500 patients per year during this time in the trauma unit (Kibel,'90) of which 1 265 (11%) were burns. In many cases (43%) the severity of skin burns was 5% of the body surface area(BSA) or less, and only 9% had burns of greater than 20%BSA, therefore an overall of 37,5% are admitted. Thus in the 8 years of this study 3800 patients were admitted to the burns unit. A large percentage (68%) of admissions arose from hot water scalds and 21% from fires.

Electrification, or rather the lack of it, is a major factor in burns. While a well-developed infrastructure existed in the previously "white" and to a lesser extent "coloured" and "indian"

group areas, the "black local authority" areas had only minimal electrification consisting of street flood-lighting. This amounted to some 150 000 households which did not have access to electricity (Du Toit,'92). The less efficient alternative fuel sources shown in Table 1.1 all pose burn hazards to children. Coal or paraffin stoves, gas burners or open fires for cooking and inadequate bathing facilities all have inherent dangers.

Table 1.1 - Fuels used by Low Income Households in the Western Cape

Fuel	Percent of Households using fuel	Percent contribution to total energy consumption
Candles	37.8	0
Dry Cell Batteries	50.0	0
Car Batteries	22.8	0
Paraffin	92.2	41
Gas (LPG)	31.7	26
Wood	1.7	16
Electricity	17.8	16

(Du Toit,'92).

The type of housing is a major factor in the causes of burns. The corrugated iron shack of the informal settlements is easily set alight because the inside walls are lined with paper. Once the wooden supporting poles are burning the corrugated iron walls collapse inwards blocking the narrow doorway so that the occupants are trapped. The plastic sheeting used for water-proofing meanwhile produces noxious fumes.

Another factor identified by de Wet ('77) is overcrowding. This increases the chances of accidents. The average household size in "black" communities during the study period was 6,37 compared with 4,69 in "coloured" and 3,34 in "white" households (Du Toit,'92).

A further causative factor in South Africa is arson, used in both domestic and political disputes (Stone,'88). Home-made incendiary bombs containing petrol were used to flush

adults out of a house or shack, with little regard for the children also sheltering inside.

1.3 Causes of injury

There are several factors contributing to the the injury suffered by the child with inhalational burns, and the presentation depends on the exact combination of insults. They can be separated into heat damage - either wet or dry, chemical damage - irritant gases and particulates, and asphyxiant gases-carbon monoxide and hydrogen cyanide.

Heat damage The upper airways bear the brunt of the heat damage as they have a very efficient heat exchange system due to a large, well vascularised mucosal surface area. Heat is rapidly conducted away from the the airway and dissipates before reaching the trachea (Chu,'81). The heat source may be dry inhaled smoke or hot water (Dye,'90). Below the larynx heat injury is less significant as air has a low heat capacity and the volume of each breath is small (Clark,'88). In the trachea the blood supply is good and can transfer heat away rapidly. When the larynx is by-passed, the trachea can withstand dry heat up to 350°C (Moritz,'45) thus dry gases have to be extremely hot to produce any lower airway burns, and these are said to accur in less than 5% of fireburns (Pruitt, 1975). Further, protective reflexes such as laryngospasm and breath-holding come into play while the patient is still conscious (Trunkey,'78;Thoming,'82). However, steam can more easily cause damage below the the cords because it releases energy in the form of latent heat of vaporisation as it condenses on the surface (Mosley,'88). The lung parenchyma is particularly sensitive to steam and rapidly becomes congested and oedematous.

Chemical damage Almost all materials produce toxic gases when burning, and the types and amounts of toxins depend on the chemical structure of the burning substance and the conditions of the fire (temperature, time and oxygen supply) (Boettner,'69). **Table 1.2** (Prien,'88) outlines some of the substances commonly encountered.

Table 1.2 Toxic Products of Combustion of Common Household Substances

Material	Location	Decomposition Products
Cellulose	Wood, paper, cotton	Aldehydes, acrolein
Wool, Silk	Clothes, blankets, furniture	Hydrogen cyanide, ammonia, hydrogen sulphide
Rubber	Tyres	Sulphur dioxide, hydrogen sulphide
PVC	Upholstery, wall, floor and furniture coverings	Hydrogen chloride, phosgene
Plastics	Household goods	Benzene
Polyurethane	Insulation, upholstery	Hydrogen cyanide, isocyanates, ammonia, acylonitrates
Polyester	Clothes, fabric	Hydrogen chloride
Polypropylene	Upholstery, carpets	Acrolein
Polyacrylonitrile	Appliances, Engineering plastics	Hydrogen cyanide
Polyamide (nylon)	Carpetting, clothing	Hydrogen cyanide, ammonia
Melamine resin	Household and kitchenware	Hydrogen cyanide, ammonia, formaldehyde
Acrylics	Textiles, paint	Acrolein

(Prien & Traber, 1988).

Particulates in smoke consist mostly of carbon particles of varying sizes which are deposited at different levels of the airways. Thus, particles of 5μm or larger are deposited in the pharynx and larynx, particles 1-5μm land in the tracheobronchial tree, and particles <1μm reach the alveoli (Klaassen,'85). The carbon particles themselves are inert and cause no injury, however they are coated with irritant compounds from the fire such as aldehydes and hydrogen chloride (Prien,'88). In animal models it has been shown that bacterial filters which remove these particles from an inspiratory line prevent all the physiological abnormalities attributable to smoke inhalation (Nieman,'80). The breathing pattern of the patient while in the fire and reflexes of the airways further determine the distribution of particles in the airways (Thoming,'82). For example, diminished airflow resulting from breath-holding or reflex bronchospasm causes particles to sediment more proximally.

Irritant gases These are either water- or lipid-soluble. Water-soluble gases such as

ammonia and hydrogen chloride combine with water in the mucosal cells to form strong acids or alkalis which exert their damage at the mucosal level. Lipid-soluble gases such as aldehydes (acrolein) are able to penetrate further, and act more slowly (Prien,'88). Acrolein is the most toxic of the aldehyde gases causing death within a few minutes at a concentration of 10ppm (Zikria,'72).

In addition some compounds, such as nitrogen dioxide, are strong oxidising agents which cause damage by means of oxygen free radicals (Thomas,'67).

Asphyxiant gases The 2 major asphyxiant gases in smoke, which exert effects through their systemic absorption, are carbon monoxide (CO) and hydrogen cyanide (HCN). CO results from the incomplete combustion of organic substances, and HCN from the decomposition of nitrogen-containing polymers at very high temperature.

CO is an odourless gas which diffuses rapidly across the alveoli without causing any local damage. It binds competitively with the oxygen receptor of the haemoglobin molecule with a 250 times greater affinity than oxygen. It therefore impairs oxygen delivery to the tissues, an effect which it aggravates by causing a left shift of the oxygen dissociation curve (Pollard,'70). In addition it has a direct effect on cellular metabolism by binding competitively with the oxygen receptor of the cytochrome oxidase enzymes.

HCN poisoning seldom occurs without simultaneous CO poisoning and need only be considered when serum carboxyhaemoglobin levels exceed 15%(Birky,'81). HCN is a cellular toxin which binds with cytochrome C of the mitochondrial oxidative phosphorylation process. Thus, by inhibiting cellular respiration it acts synergistically with CO.

1.4 Pathophysiology

The first effect to be noticed is a 10 - 15 fold increase in blood-flow to the tracheobronchial tree (Herndon,'86). Beneath the soot there is mucosal oedema and scattered sub-mucosal haemorrhages (Chu,'81). Microscopic observations show mild subepithelial oedema with margination of polymorphonuclear cells 6 hours post injury (Thorning,'82). These polymorphonuclear cells release cationic proteases such as elastase, and oxygen free radicals (Traber,'86) which mediate an increase in alveolar capilliary permeability and interstitial oedema. The lung parenchyma is macroscopically congested and oedematous (Herndon,'87). Microscopically there is perivascular oedema and intact epithelial cells are beginning to separate from bronchial and bronchiolar mucosa. By 24 hours there is evidence of necrosis with sloughing of ciliated and secretory cells. Goblet cells increase in number and cause an outpouring of mucus in response to irritant gases (Bowden,'79). This combines with a loss of mucociliary clearance to seriously compromise the lung's defences. At alveolar level damage is less common with surfactant levels often remaining normal in the first week after injury (Head,'80). When it does occur the resulting pulmonary failure is usually lethal. Microscopically there is oedema and haemorrhage, with damage to type I pneumocytes and exposure of the alveolar basement membrane (Herndon,'86).

Between 12 and 72 hours (depending on the severity of the injury) sloughing of the tracheobronchial mucosa begins. The sloughed cells form pseudomembranes which may be expectorated as mucosal casts in cylindric or bifurcate tubes. Obstruction of large airways and narrow terminal airways results in a ball-valve effect with areas of emphysema and atelectasis, compounded by decreased surfactant activity. This pseudomembranous tracheobronchitis may heal at this stage or become secondarily colonised by bacteria, usually *Staphylococcus aureus* and various nosocomial gram negative organisms. Complications include major atelectasis, necrotising bronchiolitis and bronchopneumonia. The sloughed mucosa is replaced by granulation tissue which may become hypertrophic and obstructive. With healing the tracheobronchial mucosa becomes squamous, and there is fibrosis and narrowing. The alveolar basement membrane is covered with non-ciliated, non-secretory cells (Thorning,82). Fibrous pleural adhesions may remain, as well as areas of lobular emphysema and atelectasis.

1.5 *Classification of Inhalational burns*

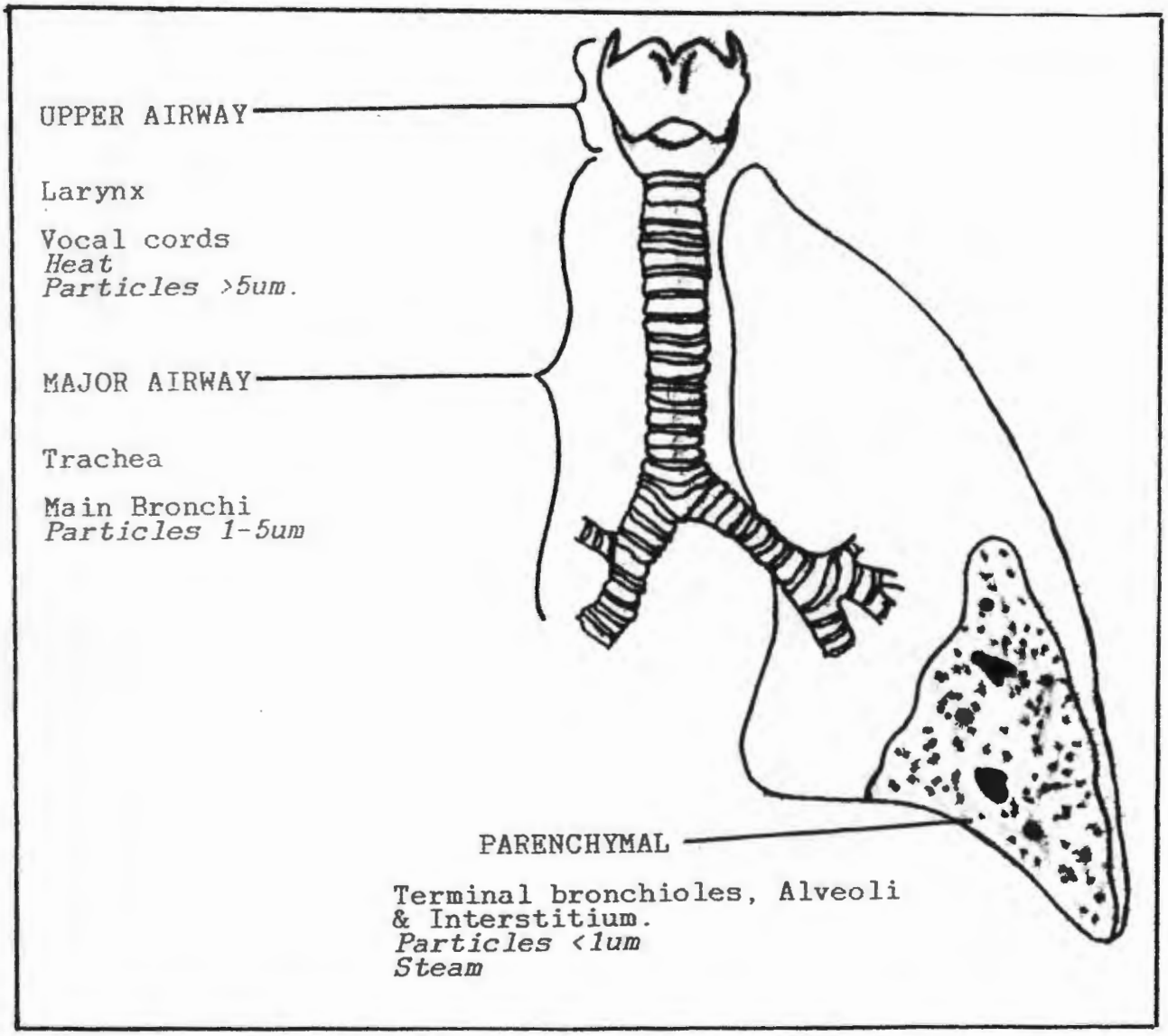
The original descriptions of presentations of the victims of the Coconut Grove fire of 1942 (Saffle,'93) are still clearly recognisable today. Other classifications have since been proposed. In 1969 Stone proposed a classification according to the time from injury to onsetof symptoms ie. Presentations within 24 hours were mainly due to pulmonary oedema; and after 48 hours due to pneumonia. However, this proved to be an oversimplification.

Moylan (1980) who did much of the pioneering work in this field proposed an anatomical classification which gave better explanations for the clinical presentations. Thus upper airway injuries involved the larynx and supraglottis, and presented with hoarseness and progressive stridor in the first 24 hours. Major airway damage involved the trachea from glottis to carina, the main and segmental bronchi and produced bronchospasm and bronchopneumonia on the second to fifth day. Injuries to the lung parenchyma itself were thought to be rare (less than 5% of the total) and presented with acute respiratory distress soon after injury and were uniformly fatal. However the advent of the 133Xenon lung scan to document parenchymal injury saw the incidence rising to 30% (Moylan,'72) and the possibility of silent parenchymal injury emerged.

A third type of classification took into account the cause of injury, namely thermal or chemical, which were then described as mild, moderate or severe (Chi-Shing Chu,'81).

All of the above classifications are precise but fall short of being clinically applicable because real patients usually have a combination of more than one injury. For example, a child caught in a fire may have thermal burns to the mouth and larynx, plus chemical burns to the trachea and bronchi, plus parenchymal burns to the lungs, plus systemic toxicity with CO. The time delay to onset of symptoms seems hardly relevant in such a patient as his or her initial symptoms may arise from several different sites and sources. Likewise the anatomical description, while providing useful information, does not explain how the various injuries combine and interact to affect the child's outcome. Added to this, the effects of shock, massive fluid infusion, sepsis and burn wound toxins also impact on the respiratory system.

Figure 1.1. Anatomical Classification of Inhalation Injuries showing the Agents Responsible for Damage at Each Level.



1.6 Clinical effects of Airway burns

Burns to the Upper Airways

These may be caused by heat and chemicals and may result from hot water or smoke inhalation. They manifest with upper airway obstruction due to oedema which begins soon after injury (Cudmore,'81). Clinically, time delays of up to 48 hours are documented. The patient develops progressive stridor and may require urgent intubation in the first 48 hours post-burn. This oedema then resolves but slough and bronchial casts can continue to threaten the airway for several weeks.

Burns to the Major Airways

The trachea is damaged by chemicals deposited on particulates, and therefore the only early warning sign is the presence of soot in the sputum or tracheal aspirate. The loss of mucociliary clearance means that the patient must rely on his cough mechanism to clear the airway (Moseley,'88). The pseudomembranous exudate acts as a culture medium for secondary infection, and the next clinical stage is bronchopneumonia occurring 2 to 5 days post-burn. When the basal cell layer is intact, healing is usually rapid and complete. However, when the basal layer is destroyed, healing is by secondary intention with prolonged granulation, cicatrisation and stenosis (Henning,'93). Granulation frequently blocks segmental bronchi causing persistent collapse of a lung segment, secondary bronchopneumonia and eventual bronchiectasis.

Burns to the Parenchyma

This injury manifests as progressive respiratory failure 12 -72 hours post-burn. The ongoing activity of toxin-coated smoke particles and secondary damage by substances released from alveolar macrophages explain this time delay (Herndon,'86). The presentation is therefore similar to that of adult respiratory distress syndrome with hypoxia and falling lung compliance in the early phase. Later, obstruction to the small airways by casts and pseudomembranes creates micro-atelectasis and hyperinflation with carbon dioxide retention. Bacterial infection then becomes superimposed on the congestion and atelectasis. Lobar consolidation on chest X-ray may become apparent at this stage, with

pyrexia and septicaemia clinically. Inhalation injury is known to increase the incidence of bacterial pneumonia in burn patients from 8 -38%(Shirani,'87).

The major clinical effects of CO poisoning are due to hypoxia of the heart and central nervous system (See **Table 1.3**). Post-mortem findings include a cherry red discolouration of the blood, petechiae in the brain and heart, and congestion of the kidneys (Finck,'66;Fisher,'69). Added to this is the fact that the fire quickly consumes oxygen in a closed space causing further hypoxia.

Table 1.3. Clinical effects of Carbon Monoxide Poisoning

CO Level (%)	Symptoms
< 20	Confusion, dyspnoeia
20-40	Irritability, Vomiting
40-60	Ataxia, Hallucinations, fatality
> 60	Uniformly fatal

(Cudmore & Vivori,'81).

1.7 Diagnosis of inhalational injury

Inhalational injury has a bewildering array of different presentations, from the brain-dead carbon monoxide victim to upper airway obstruction to the asymptomatic patient who later progresses to respiratory failure (Clark,'88). Early diagnosis of inhalation may be difficult in the absense of clinical signs of respiratory distress. Normal blood gases may exist in the presence of CO poisoning and the initial chest X-ray is usually normal (Lee,'88). Suspicion should be raised by the following features:

1. History of an enclosed space injury.

2. Carbon in the pharynx or sputum.

3. Peri-oral and/or oropharyngeal burns.

4. Singed nasal vibrissae.

5. Conjunctivitis.

6. Hoarseness.

7. Altered level of consciousness at any time following injury.

8. Ruddy complexion.

Occurring individually, each of these signs has a high incidence of false positivity, but taken together they have been found to underestimate the true incidence of inhalation injury (Robson,92).

Plain chest X-Ray shows no abnormalities in the early phase of parenchymal inhalation injury. A role has been postulated for computerised tomography to demonstrate the areas of non-segmental atelectasis (Clark,'82).

Flexible bronchoscopy is the only diagnostic tool for documenting major airway burns (Moylan,'80). The procedure is best done on arrival before the child has developed respiratory distress. It is performed under sedation and the narrow scope is passed through the cords without causing distress to the child (See **Figure 3.1**). In the trachea the presence of soot, spot burns, oedema and slough are all significant indicators of burns. The appearance of the mucosa to the experienced bronchoscopist correlates well with the pathological description of the epithelial damage (Masanes,'95). Biopsy specimens from different levels in the bronchial tree can be used to improve the accuracy of bronchoscopy (Masanes,94). The loss of the cough reflex is an indicator of the depth of laryngeal mucosal

injury. During the procedure the nature of secretions may indicate secondary bacterial infection and lavage and suctioning of soot and slough can be performed.

133 Xenon lung scan has been found to have a high degree of correlation with clinicopathological findings in inhalation injury to the lung parenchyma (Moylan,'72). 133xenon gas is injected intravenously and should be completely excreted by the lungs within 90 seconds. Delayed clearance or segmental retention of the isotope is indicative of inhalation injury. These changes may revert to normal by day 4, so the scan should be done within 72 hours. The method has the advantage of detecting asymptomatic injury and not requiring any patient cooperation. Inaccuracies can arise from hyperventilation or pre-existing lung disease.

Serum immunoreactive calcitonin has recently been reported to act as a marker for the presence of inhalational injury in burns and to positively correlate with mortality (O'Neill,'92). The source of the hormone is the pulmonary neuroendocrine cells situated in the bronchial epithelium. These are thought to act as paracrine chemosensors that monitor the composition of inhaled gases. Hyperplasia of these cells occurs in cigarette smokers, for example. In smoke inhalation a massive paracrine discharge from the cells occurs and procalcitonin, a larger form of calcitonin is released (Nylen,'92). This polypeptide may in the future become a useful clinical marker for the presence and severity of inhalational injury.

Direct carboxyhaemoglobin measurements require a mass spectrometer or co-oximeter. If unavailable a reduction in mixed venous oxygen tension is an indicator of the toxin. Arterial oxygen tension remains near normal despite severe tissue hypoxia and is notoriously unreliable. It is essential to take the blood sample early, as it dissociates from haemoglobin with a half-life of 249-320 minutes in room air. This rate increases to only 38-80 minutes if 100% oxygen is inspired, and 15-23 minutes with 3 Atm of hyperbaric oxygen.

Diagnosis of HCN poisoning is difficult as gas chromatography is only available in a few toxicology centres in South Africa. A blood cyanide level of >0.2mg/l is abnormal (Vogel,'81).

1.8 Management of inhalational injury

Treatment of the external burn wounds involve fluid resuscitation and early aggressive removal of burn eschar. This is beyond the scope of this topic, except to stress that optimal wound care removes bacteraemia as a source of haematogenous pneumonia. Treatment of inhalation is based on the initial clinical presentation, extent and progress of the disease and complications. If the upper airway is injured the patient develops progressive stridor, and may require urgent intubation in the first 48 hours post-burn. Nasotracheal intubation is favoured in the first instance due to the transient nature of the laryngeal oedema, the frequent coexistence of neck burns and the avoidance of an anaesthetic (Cudmore,'81, Robson,'92). However, if the patient must remain intubated for more than a few days tracheostomy provides easier bronchial toilet, ease of feeding and greater patient comfort (Calhoun,'88). It also becomes necessary where there is severe injury at the level of the larynx (Mellins,'75).

In tracheal burns the main aim of management is to minimise the risk of secondary pneumonias. Nebulisers, frequent suctioning and physiotherapy play an important role in clearing tracheobronchial casts. A role for heparin infusion in decreasing cast formation and improving ventilation has recently been shown in a sheep model (Cox,'93). The mechanism for this is unknown and may be unrelated to its anticoagulant properties. Repeated therapeutic bronchoscopy is also recommended to physically remove slough and secretions. Prophylactic antibiotics are less useful than specific antibiotics according to sputum cultures (Stone,'67). The management of late airway obstruction by granulation and fibrosis is exceedingly difficult. While laser treatment can shrink granulation in the short-term it quickly recurs. Acceptable results for subglottic stenoses are claimed with laryngotracheoplasty (Calhoun,'88). Definitive surgery for tracheal stenoses should be delayed for at least 2 years, as the trachea in burn victims retains a capacity for hypertrophic scar formation which causes recurrent stenoses at the anastomoses (Henning,'93). Before 2 years, tracheal stenoses are best managed with a tracheal t-tube which stents the lesion. Injuries involving the carina and main bronchi are most difficult to treat, as frequent dilatations are necessary. A T-Y-tube to stent the bifurcation is an option. Replacement of the trachea with a silicone prosthesis is also an option in the long-term.

Parenchymal injury usually remains undiagnosed until the patient develops respiratory distress, needing ventilatory support. They may then manifest a pattern of falling oxygen

saturations and tidal volumes, and require increasing pressure support. Children are particularly susceptible to barotrauma. The attendant mortality of this complication can be reduced by the use of prophylactic high frequency ventilation(HFV)(Cioffi,'89).This ventilation mode maintains oxygenation at the lowest possible peak airway pressure and fractional oxygen concentration. There is a less clear advantage of HFV when used to salvage patients in whom conventional ventilatory support has failed or barotrauma has already occurred (Cioffi,'93). It is therefore postulated that the survival advantage achieved with HFV is mainly due to a decrease in iatrogenic damage. Other studies also suggest an improvement in the clearance of secretions with HFV (Hachtenberg,'87; Freitag,'89; Thangaturai,'88).

Parenchymal injury requires very careful fluid management. The presence of inhalation has been shown to increase fluid requirements by approximately 40% in several studies (O'Neill,82;Scheulen,82;Navar, 85). However, a positive fluid balance aggravates the interstitial oedema and further decreases lung compliance. To counteract this effect the use of hypertonic saline has been advocated (Caldwell,'79) and the use of positive pressure ventilation and positive end expiratory pressure is essential (Chu,'81). A role for heparin has also been postulated as a means of decreasing pulmonary interstitial oedema (Cox,'93). The rationale is that heparin is an anionic compound which opposes the effects of leucocyte-mediated cationic proteases. It therefore limits the changes in microvascular permeability and limits pulmonary oedema. A continuous infusion of heparin commenced 30 minutes after injury has been shown in a sheep model to reduce pulmonary oedema. The result is improved oxygenation and reduced barotrauma. Histologically, reduced tracheobronchial cast formation is also documented.

After 72 hours the major aim of treatment is to prevent secondary bronchopneumonia. This life-threatening complication may follow burns to any part of the airway due to soot, slough and casts in the airways and to atelectasis. Burn management in the form of intubation, massive transfusion, endotracheal intubation and prolonged anaesthesia further increase the risk of pneumonia. Added to this is the presence of often septic skin burns and the general immuneparesis associated with burns. It is recommended that antibiotics be reserved for suspected infections with bronchoalveolar lavage to obtain specimens for microbiology (Moylan,'78), however it is exceedingly difficult to prevent the use of

prophylactic antibiotics in practice. Adrenal corticosteroids are no longer used in the initial management of parenchymal injury because of the increased risk and severity of secondary pneumonias (Stone,'67; Moylan&Chan,'78). Endotracheal suction, lavage and physiotherapy are essential to mobilise foreign material and necrotic debris from the airways. Frequent therapeutic bronchoscopy can also help in this regard.

Hyperbaric oxygen is the treatment of choice in serious CO poisoning as it rapidly reverses tissue hypoxia and can reduce cerebral oedema (Neubauer,'88).However, it requires a specialised pressurised chamber which produces problems of monitoring the patient while inside. The management of less severe cases depends on oxygen therapy, and the patient should be intubated if severely hypoxic or unconscious so that an inspired oxygen tension of 100% can be achieved (Chu,'81).

HCN poisoning has 3 specific antidotes, however 2 of them, amyl nitrite and cobalt edetate, are fairly toxic themselves (McKiernan,'80;Krantz,'40). The third antidote, sodium thiosulphate has a low toxicity, but is fairly slow-acting (Friedberg,'69). Sodium thiosulphate should be given slowly intravenously if cyanide poisoning has been confirmed. Since treatment must be commenced before toxicology results can be obtained, the ancillary measure of fluid resuscitation and volume diuresis against a high central venous pressure is wise (Chu,'81).

1.9 Mortality

In fire victims carbon monoxide is the early cause of death in 50-60% of cases with burns accounting for only 10-20% of deaths (Clark,'88). With the improvement in fluid resuscitation and wound management the mortality of burns has decreased. However, respiratory complications occur in 18-30% of all burn patients and are a major contributor to mortality (Philips,'62;Achauer,'73; Marshall,'83). The presence of inhalation significantly increases mortality (Thompson,'86) and this mortality increases with age. Thompson found the 5-14 year age-group to have the lowest mortality. This he attributed to fitness, and the absence of smoking, alcohol and drug abuse in this age group. Generaliy, the likelihood of inhalation increases with area of skin burns, so it is postulated that the mortality statistics for burn size and inhalation are more than additive, with the presence of inhalation increasing mortality in every burn size category (Thompson,'86;Shirani,'87). Thus, the presence of inhalation and pneumonia are added to age and burn size as independant comorbid factors. This fact has little predictive value, however, since the impact of inhalation is multifactorial, with some of the effects being fully reversible with correct treatment eg. CO poisoning. Comparison of mortality between the early and late 1980's shows an improvement in mortality most marked in the milder injuries. This is attributed to early accurate diagnosis, improved ventilatory support, and prevention of pneumonias (Rue,'93).

2. METHODS

2.1 Study Aims

The prime aim of this study is to increase awareness of the lethal combination of injuries that occur with inhalational burns. It is a condition that the writer feels is routinely underestimated, and a problem that must be addressed if we are to entertain hopes of salvaging major burns in children. It is hoped that this pilot study will generate further research in the field, and lead to a management protocol that can be followed by emergency and junior staff. This study reviews the case notes and post-mortem findings of 86 children admitted to the RCWMCH between January 1987 and June 1995 with inhalational burns.

2.2 Identification and Documentation of Cases

The presence of inhalation was identified by the following -:

History

History of exposure to heat, steam, smoke or toxic gases. The exact circumstances of the injury are significant, Eg if the child was attempting to drink a hot liquid, or was trapped in a closed space with a fire or smoke.

Presentation

Presentation of facial burns, singed nasal hairs and intra-oral burns. Clinical signs of inhalation include hoarseness of the voice, respiratory distress and abnormal chest auscultatory findings. Carbonaceous sputum, a ruddy complexion and altered level of consciousness are relevant to fire-burns.

Baseline investigations.

Early abnormalities of blood oxygenation or chest X-ray are also significant but are non-

specific.

Specific Investigations

Specific Investigations include flexible bronchoscopy and blood carbon monoxide levels. Xenon lung scan and bronchoscopic biopsy were infrequently used at the time of this study.

An overview of the 86 cases is presented in table form. (See **Table 3.1**).Particular attention is paid to the following-:

- **Cause of burns** - the 86 patients in this study separated into 67 fireburns and 19 hot water scalds.

- **Percentage body surface area (%BSA) burned** - this ranged from 0% in patients trapped in a smoke-filled room, to 100% fireburns in which there was no hope of survival. In such cases there was often no active management of the inhalational component diagnosed only at post-mortem.

- **Presentation of inhalational burns** - from which the anatomical site of injury was determined.

- **Investigations** - the value and accuracy of investigations done is assessed in the light of subsequent complications and compared with post-mortem findings in those who died.

- **Management** - is critically reviewed with particular reference to the prevention of complications such as airway obstruction, barotrauma and secondary pneumonia. Recognition is given to the fact that many children with massive burn injuries were not actively resuscitated, or active management was abandoned at a point where death became inevitable.

- **Outcome** - Complications are analysed with a view to future prevention. Our mortality

data are compared with those of other centres.

From this information a classification is proposed based on the anatomical descriptions of Moylan ('80). Thus fireburns are divided according to the anatomical area affected viz. upper airways, major airways and lung parenchyma, See **Figure 1.1.**

- **Upper airway burns** - These involve the pharynx, hypopharynx or the larynx as far as the vocal chords. The presenting signs are of upper airway obstruction in the first 48 hours with stertor (noisy breathing), stridor or hoarse voice. Flexible bronchoscopy shows erythema and oedema of the mucosa, with soot, ulceration and slough in the more severe fireburns.

- **Major airway burns** - These involve the trachea from subglottis to carina, the main and segmental bronchii. The presentation is in the context of severe fireburns but the tracheal injury per se is silent in the first 72 hours. Thereafter it causes secondary bronchopneumonia as the sloughed mucosa becomes colonised with bacteria. Diagnosis is again by flexible bronchoscopy which shows soot deposition, erythema, ulceration and slough in the trachea. The injury is also evident at post-mortem.

- **Lung parenchymal burns** - These involve the broncho-alveolar respiratory epithelium itself. The clinical findings are of respiratory distress and respiratory failure occurring soon after injury in the context of severe fireburns. Chest X-Ray is initially normal but may progress to perihilar consolidation or the classical "white-out". The only definitive investigation is the Xenon lung scan which shows retention of the radio isotope in the affected lung. Post-mortem the lungs are pale and hyperinflated in the first 6 hours but become heavy and congested with interstitial oedema and haemorrhage later.

Systemic toxicity is discussed separately. Hot water burns, having no previously documented classification are divided into those affecting upper airways only and those where aspiration of hot water or steam into the trachea or lung parenchyma could be documented.

2.3 Study Limitations

The data collected for this study is retrospective, and subject therefore to the limitations of observer bias, incomplete documentation and variable management plans. Hospital notes were made by doctors with very different levels of experience. Initial assessment by junior staff often omitted examination of the mouth, blood gases and chest X-rays for instance. While nursing staff took routine burn swabs, there was no routine sputum collection so retrospective diagnosis of secondary pneumonias becomes unreliable.

The study did not directly influence management of inhalation during the time period. However, awareness of the entity did increase in the latter years resulting in improved documentation of injury. The study therefore underestimates the incidence of inhalation in the earlier period. The basics of a management protocol have emerged from this data but have yet to be formally implemented. This lack of a diagnostic and management protocol limited the data since investigations were often incomplete and suspected inhalation could not be confirmed or excluded in some cases.

Post-mortem results proved a useful adjunct to retrospective diagnosis, however limitations arose from a difference in emphasis. Medicolegal autopsies are performed in terms of the Inquest Act No. 58 of 1959 on all deaths from non-natural causes, which therefore include all burns deaths. Forensic investigations designed to identify foul-play were less specific in documenting exact sites of any airway damage. Carbon monoxide levels were only determined in rapidly fatal cases. Histology of congested lungs was done in very few cases, and macroscopic descriptions such as bronchopneumonia had to suffice as confirmation of lung parenchymal involvement. This would then have to be correlated with the antemortem clinical findings and time of death to establish the source of the bronchopneumonia.

3. RESULTS

An overview of the cases studied and their outcomes is presented in **Appendix 1**. The 86 cases of inhalational burns in this study can be divided by cause into fireburns and hot water scalds. The numbers involved are shown in **Table 3.1**.

Table 3.1. Causes of 86 Inhalational burns.

FIREBURNS	Number of cases
Trapped in burning house	19
Trapped in burning shack	18
Tent on fire	2
Caravan on fire	2
Accident with paraffin stove	8
Accident with paraffin lamp	1
Paraffin poured onto open fire	3
Accident with coal stove	1
Overturned candle set bed alight	3
Fell into an open fire	3
Trapped in burning car	4
Electrocution and clothes set alight	1
Playing with matches	1
Accident with Petrol	1
TOTAL	67
HOT WATER SCALDS	**Number of cases**
Spilt or fell into hot water	12
Tried to drink hot drink	7
TOTAL	19

3.1 Fire Burns to the Upper Airways

54 children sustained burns of the larynx and pharynx due to fire and 18 due to hot water. The hot water injuries are considered separately.

Table 3.2. Presenting signs in Children with Upper Airway fireburns

Presentation	Number of cases
Stridor	22
Respiratory Distress	17
Asymptomatic	6
Hoarse Voice	3
Stertor (noisy breathing)	3
Unconsciousness	3
TOTAL	54

Presentation was as gradual onset of stridor in 22 cases (See **Table 3.2**), with time delays ranging from 2 to 48 hours. The diagnosis was therefore usually delayed until stridor was recognised. 17 cases presented with predominant signs of their parenchymal injury, 3 had decreased level of consciousness and 6 had no specific respiratory signs.

In 43 cases the injury was demonstrated by flexible bronchoscopy (See **Figure 3.1**). This typically showed oedema, erythema, soot and slough on the epiglottis, supraglottis and vocal cords. Other means of making the diagnosis were direct laryngoscopy just prior to intubation (8 cases) and post-mortem (5 cases). 24 cases had flexible bronchoscopy on admission, while the remainder had bronchoscopy only when they became symptomatic, a time delay ranging from 5 - 48 hours. This usually occurred when the patient arrived late at night, or the possibility of inhalation was not realised on admission. 6 patients were only scoped later because they were intubated urgently.

Bronchoscopy proved misleading in 1 patient (case No.16) where the initial bronchoscopy

noted only an abnormally shaped subglottis from previous croup. The child was therefore not given any special treatment, and repeat bronchoscopy was done on day 9 when a chest infection supervened. At this procedure she was found to have both glottic oedema and a tracheitis secondary to the burn. She then succumbed to pneumonia. In case No.42 bronchoscopy done on admission was misinterpreted. She was reported to have carbon on the vocal cords but no oedema or compromise of the airway. She was therefore not intubated until she had a respiratory arrest at 48 hours. Post-mortem reported a markedly swollen larynx, plus a congested, swollen trachea.

Direct laryngoscopy, which was used in 8 cases, was correct in identifying 5 laryngeal burns, later confirmed at post-mortem. However, it described normal cords in Case 59 who later died and had laryngeal burns at post-mortem. Laryngomalacia was reported in Case 62 who progressed to recurrent chest infections and died after 40 days. At this stage laryngeal injury could not be confirmed by post-mortem.

8 children required intubation purely for the airway obstruction, and 20 were intubated for ventilation purposes. All cases were intubated nasally at first, and 4 cases later required tracheostomy. The indications for tracheostomy were severe laryngeal burns in all cases. 3 were done electively because a prolonged course was anticipated and 1 was done as an emergency (Case No 23).

Residual laryngeal damage was demonstrable in 4 children, all of whom had severe burns with healing by granulation, and 3 of whom had required tracheostomy. Damage was evidenced by subglottic stenosis in 1 case, impaired sensation with recurrent aspiration pneumonias in 2 cases, and complete stenosis at the level of the cords in 1 case.

Table 3.3. Causes of death in patients with upper airway fireburns.

Cause	Number of cases
Airway obstruction	1
Secondary Pneumonia	3
Severe parenchymal injury	7
Overwhelming skin burns	11
Late burn sepsis	3
Septic tracheal replacement	1
TOTAL	26

Table 3.3. shows that almost all deaths in this group were unrelated to the upper airway injury. Case No 23. died after suffering hypoxic brain damage, and this was the only death attributable to the upper airway injury itself. His larynx appeared ulcerated at extubation but the airway was widely patent. The subsequent obstruction was therefore probably caused by more slough and fibrin. Emergency tracheostomy was then done, but not before the child had sustained hypoxic brain damage.

The other causes of death are listed in **Table 3.3** and show the severity of burns which are the usual context of lethal upper airway injury.

3.2 *Fireburns to Major Airways*

There were 28 confirmed cases with major airway damage. In 3 other cases (No.s 4,37 and 43) it could be suspected in retrospect, but the necessary investigations were not done to confirm it.

Table 3.4. Presenting Symptoms of 28 Major Airway Burns.

Symptom	Number of cases
Respiratory Distress only	14
Stridor only	4
Respiratory Distress and Stridor	3
Asymptomatic	7

Presenting symptoms tended to reflect the co-existing upper and parenchymal injuries with stridor or hoarseness in 7 cases and respiratory distress in 17 cases, while 7 had no respiratory signs. These cases presented later with signs and chest X-ray findings of secondary pneumonia.

Injury was confirmed by bronchoscopy in 13 cases, post-mortem in 11 cases, and both in 6 cases. In cases confirmed by both bronchoscopy and post-mortem, the progression of the injury could be documented. In 3 cases bronchoscopy was inadequate as the trachea could not be seen, and in 1 case bronchoscopy and post-mortem findings conflicted (Case No.46) Bronchoscopy findings included reddening and oedema in 5 cases and soot in 10 cases. Repeat bronchoscopy showed progression of soot-lining to spot burns and ulceration after 6 days in Case 26, and to ulceration and slough then to hypertrophic granulation and narrowing of the bronchi in 3 others (Case No.s 35,65 and 78).

Management was largely influenced by the concomitant skin, upper airway and parenchymal burns. 6 patients had overwhelming skin burns and were not managed

actively (Case 3, 41, 52, 67, 70, 75).

14 patients were intubated and ventilated for parenchymal injuries, although in 7 of these there were large skin burns and no realistic chance of survival. 2 of these children survived, Case 28 with mild tracheal burns, and Case 35 with isolated subglottic granulations.

7 cases did not have associated parenchymal burns, and their upper airway burns did not obstruct sufficiently to warrant early intubation (Case No's 16,42,56,57,65,71 and 82). None were intubated initially and only Case 71 had an uneventful recovery. 5 developed secondary pneumonias (Case No.s 16,42,56,65 and 82), 2 of which were fatal (Case 16, 42). The seventh died later of wound sepsis (Case 57).

2 cases required tracheostomy for prolonged major airway damage (Case 65, 78). One survived a prolonged hospital course with recurrent secondary pneumonias and bronchial narrowing and was successfully extubated after 46 days. Case 78 was not extubatable because of complete laryngeal stenosis, and suffered recurrent obstruction of the trachea and main bronchi by hypertrophic granulation tissue (See **figure 3.2**). Numerous bronchoscopic dilatation's were done, however neither endoscopic removal nor laser removal of granulation produced lasting relief of obstruction. This problem continued in the main bronchi after placement of a silicone tracheal replacement at 11 months post-burn. His sudden death 3 months later was caused by a fungal infection of the graft with erosion into the aorta (See **Figure 3.3**).

17 of the patients in this group died within 48-72 hours, before secondary pneumonias could develop. 7 of the remaining 11 cases developed secondary bronchopneumonias as evidenced by high temperatures, respiratory distress or new opacities on chest X-ray and 4 of these died. 4 patients did not develop secondary pneumonias (Case 8,26,28 and 71).

These 4 had the advantage of only minor skin burns (2-20% BSA burned) and their major airway injuries were documented by early bronchoscopy. Actual bronchoscopic features were no less serious than the others however.

3.3 Fireburns to Lung Parenchyma

38 patients with fire burns were found to have evidence of parenchymal injury. 27 cases presented with early respiratory distress on or soon after arrival (See **Table 3.5**). 6 had stridor (the only sign in 3), and 2 had signs of hyperinflation with chest wheezes. 3 had a respiratory arrest on admission or in the ambulance.

Table 3.5. Presenting signs in 38 parenchymal injuries.

Presenting Signs	Number of cases
Respiratory Distress	27
Stridor	6
Air-trapping	2
Respiratory Arrest	3

14 patients had abnormalities on initial Chest X-Ray; 3 of these had the classical "white-out" or diffuse consolidation (Case No.s 15, 59 and 78) See **Figure 3.4,** and none of the other X-ray signs could be described as diagnostic (See **Table 3.6**).

Table 3.6. 27 Initial Chest X-Ray findings in Parenchymal Burns.

Chest X-Ray finding	Number of cases
Normal	10
Perihilar consolidation	5
Lobar consolidation	6
Hyperinflation	3
Diffuse consolidation	3

One patient had confirmation of the parenchymal injury with a Xenon lung scan 24 hours

post-burn (Case 44). This showed delayed washout of isotope from the left lung (See **Figure 3.5 and 3.6**).

18 patients were ventilated with a hope of cure, and 8 of these demonstrated a pattern of decreasing lung compliance, requiring increasing ventilatory support. The high pressures required caused barotrauma in 4 cases; a pneumoperitoneum and extensive surgical emphysema in Case 59 (See **Figure 3.7**), a pneumomediastinum in Case 28, surgical emphysema only in Case 19 and a pneumothorax in Case 78. Case 28, presenting in 1993, was then put onto high frequency ventilation which prevented further barotrauma, and she survived the injury.

Only 8 patients survived in total. 2 had mild parenchymal injury not requiring ventilation (Case 8, 83). 5 were ventilated with normal pressure support (Case 15, 19, 35, 36, 55) and one required high frequency ventilation (Case 28).

3.4 Bacteriology

Relatively few positive sputum cultures were obtained due to widespread use of prophylactic antibiotics. Positive blood cultures were not regarded as proof of a pneumonia because of the potential skin surface burn source. Positive sputum cultures were obtained from 5 patients only and included 3 *Pseudomonas aeruginosa*,3 *Staphylococcus aureus, 2 Streptococcus pneumoniae, 2 Klebsiella pneumoniae, 2 Acinetobacter anitratus and 2 Haemophilus influenzae.* All cases were treated with intravenous antibiotics, and most children recovered without further chest infections once the skin burns had healed.

3.5 Mortality

Of the 30 deaths, 4 patients had arrived *in extremis* and died in the emergency room (Case 7, 32, 43, 68). 5 patients were managed actively and ventilated, but died between 2 and 40 days post-burn with post-mortem signs of parenchymal injury (Case 11, 25, 53, 59, 62). 8 patients were ventilated initially despite large skin burns and active treatment withdrawn after deterioration (Case 9, 10, 14, 31, 38, 45, 61, 66). 9 patients had extensive skin burns and were not treated actively for the inhalational injury (Case 3, 34, 37, 46, 48, 51, 75, 76,

79). One case had no documented symptoms until a respiratory arrest 40 hours post-burn and post-mortem findings of parenchymal inhalation (Case 12). 2 did not require ventilation but died from secondary infections after 12 and 18 days (Cases 44, 81). The last case survived his parenchymal injury, but died over a year later due to septic complications of tracheal replacement (Case 78).

3.6 Secondary Bronchopneumonia

20 cases of secondary pneumonia were isolated from the fireburn group using the criteria discussed above, viz. fever and respiratory signs more than 48 hours post injury, focal consolidation on chest X-ray and positive sputum cultures of single organisms. These cases were then analysed with respect to site of associated airway burn, specific predisposing factors, organisms cultured and outcome.

Table 3.7 Anatomical sites of the associated inhalational burns

	Upper	Major	Parenchymal
Number of cases	19	7	8

It would appear from **table 3.7** that the upper airway burns are disproportionately represented in the development of secondary pneumonia, but this finding must be interpreted in light of the fact that 69% of the parenchymal burn cases died within 72 hours, before any secondary pneumonia could manifest. The real incidence of secondary pneumonias is therefore better reflected in the revised incidence column of **table 3.8.**

Table 3.8 Number of fireburn cases with secondary pneumonias out of cases surviving more than 72 hours.

Anatomical site	Total number	No. surviving >72 hours	No. of secondary pneumonias	Revised Incidence
Upper	54	38	19	50%
Major	27	13	7	54%
Parenchymal	37	12	8	66%

Table 3.8 shows that the incidence of secondary pneumonias in survivors beyond 72 hours increases slightly with more distal injury to the respiratory tree.

Table 3.9 Incidence of Secondary Pneumonias in 19 Isolated Upper Airway Burns.

Anatomical site	Total number	Subsequent Pneumonia
Intubation for Stridor	8	3
Not Intubated	11	4

Table 3.9 shows the incidence of secondary pneumonias in 19 patients who had isolated upper airway injuries with no tracheal or parenchymal damage. Secondary pneumonia was diagnosed by clinical features of fever, productive cough and chest signs, or localised consolidation on chest X-ray as sputum cultures were not routinely taken. 8 were intubated and 11 were not. 3 of the intubated cases developed secondary pneumonias, 1 of which was fatal. However, 4 of the patients who were not intubated also developed non-fatal secondary pneumonias.

Specific predisposing factors were identified in 6 cases, as follows in **Table 3.10**.

Table 3.10 Specific Predisposing Factors to Chest Infection in 6 Patients.

Factor	No. of Cases
Tracheostomy	3
Laryngeal incompetence	2
Emergency Intubation	1
Untreated Tracheal Injury	1

One patient had a tracheostomy for the duration of his 14 month hospital stay, and a second had a tracheostomy only after 2 failed extubations. 2 patients (the third tracheostomy patient and one other) had decreased laryngeal sensation after severe upper airway burns, with impaired cough reflexes and evidence of recurrent aspiration. The fifth had a hypovolaemic arrest, with emergency intubation and resuscitation again raising the possibility of aspiration. The sixth had a major airway burn which was initially missed on bronchoscopy and only diagnosed on day 9 after developing pneumonia.

3.7 Long Term Sequelae

Long term follow-up was possible in very few cases, usually only those with defined respiratory problems or skin burns requiring prolonged treatment. Therefore of the 15 patients surviving hot water burns there is no ongoing record. Of the 30 patients with fireburns surviving beyond 3 months, 20 have no long term follow-up, 6 have follow-up beyond 3 months with no sequelae of inhalation, although one was found to have pulmonary tuberculosis (Case 5). These 6 patients all had prolonged in hospital care due to septic skin burns (Case 5, 24, 55, 56, 73, 82). One patient had persistent left lower lobe collapse on chest X-ray 4 months after upper and parenchymal airway burns (Case 36).

Two patients had residual laryngeal incompetence following upper airway damage (Case 35, 65). In Case 35 this was manifested by recurrent chest infections and a pectus excavatum chest deformity. In Case 65 it was manifested by recurrent chest infections with fat-laden macrophages on sputum cytology. One patient had residual laryngeal, tracheal and bronchial damage (Case 78). He had a permanent tracheostomy and died after sepsis

of a tracheal implant 13 months post burn.

3.8 Systemic Toxicity due to Fire Burns

Evidence of systemic toxicity could be found in 25 of the 67 fireburns. However this is an underestimate of the incidence as blood tests were not routinely done especially in the earlier years of the study. Post-mortem findings were only present in cases who died soon after injury, so many cases may have been missed. **Table 3.11** lists the incidence of positive findings.

Table 3.11 Clinical, Laboratory and Forensic Evidence of Carbon Monoxide Poisoning in 25 cases.

Evidence of CO poisoning	No. of Cases
Comatose on admission	7
Seizures	2
Tachypnoeic	8
Disorientated	4
Ruddy Complexion	2
Cyanosis	2
Elevated COHb level	16
Post-Mortem:	
Pink discolouration of blood and tissues	4
Cerebral oedema	7
Sub-pleural petechial haemorrhages	5

3.9 Hot Water Burns to the Airways

19 Children sustained injuries to the airways after hot water scalds. These were the younger age group (mean age 18 months) and there were no skin burns greater than 30% BSA. The injury tended to occur either after hot water fell off a table or the child attempted to drink a hot beverage. 18 of them involved the upper airway, 1 the trachea and 12 had additional lung involvement.

Table 3.12 shows the presenting symptoms and signs of these cases. The majority of patients presented with increasing stridor first observed 2 - 48 hours after injury. Signs of respiratory distress were also common however. Fits occurred as a late sign of hypoxia in 2 patients, and hyponatraemia in 1 case.

Table 3.12 Presenting Clinical signs in 19 patients with Hot Water burns of the Airways.

Clinical Signs	No. of Cases
Increasing Stridor	14
Tachypnoeia	6
Recession	4
Chest crackles	6
Copious Sputum	4
Fit	3

Flexible bronchoscopy was done in 12 cases, at the onset of stridor. Oedema and hyperaemia of the pharynx, supraglottis and cords were the commonest findings, however 5 cases also had ulceration and fibrin formation. 2 cases had direct laryngoscopy only, and the supraglottic oedema was adequately documented. 3 cases had no visualisation of the larynx and these included 1 fatality (Case No 72).

9 children settled on adrenaline nebulisers and headbox oxygen, without requiring intubation. One of these developed a mild bronchopneumonia with fever and chest crackles coinciding with initial symptoms. 9 children required intubation, 2 with tracheostomies, and 2

also required ventilatory support for respiratory distress and hypoxia.

Case No 72 who presented in 1992 with tachypnoeia and chest crackles had an unexpected respiratory arrest 40 hours post-burn. He was not intubated at the time, but had copious secretions in his mouth and had a seizure shortly before arresting. He had congestion of the trachea and bronchi at post-mortem.

11 patients had signs of pneumonia as shown by chest X-ray opacities (see **Table 3.13** and **Figure 3.8**) and one had bilateral bronchopneumonia at post-mortem (Case 72). 10 of the X-rays were taken at the onset of stridor ie. within 48 hours of the burn injury and one on Day 6 (Case 58).

Table 3.13 Chest X-ray opacities in 10 patients with lung parenchymal involvement.

Chest X-ray	No. of Cases
Opacities	8
Bilateral perihilar opacities	2
Left sided opacities	1

6 of these patients had clinical signs of lower airway respiratory distress with tachypnoeia, recession and chest crackles as well as stridor Case 17, 29, 47, 58, 72, 80). 3 positive sputum cultures were obtained, one with *Streptococcus pneumoniae*, and 2 with *Haemophilus influenzae* (Case 29, 30). 2 children had tracheostomies done on admission to secure the airway (Case 18, 40), the latter through neck burns. This child developed bilateral pneumonia, and Case 18 had a right upper lobe collapse on admission. Both cases were ventilated as well, yet extubation of the tracheostomy was possible within 4 days. 2 other cases required ventilation for ongoing pneumonias (Cases 69, 85) and 4 settled without ventilation (Case 17, 29, 30, 54).

4 of the patients with lower respiratory complications died (Case 47, 58, 72, 80) The only anticipated death was that of Case 58 from a severe *Klebsiella pneumoniae* pneumonia and tetraplegic cerebral palsy 12 days post-burn. Post-mortem findings in the lungs were

described as congestion with early pneumonitis on histology.

3.10 Post-mortem Findings

All 42 burns underwent medicolegal autopsies at the Salt River Medicolegal Laboratory. Post-mortem findings varied according to how long the child had survived post-burn. Early findings included hyperinflation (Case No.7), congestion and oedema (Case No.s 32,43,46,66,68,75,76 and 79). Histology in case 43 showed diffuse acute pneumonitis. In the next 24 hours congestion and atelectasis became more prominent (Case No.s 3,12,37 and 61) with a macroscopic appearance reported in some as adult respiratory distress syndrome (case No.s 38,45 and 51). Subpleural petechial haemorrhages were reported in several cases at this stage (Case No.s 38,45 and 61). From 36 hours onwards inflammatory response, epithelial desquamation and formation of pseudomembranous exudate was first noted (Case No. 11,38,59 and 62). In the deaths occurring after 48 hours the consolidation was more focal, usually basal and was described as bronchopneumonia (Case No.s 9,6,14,25,44,62 and 81). There was extensive intra-alveolar bleeding in Case No. 34 and haemorrhagic areas with atelectasis in case No 31 who died on day 15. There was one surprising post-mortem finding of a purplish grey exudate and foul-smelling pleural effusion in case No.48 who died only 6 hours post-burn. Parenchymal sections of the lung of case No.78 who died 13 months post-burn were suggestive of bronchiectasis with dilated bronchial lumens and mucopurulent plugs; in addition there was chronic inflammatory infiltrate, cytomegalovirus inclusion bodies, and a focus of necrotising bronchitis in the right lower lobe.

3.11 Mortality Statistics

In the study period 3978 children were admitted to the burns unit, at approximately 500 per year. Total deaths numbered 72, giving an overall mortality of 1,8%. There were 86 inhalational burns, 42 of whom died, giving a mortality of 48,8%.

An improvement in mortality occurred during 1994 and 1995 as shown in **Table 3.14.**

Table 3.14 Mortality of Inhalation Injury with time.

Year	No. with Inhalation	Deaths	Mortality
1987	6	3	50%
1988	2	1	50 %
1989	8	6	75 %
1990	13	7	54 %
1991	9	6	66 %
1992	10	5	50 %
1993	17	12	70 %
1994	15	1	7 %
1995	6	1	17%

Table 3.15 Mortality of all Inhalation Injury by Age of Patient

Age	Total Cases	Deaths	Fire	Scalds
0 - 1	17	10	9 of 13	1 of 4
- 2	21	6	4 of 8	2 of 13
- 3	9	6	6 of 8	0 of 1
- 4	7	3	3 of 7	
- 5	8	6	5 of 7	1 of 1
- 6	4	1	1 of 4	
- 7	7	4	4 of 7	
- 8	5	3	3 of 5	
- 9	2	0	0 of 2	
- 10	1	1	1 of 1	
- 11	3	1	1 of 3	
- 12	1	0	0 of 1	
- 13	1	1	1 of 1	

Table 3.16 Mortality related to extent of skin surface burned.

%BSA	No. of Cases	Deaths	Mortality
0 - 9	13	2	15 %
10-19	14	3	21 %
20-29	12	1	8 %
30-39	10	5	50 %
40-49	7	3	43 %
50-59	7	6	85 %
60-69	4	4	100 %
70-79	6	6	100 %
80-89	8	7	88 %
90-100	5	5	100 %

Table 3.16. shows that mortality rises steeply to 100% with burns over 50% BSA. Above that extent there was only one exceptional survivor of an 80% fireburn. Because of %BSA burned being an overwhelmingly lethal factor it was impossible for any other factor to reach statistical significance in terms of influencing mortality. Those studied included presence of carbon monoxide poisoning, time delay to intubation and development of secondary pneumonia.

A decision not to treat actively was made on admission in 13 cases. In 11 of these there were massive skin burns, averaging 88% BSA burned. Case 66 was not treated actively because of severe facial burns and inhalation although this was not a uniform policy, and Case 58 was not treated for a secondary pneumonia because of tetraplegic cerebral palsy. There were obviously no survivors in this group.

Figure 3.1. Flexible bronchoscope being introduced in an awake child (Case 26) with facial burns.

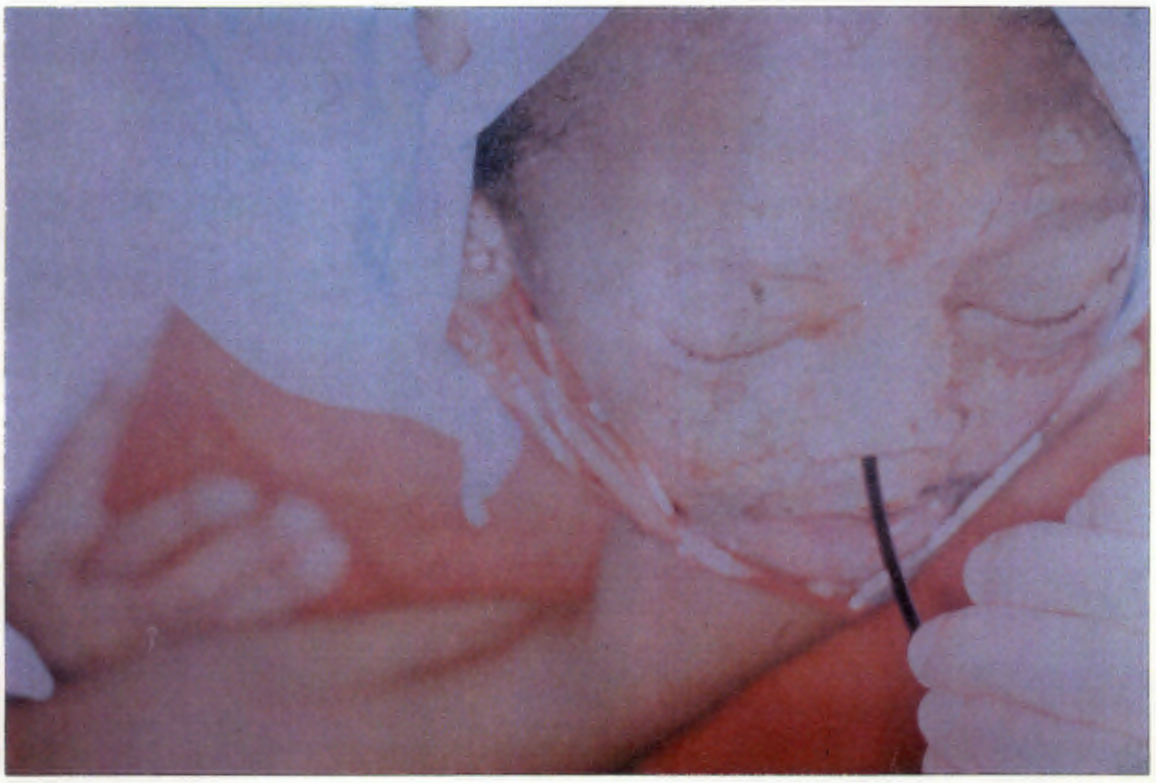


Figure 3.2. Bronchogram of Case 78 showing narrowing of the lower trachea and main bronchi by granulation tissue 5 months post-burn.

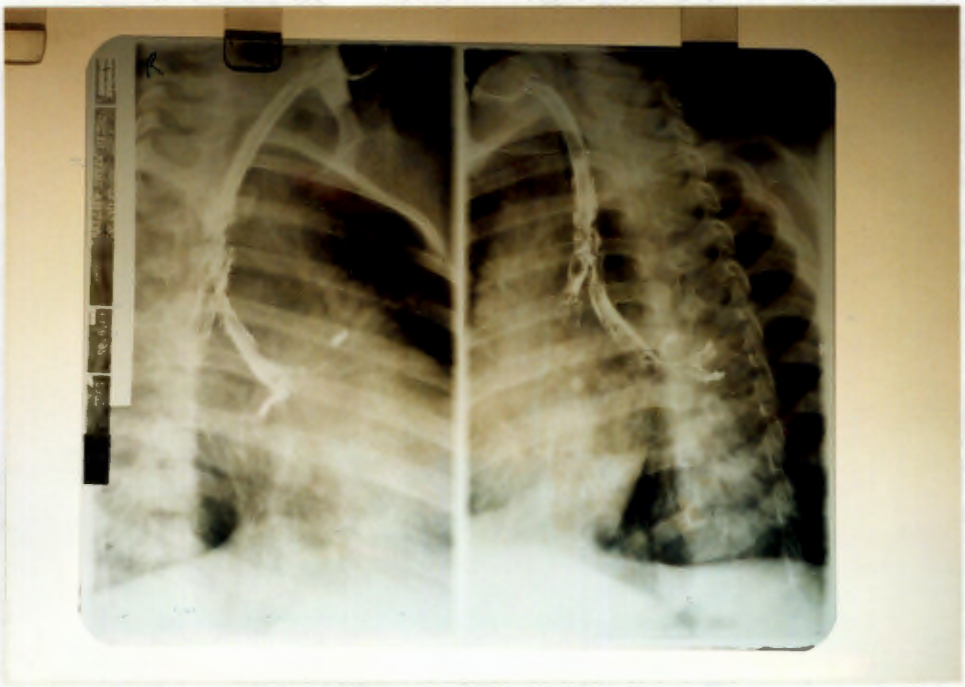


Figure 3.3 Media Interest surrounding Case 78 who underwent a tracheal Implant.

Windpipe implant toddler dies

2/6/93

Health Reporter

OCEAN View toddler Oslin Williams, the first child to have an artifical windpipe implanted, has died in Red Cross Children's Hospital.

Two-year-old Oslin battled to stay alive for more than a year after he was badly burnt in a gas explosion which killed his father.

Internal burns damaged his windpipe so severely that the scar tissue was slowly asphyxiating him. But there was new hope when British surgeon Mr Stephen Westaby visited South Africa to implant the silicone windpipe.

Mr Westaby was touched by Oslin's plight after he was introduced to him by the former head of cardiothoracic surgery at the University of Cape Town, Professor John Odell.

Oslin, who would have turned three this month, died of a lung haemorrhage early on Sunday. Professor Max Klein, head of the paediatric intensive care unit at the Red Cross Children's Hospital, said Oslin's death was like a "bolt out of the blue".

His funeral is in Ocean View at 1pm tomorrow.

Figure 3.4. Chest X-ray of Case 46 showing classical "white-out" sign on the right.

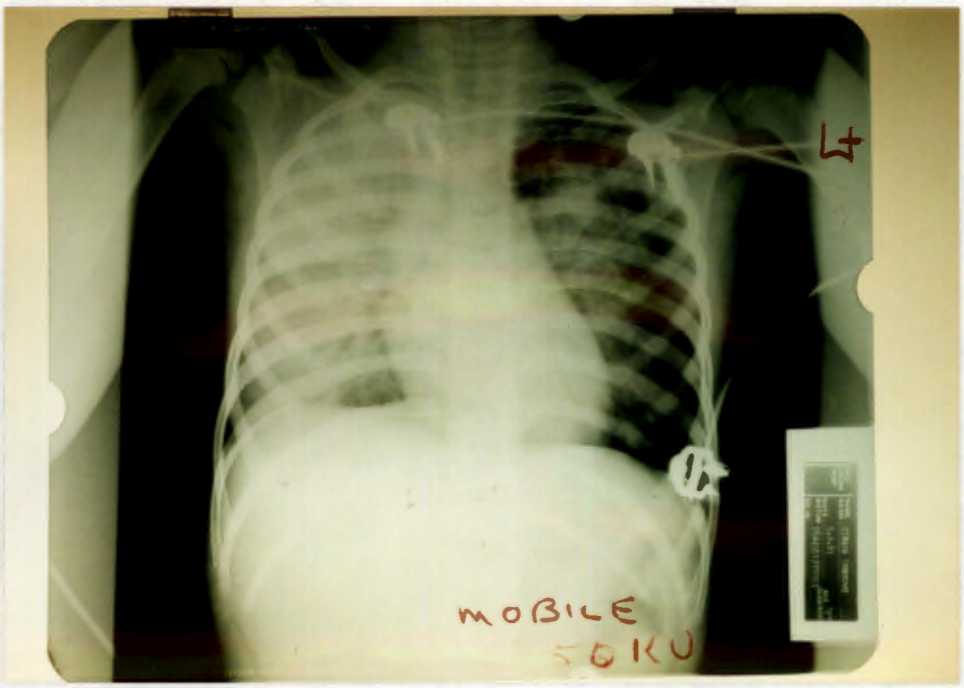


Figure 3.5. Xenon lung scan of Case 44 showing decreased perfusion of the right base (top left), and delayed washout of isotope from the left base (bottom right), characteristic of inhalational injury.

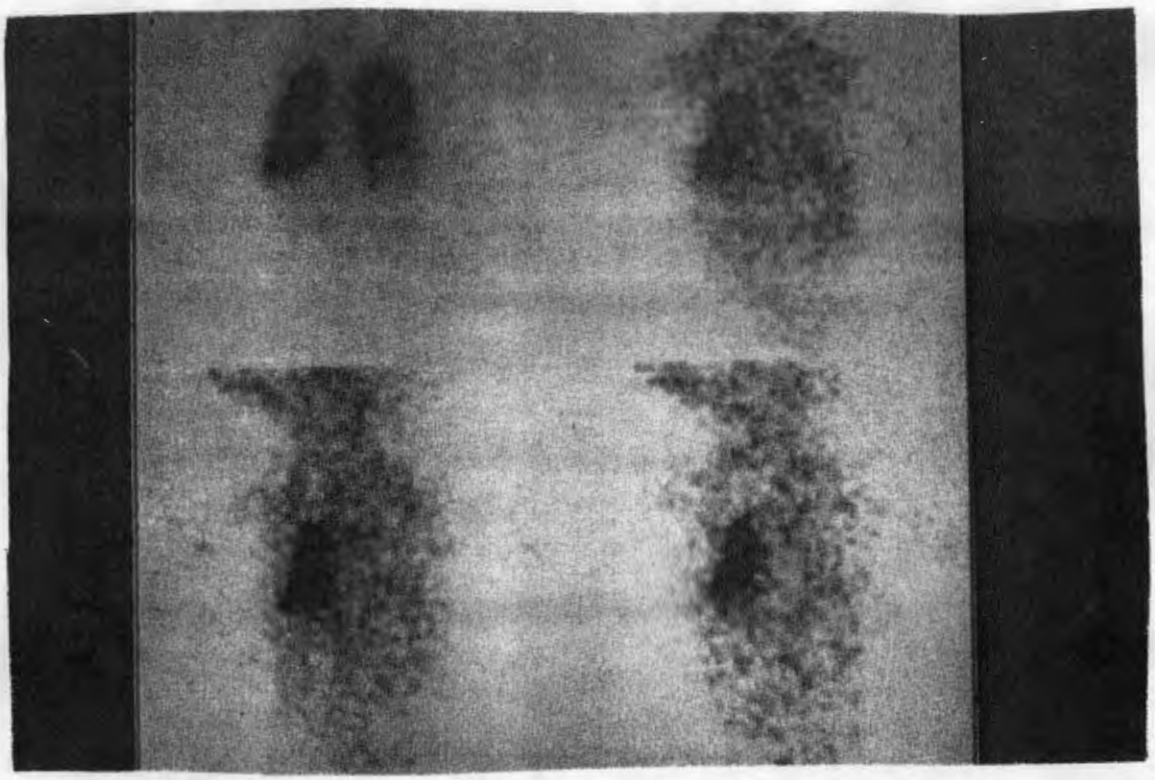


Figure 3.6. Normal chest X-ray in the same child (Case 44).

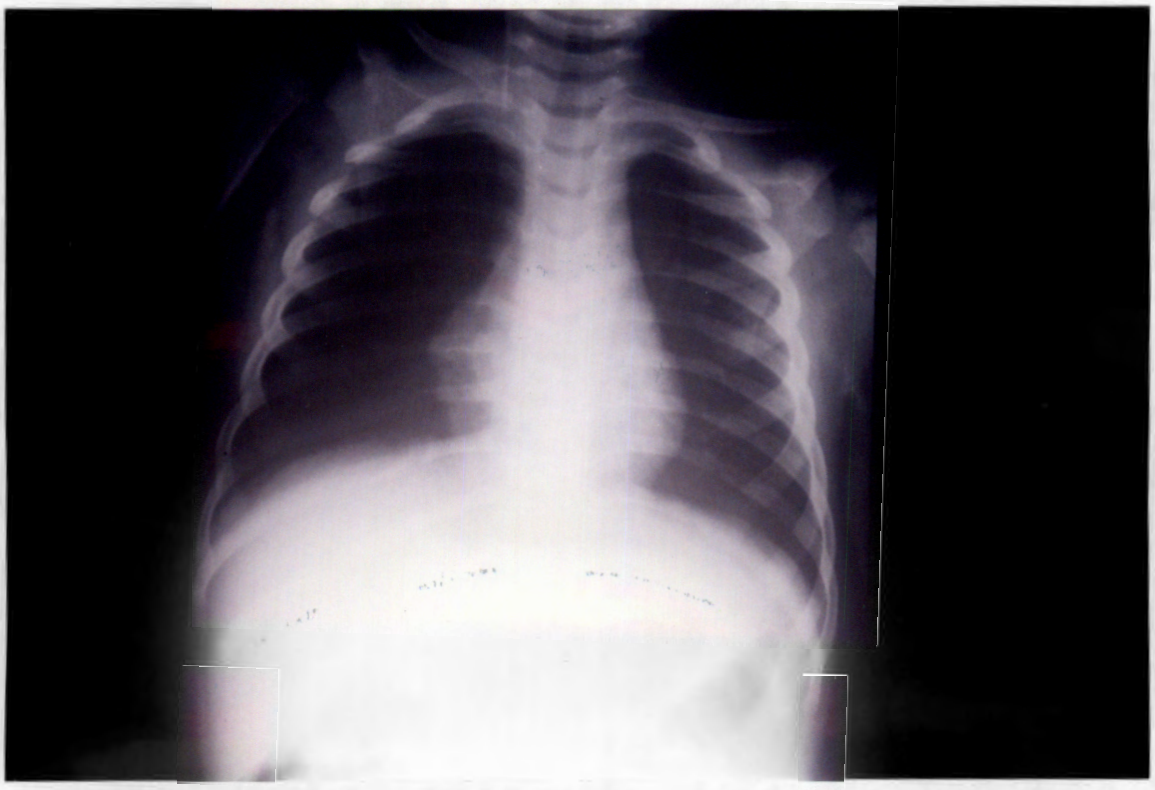


Figure 3.7: Chest X-ray 48 hours post-admission of a 2 month old boy (Case 59)with pneumoperitoneum and extensive subcutaneous emphysema caused by barotrauma.

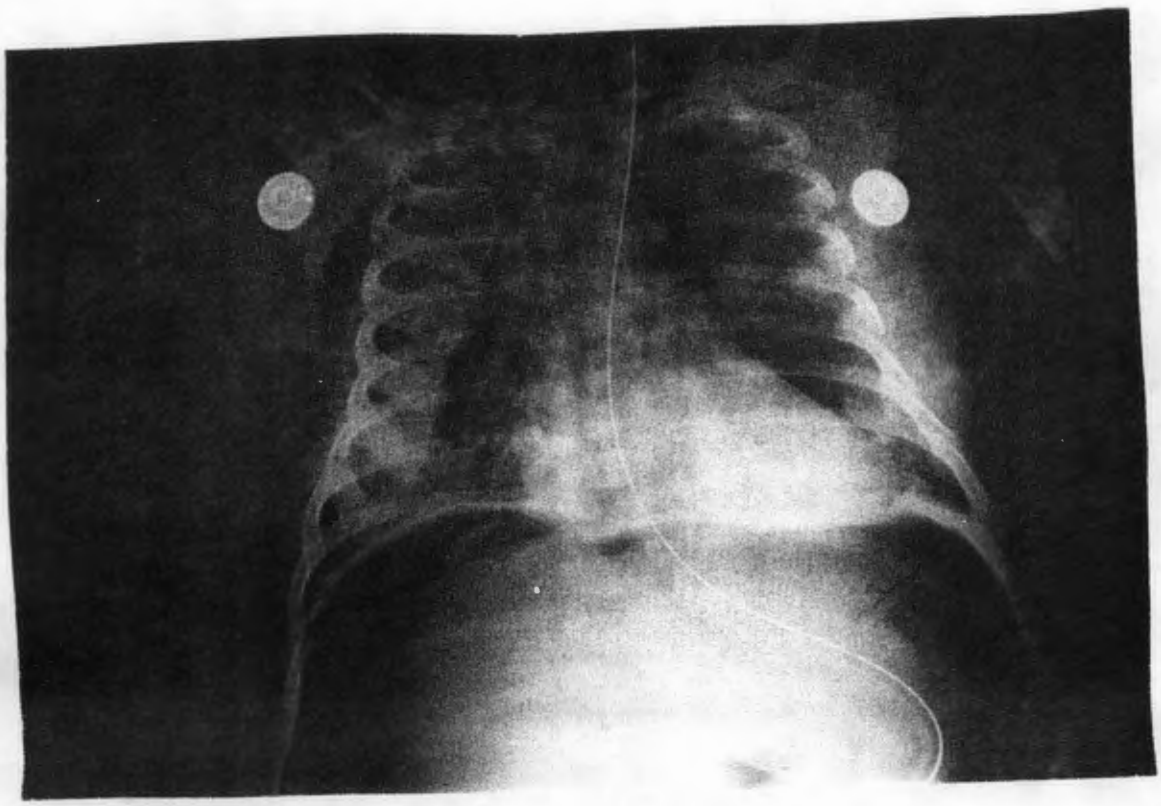
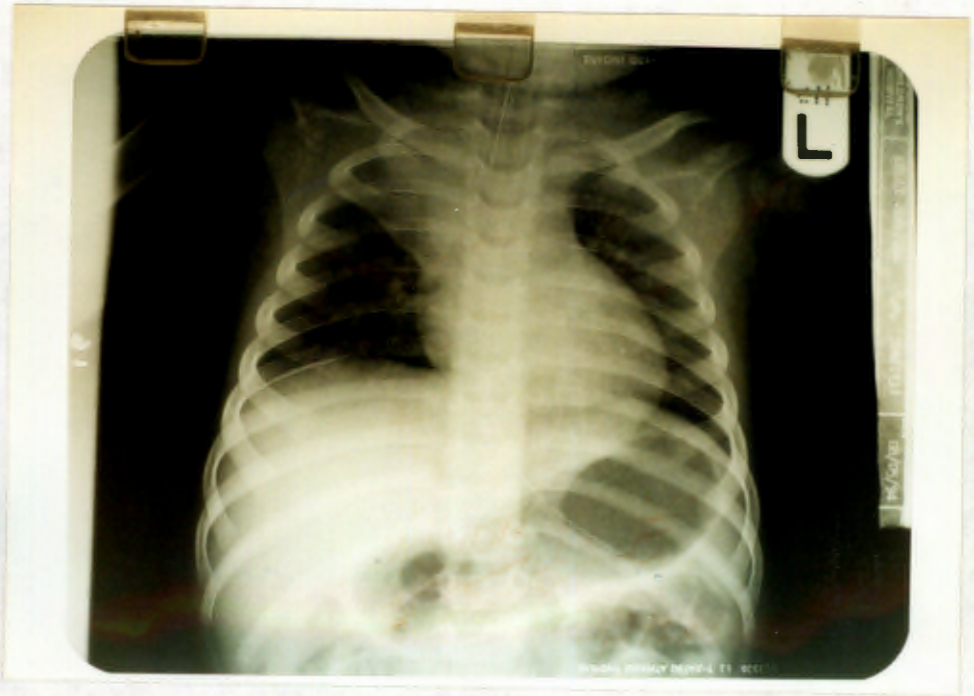


Figure 3.8. Right upper lobe consolidation in Case 29, 7 hours post-admission with hot water scalds.



4. CONCLUSIONS

4.1 Epidemiology

As with all accidents the main emphasis in childhood burns should be on prevention. Although data regarding circumstances of the fire was not always complete, 22 of the firebums could be linked to inadequate housing, since 1 of the burning cars was in fact an old minibus which was the family home. 10 cases were due to dangerous cooking facilities, 6 to open fires, 4 to dangerous lighting, and only 1 to an electric power station where the child was playing. Improvements in housing and safe electrification will therefore play a major role in decreasing the incidence of childhood burns.

5 of the firebums were non-accidental; 3 firebums resulted from domestic feuds, and one involved the murder of a mother shot by assailants as she tried to escape from the burning shack. A sibling trapped in the shack was dead on arrival. The much-debated causes of violence in the Cape peninsula are beyond the scope of this discussion.

4.2 Hot Water Burns

The hot water burns were all the consequences of household accidents, such as a child pulling a container of hot water off a stove or table. The mean age of these children was 18 months, with a median of 14 months and range of 8 - 50 months. They were therefore almost all in the toddler age group where such accidents are common but preventable. It is noteworthy that 50% of cases required intubation to secure the airway. Tracheostomy was an unfavourable option in 2 cases (Case 18, 40), as it was only needed for 48 - 72 hours and led to pneumonias developing in both cases. The finding of 11 cases of pneumonia among these 19 cases is surprising, since the mechanism of injury is direct heat to the upper airways. The predominance of right sided chest opacities suggests that the mechansim of pneumonia is aspiration. However, the stage at which it occurs is unknown. It may be the hot water itself that is aspirated, or aspiration of stomach content due to impaired laryngeal defences, for example at the time of intubation.

4.3 Classification of Injuries

In inhalational burns the need for a classification system to be simple and clinically applicable is confounded by the complexity of the spectrum of the disease. It is difficult to describe the mechanism causing patterns of injury in individual cases. Factors such as breath-holding while in the fire, loss of consciousness, the type of substance burning, and the temperature of the fire could all play a role. Isolation of such factors are beyond the control of any researcher.

4.4 Upper Airway Injuries

Isolated injury to one anatomical zone was more the exception than the rule, and this is logical when one considers the range in size of smoke particles which will concentrate at different levels in the tracheobronchial tree, and the concomitant presence of heat and toxic gases. Where isolated injuries did occur they may have been erroneous due to conservative indications for flexible bronchoscopy and xenon lung scans, and the retrospective nature of the data. eg. Case 52 had 85% fireburns with tracheal burns documented at post-mortem but no mention was made of the larynx. Ante-mortem bronchoscopy was not done because of his poor prognosis; with this information an upper airway injury which would one would have expected, cannot confidently be excluded.

The same pattern of damage at different anatomical levels emerges in the hot water burn group. However this is more difficult to explain, with heat as the only damaging agent. It is therefore proposed that aspiration occurs either at the time of injury, when the child attempts to drink the hot liquid or later due to laryngeal impairment by airway oedema. This is borne out by a predominance of right-sided pneumonias but would need further elucidation with bronchoscopy to identify the substance aspirated.

Fireburns tended to cause a deeper level of damage to the upper airways than hot water burns, probably due to higher temperature and toxic effects of the inhaled fumes.

Diagnosis of the upper airway burn on admission was a rare event because of the characteristic delay in onset of symptoms. This was described in survivors of the Coconut Grove fire, so it would seem that even adults are not able to communicate the symptoms

after the stress of a burn injury. Added to this is the understandable reluctance of emergency staff to thoroughly inspect the mouth of such a severely injured patient. Stridor progresses with burn oedema elsewhere as the patient is resuscitated, so that symptoms usually begin after leaving the emergency unit for the ward.

Diagnosis on admission is important as it allows for optimal timing of bronchoscopy. If there are no signs of airway compromise, the procedure can be done more safely and visualisation below the cords can be achieved. However, if delayed until the onset of stridor, the child is more distressed, nothing can be seen below the obstruction, and the event frequently occurs in the middle of the night when expertise is limited. Expertise is required both to perform and interpret this investigation.

The timing and need for intubation of upper airway oedema is difficult to judge. While the burn oedema tends to resolve in 48 hours, those with deeper burns have necrosis and slough which can continue to obstruct the airway for some time. Initial assessment and regular follow-up bronchoscopies are indicated, especially where clinical progression does not fit the initial bronchoscopy findings. With increasing experience in this field we may be able to make use of serial bronchoscopic biopsies to increase the initial diagnostic accuracy of the procedure.

One factor that does emerge is that intubation does not seem to have made a difference to the incidence of secondary pneumonias in isolated upper airway injury (42 vs. 36% in **Table 3.5**). This suggests that the injured airway with impaired clearing of secretions and impaired cough is at equal risk of infection as the intubated airway. The same level of surveillance, with regular suctioning and bronchial lavage should therefore be employed regardless of whether the airway is critically narrowed.

Residual laryngeal damage seemed to be the exception rather than the rule, with 4 cases out of 26 survivors. One of these was probably the result of intubation trauma, and the other 3 due to full thickness mucosal loss with healing by granulation and fibrosis. This was demonstrated by serial bronchoscopy, however there is little that can be done to minimise the damage (Calhoun, '88).

The one death due to loss of the airway and hypoxic brain damage was preventable. In this case it was not realised that the presence of ulceration of the larynx indicates the potential for more slough and fibrin to threaten the airway. Extubation is premature under these conditions.

4.5 Major Airway Injuries

Major airway damage is essentially a silent injury, which presents with symptoms of coinciding upper and parenchymal damage. The contribution it makes to secondary pneumonia and mortality is therefore hard to assess.

Early bronchoscopy has great value in documenting this injury, and improving the level of surveillance. The descriptive findings at bronchoscopy do not have any predictive value as to severity of injury, especially where the trachea is lined with soot. Regular repeat bronchoscopy will however show severity as the soot gives way to ulceration, slough and granulation. The role of heparin infusion in decreasing tracheobronchial cast formation (Cox, '93) warrants further investigation, but may be hampered by the need for early tangential excision of the burn wounds.

The poor outcome in 6 patients who did not require intubation is surprising. It suggests that prophylactic intubation may improve the removal of slough and debris, thus preventing the potentially lethal complication of secondary pneumonia.

Tracheostomy is needed when the airway is compromised for more than approximately a week and the improved access for lavage and tracheal toilet plays an important supportive role.

Severe tracheal damage occurs in the exceptional case and is extremely difficult to manage. Case 78 who came to tracheal replacement had 2 endoscopic and 2 laser attempts to remove granulation. It always re-formed and numerous dilatations provided only temporary relief of obstruction (see **Fig 3.2**). When tracheal replacement was finally done it still could not overcome the problem of scarring in the main bronchi. The problem of graft

sepsis in the setting of bronchiectasis and a tracheostomy also proved insurmountable.

4.6 Parenchymal Injuries

Of all the anatomical categories, the injuries in this group were the least well-documented clinically. Early respiratory distress was the most consistent feature, occurring in 27 cases.

In keeping with other studies the initial chest X-ray was unhelpful with a large proportion having normal X-rays on admission (See **Fig 3.6 and 3.7b**) and developing focal consolidation after 2 -3 days. Only 3 cases had the characteristic "white-out" or pulmonary oedema picture. In only 1 case was it feasible to arrange a Xenon lung scan as she had mild signs and was not on a ventilator Case 44). The scan showed signs characteristic of inhalation injury. Ideally the facility is required for ventilated patients. It must be done within the first 3 days of injury, since the clearance pattern reverts to normal after 4 days in 80% of cases (Moylan,1972).

The ventilatory pressure requirements were standard in 10 out of the 18 cases ventilated with a hope of cure. This does not exclude minor degrees of loss of lung compliance, which were not clinically problematic. In those requiring increased pressure support this proved to be a downward spiral of hypoxia and barotrauma, which was turned around in 1 case by the administration of high frequency ventilation. This is a modality which we will definitely use in the future, with a view to instituting it early, before barotrauma is sustained.

7 of the 8 survivors in this series presented after 1992 when there was a greater awareness of the severity and reversible nature of this injury. More sophisticated ventilators, and establishment of an intensive case unit staffed by a paediatric intensivist almost certainly contributed.

4.7 Secondary Bronchopneumonias

The equal incidence of secondary pneumonias with upper, major and parenchymal burns suggest that general factors as listed in the introduction are still important factors contributing to secondary pneumonias. A more constructive approach would be to examine

the cases who did not develop secondary pneumonias to see what aspects of their management were better. One such case had upper and major airway fireburns. Bronchoscopy was done on admission and the injury was accurately documented. She was treated in ICU from admission with nebulisers and frequent suctioning. Oral intubation was performed before her airway became critical and she then had humidified oxygen, frequent suctioning and physiotherapy. Repeat bronchoscopy also assisted with lavage of her trachea. Post-extubation she remained hoarse for another week but did not develop any chest infections. This proved to be the ideal treatment.

Of the 28 deaths in parenchymal injury group, 14 were clearly not salvageable at the time. However, the salvage rate may increase in the future and 1994 saw the first survivor of an 80% BSA burn with upper, major and parenchymal inhalation. There was only 1 obviously preventable death, in a child with 35% BSA skin burns who was noted to have respiratory distress while on headbox oxygen in the ward but had a respiratory arrest before ventilatory support was implemented.

4.8 Post-Mortem Findings

In many post-mortems the findings were not suggested by the preceding clinical picture. The question of sub-clinical damage to the major airways and parenchyma with its potential consequences of ciliary impairment and later pneumonias is one that needs to be addressed.

Post-mortem results were particularly useful in the category of parenchymal injury, since most of the cases died without a specific diagnosis. Post-mortem signs correlated well with duration of survival as documented by various authors (Chu, '81; Bowden, '79; Herndon, '84). One criticism of the post-mortem reports is the frequent description of congested heavy lungs as bronchopneumonia. The accuracy of macroscopic findings to draw such a conclusion has to be questioned, given that all forms of interstitial and alveolar oedema would also produce heavy congested lungs. It has to be remembered that all but one of the post-mortems were done by forensic pathologists from the police mortuary, with the purpose of excluding foul play rather than making specific diagnoses.

4.9 Mortality

Isolation of the variables affecting mortality has treatment implications, since some of the variables can be altered by treatment, while others eg. the % BSA burned, are fixed.

The effect of time on mortality reflects improvements in the expertise in our ICU in the latter years of the study, as well as increased awareness amongst all staff of the symptoms and dangers of inhalation.

Age as an independent variable appears to show an increased survival in the younger age groups, however this is spurious since most of the hot water scalds occurred in this age group, while older children tended to be involved in serious fires, from which they could not escape.

Many authors have stated that the presence and extent of skin burns determines mortality (Thompson,' 86; Clark, '93). The above data concurs in that only 23% of patients with burns over 30% BSA survived. Having established that this is the most important variable governing mortality, it becomes difficult to assess any other variables independently. All of the other factors should be viewed in the light of this. eg. a problem of time delay was identified in 6 cases but this included time delay in reaching the hospital and underassessment of the airway damage on arrival. However, all of these 6 patients had skin burns of 35-60% BSA therefore they all fell into a very high risk category.

Finally, the decision to treat actively impacts on the mortality for obvious reasons. In the beginning of the study it was accepted that patients with burns of 75% and over could not survive, and the presence of inhalation in such patients was of academic interest only. In the middle of the study period (1990-2), an even more circumspect policy was adopted, with burns of lesser areas ie.50-70% BSA were being recognised as unsalvageable, as well as very deep burns eg. burns through the skull, charred limbs or trunk. Case 58 was treated conservatively with relatively minor (19% BSA) hot water burns because she had cerebral palsy and tetraplegia. Inhalation per se was recognised as a lethal injury in case 66 with 38% skin burns. Towards the end of the study (1993-5) aggressive attempts were being made to salvage even 80% burns, with the only proviso being that the hands and face

were not too badly affected to preclude a good functional and cosmetic outcome.

The variability of outcome in the grey area of surface burns between approximately 35 and 75% BSA, make one very reluctant to include inhalation as an adverse prognostic factor when deciding whether to continue active management. This variability makes it especially important not to regard inhalation as a separate prognostic factor, but rather as a factor which rises in incidence with the area of skin burns, and which has reversible and irreversible elements. It therefore presents us with a challenge in salvaging major burns.

As with all retrospective studies the inevitable conclusion to be drawn from these 86 cases is that more research is required. While the subject matter is not amenable to a randomised controlled trial, many advances could be made by the RCWMCH by the establishment of a research protocol. At present a few guidelines for a management protocol are also beginning to emerge. Inhalation should be suspected in all major fire burns (30% BSA and over), and smaller burns involving the face and mouth.

5. A Proposed Research Protocol for inhalational burns

In all suspected cases the following data needs to be accurately recorded on admission.

History	Examination Findings
Causative Agent	Level of Consciousness
Time Delay	Presentation of Airway
Duration of Exposure	Respiratory Pattern
Material Burning	%BSA burned
Situation Open or Closed	Singeing of nasal hairs
Level of Consciousness	Oral and Pharyngeal burns
	Carbonaceous sputum
Background History	Hoarse voice or stridor
Previous Chest Pathology	Tachypnoeia
Neurological Impairment	Respiratory Distress
	Chest Signs
Investigations	
Immediate:	**Management**
Arterial Blood: - Gases	Fluid resuscitation volume
- Carbon monoxide	Oxygen Requirements
Blood for cyanide levels	Ventilatory requirements
(to toxicology lab)	Timing of Intubation
Flexible bronchoscopy	
Later:	**Outcome**
Chest Xray	Survival
Xenon Lung scan at 48hrs	Secondary pneumonia
Microbiology - +ve cultures	Laryngeal competence
Post Mortem:	Residual lung damage
Histology of lung parenchyma	Psychological sequelae
and tracheal mucosa	

Problems with the above protocol mainly concern the limited availability of sophisticated toxicology, radionuclide scans, bornchoscopic expertise and forensic histology. However, our data suggests that certainly for the xenon lung scan the yield for positive results should exceed 40% thus making it a valuable research tool. The optimal time for this investigation is at 48 to 72 hours, therefore it need not be arranged after hours. The incidence of cyanide poisoning may be a lot lower than this, but needs such a screening programme to be quantified. The problem of obtaining detailed information from forensic post-mortems done

by the Department of Forensic Medicine would require the introduction of a standard autopsy protocol for all burns deaths.

5.1 Management Guidelines

The following questions need to be addressed-:

Should the fluid resuscitation volume be altered to take account of an inhalational element?

There is little data to support the common practice of adding 10% to the surface area burned in calculating replacement for inhalational burns. This may theoretically lead to over-enthusiastic fluid resuscitation in small burns, with consequent aggravation of upper airway oedema. In larger burns it has prognostic implications which may unfairly influence the decision to treat actively. Herndon has proposed a figure of 2ml/Kg/% BSA burned above the calculated volume, a figure which he found did not adversely affect extravascular lung water volumes, and which seems more realistic than 10% (Herndon, 1988). However, the wide variety of presentations and outcomes demonstrated here show that inhalation is not a single factor that can be added to an injury score but is a complex combination of insults with varying degrees of severity, many of which are fully reversible if treated correctly.

Who needs intubation for upper airway obstruction?

Our data shows that approximately half of the isolated upper airway injuries whether caused by fire or hot water will not settle on nebulisers and will require intubation. Bronchoscopic findings of ulceration or slough are more significant than just oedema, but no case can be dismissed from careful observation on bronchoscopic grounds.A safe policy is to treat all cases with positive findings on bronchoscopy in a high observation ward, with nebulisers, regular suctioning and physiotherapy. Children whose stridor is progressing can then be recognised early.

Who needs a tracheostomy?

Securing of the airway in upper airway burns is best done through the endotracheal route at

first. This is ideally done after bronchoscopy has identified burns that may compromise the airway in the first 48 hours, and before the child has progressed to grade 3 stridor. Repeat bronchoscopy can then be done in theatre every 2 -3 days to reassess damage, and to remove fibrin and slough. Tracheostomy can then be planned as an elective procedure if the laryngeal injury shows no signs of subsiding after a few days. By this approach the heat-induced hot water injuries will have resolved before tracheostomy need be considered, while the fireburns with ongoing slough and associated tracheal damage will not be exposed to the hazard of premature extubation.

What about prophylactic antibiotics?

It is our present policy to commence prophylactic gram-positive cover in all major fire-burns of 30% and over. This is based on the known pattern of colonisation of the burn wound and the particularly invasive nature of these organisms. However this policy does not directly address the issue of inhalation with the secondary pneumonias that we have seen to occur in 25% of cases. These children are best managed with regular sputum and blood cultures, followed by appropriate antibiotics. In the absence of positive sputum cultures, burn cultures should also be considered since these organisms are known to seed out in the lung.

This study increases our awareness that few burn centres handle the volume of paediatric burns that we encounter at the Red Cross Children's Hospital, while still having expertise available to investigate and salvage major burns. Thus, poised at the interface between first world medicine and third world living conditions we have the rare opportunity to make great advances in this field.

APPENDIX

LIST OF CASES

An overview is presented of all cases showing size, site and outcome of burn.

Case	Age (months)	%BSA burned	Cause	Inhalation	Outcome
1	13	12%	water	upper	resolved
2	14	6%	water	upper	resolved
3	95	95%	fire	upper, major & parenchymal	died after 36 hrs
4	3	30%	fire	upper	resolved
5	69	20%	fire	upper	resolved
6	8	3%	water	upper	resolved
7	31	0%	fire	parenchymal & CO poisoning	died after 20 minutes
8	47	2%	fire	upper, major, parenchymal & CO poisoning	resolved
9	78	82%	fire	upper,major & parenchymal	died after 9 days
10	9	60%	fire	parenchymal & CO poisoning	died after 6 days
11	91	45%	fire	upper,major & parenchymal	died after 2 days
12	27	35%	fire	major & parenchymal	died after 2 days
13	82	10%	fire	upper	resolved
14	55	65%	fire	upper,parenchymal and CO	died after 7 days
15	106	0%	fire	upper,parenchymal and CO	resolved
16	79	60%	fire	upper & major	died after 12 days
17	14	15%	water	upper with aspiration	resolved
18	19	0%	water	upper with aspiration	resolved
19	78	25%	fire	upper & parenchymal	resolved
20	72	34%	fire	upper	resolved
21	24	10%	water	upper	resolved
22	24	33%	fire	upper	resolved
23	77	50%	fire	upper	died after 28 days
24	5	40%	fire	upper	resolved
25	144	85%	fire	upper & parenchymal	died after 28 days

26	72	13%	fire	upper & major	resolved
27	14	8%	water	upper	resolved
28	106	18%	fire	upper,major parenchymal & CO	resolved
29	15	8%	water	upper with aspiration	resolved
30	32	28%	water	upper	resolved
31	13	75%	fire	upper,parenchymal & CO	died after 15 days
32	45	70%	fire	upper,major,parenchymal & CO	died after 40 minutes
33	74	12%	fire	upper	resolved
34	1.5	35%	fire	upper,parenchymal & CO	died after 2 days
35	140	80%	fire	upper,major & parenchymal	survived
36	43	35%	fire	upper & parenchymal	resolved
37	25	64%	fire	parenchymal	died after 4 days
38	53	72%	fire	upper,major & parenchymal	died after 2 days
39	12	28%	fire	upper & CO	resolved
40	12	6%	water	upper with aspiration	resolved
41	58	75%	fire	upper,major & CO	died after 12 hours
42	59	35%	fire	upper & major	died after 3 days
43	0.3	50%	fire	upper,parenchymal & CO	died after 1 hour
44	12	75%	fire	parenchymal	died after 12 days
45	63	50%	fire	upper,major & parenchymal	died after 3 days
46	43	50%	fire	upper,major & parenchymal	died after 5 hours
47	50	25%	water	upper with aspiration	died after 3 days
48	140	84%	fire	parenchymal	died after 6 hours
49	39	30%	fire	upper	resolved
50	42	35%	fire	upper	resolved
51	114	95%	fire	upper & parenchymal	died after 2 days
52	22	85%	fire	major & CO	died after 6 hrs
53	75	4%	fire	upper,major,parenchymal & CO	died after 28 hrs
54	12	7%	water	upper with aspiration	resolved
55	21	25%	fire	upper & parenchymal	resolved
56	80	55%	fire	upper & major	resolved
57	5	55%	fire	upper & major	died after 88 days

58	19	17%	water	upper with aspiration	died after 12 days
59	2	13%	fire	upper,major & parenchymal	died after 3 days
60	25	5%	fire	upper	resolved
61	132	75%	fire	upper,major,parenchymal & CO	died after 2 days
62	28	50%	fire	upper & parenchymal	died after 40 days
63	56	8%	fire	CO	resolved
64	120	12%	fore	upper	resolved
65	35	20%	fire	upper,major & CO	resolved
66	35	38%	fire	upper,major,parenchymal & CO	died after 7 hours
67	3	85%	fire	upper,major & CO	died after 1 hour
68	8	85%	fire	parenchymal & CO	died after 2 hours
69	22	20%	water	upper with aspiration	resolved
70	90	100%	fire	upper & major	died after 12 hours
71	132	20%	fire	upper & major	resolved
72	11	10%	water	upper with aspiration	died after 2 days
73	95	40%	fire	upper	resolved
74	14	12%	water	upper	resolved
75	7	85%	fire	major,parenchymal & CO	died after 4 hours
76	34	95%	fire	parenchymal	died after 6 hrs
77	8	25%	fire	upper	resolved
78	22	45%	fire	upper,major,parenchymal & CO	died after tracheal transplant, 14 months post burn
79	39	98%	fire	upper & parenchymal	died after 6 hours
80	14	30%	water	aspiration	died after 2 days
81	50	45%	fire	upper & parenchymal	died after 18 days
82	24	48%	fire	upper & parenchymal & major	resolved
83	56	35%	fire	parenchymal & CO	resolved
84	15	25%	fire	upper & CO	resolved
85	14	15%	water	upper with aspiration	resolved
86	14	7%	water	upper	resolved

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BIBLIOGRAPHY

Aub,J.C.,Pittman,H.,Brues,A.M.:Management of the Coco nut Grove burns at the Massachusetts General Hospital: The pulmonary complications - a clinical description. *Ann.Surg.,***117**:834-840,1943.

Birky M.M.,Clarke F.B.Inhalation of toxic products from fires. *Bull N.Y.Acad Med.***57**.997.1981.

Boettner E.A.,Ball G., Weiss B. Analysis of the Volatile Combustion Products of Vinyl Plastics.*J Appl Polymer Sci.***13**,377.1969.

Bowden D.H. Reaction of the Lung to Injury. Scientific Foundations of Respiratory Medicine. Ed. J.G Scadding & G. Cumming.Chapter 42,529-545,1979.

Bridgman,D(ED).South Africa's Leading Edge? A guide to the Western Cape Economy. Wesgro, Capetown.1992.

Caldwell F.T.,Bowser B.H. Critical Evaluation of Hypertonic and Hypotonic Solutions to Resuscitate Severely Burned Children: A Prospective Study. *Ann Surg* **189**,546-552.1979.

Calhoun K.H., Deskin R.W., McCracken M.M., Hokanson J.A., Garza C., Nichol R.J., Herndon D.N. Long-term Airway Sequelae in a Paediatric Burn Population. *Laryngoscope* **98**,721-725, 1988.

Chu,C.New Concepts of Pulmonary Burn Injury. *J Trauma.***Vol 21**, No 11,958-961,1981.

Cioffi W.G., deLemos R.A., Coalson J.J., Gerstmann D.A., Pruitt B.A. Decreased Pulmonary Damage in Primates with Inhalation Injury Treated with High-Frequency Ventilation. Ann. Surg.**218**(3), 328-337,1993.

Cioffi W.G., Graves T.A., McManus W.F., Pruitt B.A.Jr. High-frequency Percussive Ventilation in Patients with Inhalation Injury. *J Trauma* **29**(3) 350-54.1989.

Clark W.R., Grossman Z.D., Nieman G.F. Positive Computed Tomography of Dog Lungs Following Severe Smoke Inhalation; Diagnosis of Inhalation Injury. *J.Burn Care Rehabil.*

3,207,1982.

Clark W.R., Smoke Inhalation:Diagnosis and treatment. *World J.Surg*. **Vol 16**, 24-29,1992.

Cohen M.A.,Guzzardi L.J.Inhalation of products of combustion. *Ann Emerg Med*;**12**,628-632,1983.

Cope,O:The management of the Cocoanut Grove burns at the Massachusetts General Hospital, treatment of surface burns. *Ann Surg.,***117.**;885,1943.

Cox C.S., Zwischenberger J.B., Traber D.L.,Traber L.D., Haque A.K., Herndon D.N.Heparin Improves Oxygenation and Minimizes Barotrauma after Severe Smoke Inhalation in an Ovine Model. *Surgery, Gynaecology & Obstetrics*.**176**,339-349,1993.

Cudmore R.E.,Vivori E.Inhalational injury to the respiratory tract of children. *Prog Paed. Surg.***14**:173,1981.

De Wet B., Davies M.R.Q.,Cywes S.Die oorsake van brandwonde by kinders. *S Afr Med J.***52**.969-72.1977.

Du Toit & Van der Vyver(Consulting electrical engineers). The World Bank, Introduction to the Cape Metropolitan area, Electricity supply and distribution. July 1992.

Dye D.J., Milling M.A.P., Emmanuel E.R. et al. Toddlers, Teapots and Kettles: Beware Intraoral Scalds. *Br Med J* **300**.597-98.1990.

Finck P.A. Exposure to Carbon Monoxide: Review of the Literature and 567 Autopsies. *Milit Med* **131**.1513.1966.

Fisher A.B., Hyde R.W., Baue A.E et al. Effect of Carbon Monoxide on Function and Structure of the Lung. *J Appl Physiol* **26**.4.1969.

Friedberg K.D., Schwarzkopf H.A.Blausaeureexhalation bei der Cyanidvergiftung. *Arch Toxicol* **24**.235.1969.

Freitag L., Kim C.S., Long W.M., et al. Mobilisation of mucus by airway oscillations. *Acta*

Anaesthesiol Scand **33**(Suppl 90)93-101,1989.

Fry W.A., Semerjdjian R.A. Inhalation injury evaluation and management in a burns unit. *Ann Otol.***86**.667-670.1977.

Garland J.S., Rice T.B., Kelly K.J.Airway Burns in an Infant Following Aspiration of Microwave-heated Tea. *Chest* **90**(4), 621,1986.

Hachtenberg T., Wendt M., Deitmer T., Lawin P. Viscoelasticity of Tracheobronchial Secretions in High-frequency Ventilation. *Crit Care Med* **15**(2) 95-98,1987.

Head J.M. Inhalation Injury in Burns. *Am J Surg* **139**,508-513,1980.

Herndon D.N., Traber D.L., Niehaus G.D., Linares H.A., Traber L.D. The Pathophysiology of Smoke Inhalation Injury in an Sheep Model. *The Journal of Trauma* **24**(12)1044-1051,1984.

Herndon D.N., Thompson P.B., Linares H.A., Traber D.L.Postgraduate Course: respiratory Injury Part 1.*JBCB* **7**.No2.184-191.1986.

Herndon D.N, Barrow R.E, Traber D.L, Rutan T C, Rutan R.L, Abston S. Extravascular Lung Water Changes following Smoke Inhalation and Massive Burn Injury. *Surgery* **102** 341,1987.

Herndon D.N., Barrow R.E., Linares H.A., Rutan R.L., Prien T., Traber L.D., Traber D.L. Inhalation Injury in Burned Patients: Effects and Treatment. *Burns.***14**,349-356, 1988.

Hollingsed T.C., Saffle J.R., Barton R.G., Craft W.B., Morris S.E. Aetiology and Consequences of Respiratory Failure in Thermally Injured Patients. *Am.J Surg.***166**: 592-597,1993.

Hudson D.A., Jones L.W., Rode H. Respiratory distress secondary to scalds in children.*Burns.***20** *(5)434-437,*1994.

Jung R.C., Gottlieb L.S. Respiratory Tract Burns after Aspiration of Hot Coffee. *Chest* **72**,125-127,1977.

Klaassen C.D. Non-metallic environmental toxicants: air pollutants,solvents and

vapours,and pesticides. In Goodman Gilman A., Goodman L.S.Rall T.W. et al.(eds). The pharmacologicalBasis of Therapeutics. New York:Macmillan,p1628.

Kibel S.M., Bass D.H., Cywes S., Five years experience of injured children. *S Afr Med J* **78**:387,1990.

Krantz J.C., Carr C.J., Forman S.E. et al. Alkyl nitrates VI: a Contribution to the mechanism of Action of Organic Nitrates. *J Pharmacol. Exp Ther* **70**.323.1940.

Lee J.M., O'Connell D.J. The Chest Radiograph after Acute Smoke Inhalation. *Clin Radiol.* **39**,33,1988.

Lloyd E.L., Macrae W.R.Respiratory tract damage in burns, case reports and a review of the literature.*B J Anaes,* **43**: 365-79,1971.

Masanes M-J, Legendre C, Lioret N, Maillard D, Saizy R, Lebeau B. Fibreoptic Bronchoscopy for the Early Diagnosis of Subglottal Inhalation Injury: Comparative Value in the Assessment of Prognosis. *J Trauma* **36,1**, 59, 1994.

Masanes M-J, Legendre C, Lioret N, Saizy R, Lebeau B. Using Bronchoscopy and Biopsy to Diagnose Early Inhalation Injury. *Chest* **107,5** 1365, 1995.

McKiernan M.J. Emergency Treatment of Cyanide Poisoning. *Lancet* **ii**,86.1980.

Mellins R.B., Bark S. Respiratory complications of smoke inhalation in victims of fires.*J Paediatr.***87**, 1-7.1975.

Moritz A.R., Henriques F.C., MacLean R., The effects of inhaled heat on the air passages and lungs: an experimental investigation.*Am J Path.***21**:311-25, 1945.

Mosley S. Inhalation injury: A review of the literature.Pulmonary aspects of critical care. *Heart &Lung* **17**. 3-9.1988.

Moylan J.A., Wilmore D.W., Mouton D.E., Pruitt B.A. Early diagnosis of inhalation injury using 133 Xenon lung scan.*Ann Surg* **Vol 176**.No4.1972.

Moylan J.A., Abib K., Birnbaum M. Fibre-optic bronchoscopy following thermal injury. *Surg*

Gynaecol Obstet **140** 541-543.1975.

Moylan J.A., Alexander G.Jr.Diagnosis and treatment of inhalation injury. *World J Surg* **2**, 185-91.1978.

Moylan J.A., Chan C-K.Inhalation Injury-an increasing problem.*Surgery* **188**.34-37.1978.

Moylan J.A., Smoke inhalation and burn injury. *Surg Clin N Am.* **Vol 60,** No6, December 1980.

Moylan J.A., Inhalation Injury - a primary determinant of survival following major burns. *JBCR.* Mar/Apr 1981.80-84.

Navar P.D., Saffle J.R., Warden G.D. Effect of inhalation injury on fluid resuscitation requirements after thermal injury. *Am J Surg*.**150**.716-20.1985.

Neubauer R.A. Smoke inhalation injury(Letter). *Postgraduate Medicine*.**83,** no 1. 1988.

Nieman G.F., Clark W.R., Wax S.D. The Effect of Smoke Inhalation on Pulmonary Surfactant. *Ann.Surg*,**191**,171,1980.

O'Neill J.A. Fluid Resuscitation in the Burned Child: A Reappraisal. *J Pediatr Surg* **17**, 604-7.1982.

O'Neill W.J., Jordan M.H., Lewis M., Snider R.H., Moore C.F., Becker K.L. Serum Calcitonin is a Marker for Inhalational Burn Injury. *J. Burn Care Rehab.* **13**.March-April, 1992.

Nylen E.S., O'Neill W.J., Jordan M.H., Snider R.H., Moore C.F., Lewis M., Silva O.L., Becker K.L. Serum Procalcitonin as an Index of Inhalation Injury in Burns. *Horm Metab. Res.* **24**,439-442.1992.

Philips A.W., Cope O. Burn Therapy II: The Revelation of Respiratory Tract Damage as a Principal Killer of Burned Patients. *Ann Surg* **155**:1,1962.

Pollard, S.A.Burns and Carbon Monoxide Poisoning. *Br J Surg* **57**.635-637.1970.

Prien T., Traber D.L.Toxic smoke compounds and inhalation injury - a review. *Burns*

14,(6),451-60. 1988.

Pruitt B.A., Ericksen D.R., Morris A.,Progressive pulmonary insuffiency and other pulmonary complication of thermal injury.*J Trauma* **15**,369,1975.

Robson M.C., Burns B.F., Smith D.J. Acute Management of the Burned Patient.*Plastic & Reconstructive Surgery* **89** No.6,1155,1992.

Rue L.W., Cioffi W.G., Mason A.D., McManus W.F., Pruitt B.A. Improved Survival of Burned Patients With Inhalation Injury. *Arch Surg* **128**,772-780,1993.

Saffle J.R. The 1942 Fire at Boston's Cocoanut Grove Nightclub. *Am.J. Surg*. **166**, 581-591,1993.

Scheulen J.J., Munster A.M. The Parkland Formula in Patients with Burns and Inhalation Injury. *J Trauma* 22,869-71.1982.

Shirani K.Z., Pruitt B.A., Mason A.D.,Jr. The Influence of Inhalation Injury and Pneumonia in Burn Mortality. *Ann Surg* **205**,82-87,1987.

Stone H.H.,Rhame D.W., Corbitt Y.D., Given K.S., Martin J.D.Respiratory burns. A correlation of clinical and laboratory results. *Ann Surg* **165**,157-168,1967.

Stone M.J. Assault by Burning and its Relationship to Social Circumstances. *Burns*. **14**,461-467,1988.

Thangaturai D., Holm A.P., Mikhail M., et al. HFV in Management of a Patient with Severe Bronchorrhea.*Resp Mgmt* Jan/Feb 31-33,1988.

Thomas H.V., Mueller P.K., Lyman R.L. Lipoperoxidation of Lung Lipids in Rats exposed to Nitrogen Dioxide. *Science*.**159**.532.1967.

Thompson P.B.,Herndon D.N., Traber D.L., Abston S. Effect on Mortality of Inhalation Injury.*J Trauma*.**26**,No2.163-165.1986.

Thorning D.R., Howard M.L., Hundson L.D., Schumacher R.L. Pulmonary responses to smoke inhalation; morphologic changes in rabbits exposed to pine wood smoke. *Hum*

Pathol **13**.355-64.1982.

Traber D.L., Herndon D.N., Stein M.D. et al. The Pulmonary Lesion of Smoke Inhalation in an Ovine Model. *Circ. Shock.* **18**,311-323,1986.

Traber D.L., Linares H.A., Herndon D.N. The Pathophysiology of Inhalation Injury - a Review. *Burns* **14**, 357-364,1988.

Trunkey, D.D. Inhalation Injury. *Surg Clin North Am* **58**.1133-40,1978.

Zikria B.A., Weston G.C.,Chodoff M.,Ferrer J.M.Smoke and carbon monoxide poisoning in fire victims. *J Trauma* **12**;641-645,1972.

Zikria B.A., Ferrer J.M., Floch H.F. The chemical factors contributing to pulmonary damage in "smoke poisoning".*Surgery* **71**, No 5. 704-09.1972.