RESPIRATORY TRACT DAMAGE IN BURNS

Case Reports and Review of the Literature

BY

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SUMMARY

Two case reports are presented of respiratory tract damage in a burn (RTDB) and the management of the cases is noted. It is suggested that the scarcity of reports in the British literature may in part be due to a lack of awareness of the problem and not entirely due to the rarity of the condition. Patients with burns of the face, especially if sustained in an enclosed space, should be observed for development of respiratory distress. If distress develops the patient should be treated in an area of respiratory intensive care. The literature on the subject has been reviewed as to the clinical and pathological features of RTDB and the recommended treatments. A scheme of management has been suggested.

Reports of damage to the respiratory system associated with burns are rare in Britain (Muir, 1966). The only cases reported in the British literature appear to be the babies involved in the Dellwood fire (Cox et al., 1955) and a recent case of carbon monoxide poisoning in a fire (Pollard, 1970). In a survey of 2,807 cases of burns from the Birmingham Burns Unit (Bull and Fisher, 1954) respiratory tract damage was mentioned by saying that “a few cases had respiratory tracts burned as well as body surface but no account was taken of this in the mortality figures”. The reason for the rarity in the U.K. as compared to North America (Muir, 1966; Editorial, 1967) is stated to be that in this country most burned victims run about breathing normal air till the flames are put out (Editorial, 1967). This lack of cases is somewhat difficult to understand when case reports and comments appear in the French (Mounier-Kuhn et al., 1968), German (Ganzoni, 1963; Müller, 1968), Swedish (Liljedahl, 1965), and Russian (Klyachkin et al., 1962; Katrushenko, 1965) journals as well as the many articles in the American literature. Within the last five years, in the south-eastern region of Scotland, there have been four known cases of respiratory tract damage causing severe respiratory problems in association with superficial burns. One child was managed without intermittent positive pressure ventilation (IPPV) and survived. The mother required IPPV and died (Howie, C. N., 1970, personal communication). The other two cases were treated in the Artificial Ventilation Unit of the Edinburgh Royal Infirmary and these are reported below.

CASE REPORT 1.

A 56-year-old female, who was a severe intractable chronic alcoholic, was found in a smoke-filled room of a burning building on the night of August 24, 1965. On admission to the Emergency Department of the Edinburgh Royal Infirmary she was comatose and had signs of pulmonary oedema. She appeared grossly neglected. An endotracheal tube was passed, the trachea aspirated, and frusemide 20 mg given intravenously. This cleared the oedema but there remained widespread bronchospasm which failed to respond to aminophylline. At 0000 hours on August 25 she was transferred to the Artificial Ventilation Unit, spontaneously breathing 100 per cent oxygen via a Waters canister. While being moved into bed she had a cardiac arrest. Following resuscitative measures, though her pupils were small, she remained unconscious. She was ventilated with a Smith-Clarke volume-preset time-cycled ventilator with water bath humidifier and approximately 75 per cent inspired oxygen, at a minute volume of 12 L/min and an inflation pressure of 25 cm H2O. At 08.30 hours she was not synchronizing with the ventilator and was hand-ventilated with a Waters canister for 30 min. Then, since the chest sounded less moist, she was allowed to self-ventilate on 100 per cent oxygen through the same circuit.

At 10.00 hours she had respiratory arrest accompanied by profound hypotension. Artificial ventilation was resumed, using the Smith-Clarke ventilator, at minute volumes of 10-16 L/min and inflation pressures of 15-18 cm H2O on 100 per cent oxygen. At these settings the...
blood-gases were $P_{aO_2}$ 99 mm Hg, $P_{aCO_2}$ 70 mm Hg, pH 7.24, St. bicarb. 24 m.equiv/l, and they remained virtually unchanged in spite of altering ventilator settings.

Her arterial pressure, which had been variable since admission, was restored with metaraminol but later became refractory to this drug and she died at 13.17 hours. During her stay in the unit the fluid intake was 5 per cent sodium bicarbonate 500 ml and 10 per cent levulose 750 ml. There was no urinary output nor did she regain consciousness.

Postmortem examination revealed the following:
(1) Severe congestion and oedema of the trachea and main bronchi with thin blood-stained pus.
(2) Severe congestion and oedema of both lungs, acute bronchitis and bronchiolitis in all areas and lower lobe collapse.
(3) Cerebral oedema.
(4) A grossly dilated heart with minimal patchy fibrosis.
(5) Fine portal cirrhosis.

CASE REPORT 2.

The patient, a 46-year-old female was admitted to the Edinburgh Royal Infirmary on March 2, 1970. Early that morning the fire brigade had found her lying unconscious in the smoke-filled hall of her house. The fire itself was confined to the living-room which had the windows and door shut. The patient regained consciousness when given oxygen by the ambulance men.

On admission she was noted to be suffering from extensive superficial burns of the face, neck and upper thorax, front and back, shoulders and hands, with involvement of the interior of the nares (figs. 1 and 2).

It was noted that mitral valvotomy had been performed 14 years previously and that therapy with digoxin had been continued since.

Soon after admission her condition deteriorated and the systolic arterial pressure fell to 60 mm Hg. For initial resuscitation she was given intravenously plasma 1500 ml, Ringer lactate 300 ml, 5 per cent levulose 400 ml and 5 per cent mannitol 500 ml. The arterial pressure rose to 110/70 mm Hg. Shortly thereafter the respiratory rate increased; the normal respiratory movements required a great deal of effort and involved the accessory muscles of respiration. Coarse crepitations were noted over the lung fields, consistent with a diagnosis of pulmonary oedema. A central venous pressure catheter was inserted and a reading of 24 cm H$_2$O obtained. Further fluids were not given and frusemide 40 mg was injected intravenously. She was conscious but irrational.

An endotracheal tube was passed (8.0 mm Portex) without anaesthesia and it was noted that the pharynx was reddened, and the larynx only slightly oedematous. On aspiration the tracheal secretions were found to contain particles of soot.

Ventilation was manually assisted using 100 per cent oxygen. Blood gases at that time (07.30 hours) were pH...
After the initial resuscitation of the patient, chest physiotherapy was started and repeated 3-4 times daily, combined with tracheal suction at first and then later with vigorous coughing. This minimized retention of secretions.

The radiological appearances, also confused by the mitral disease, showed a variety of transient signs: patchy atelectasis, a miliary mottling and pulmonary oedema (figs. 3-6). Acute gastric dilatation did not develop in this case.

The prevention of oliguria and renal failure and maintenance of fluid and electrolyte balance and nutrition.

This was again complicated by the mitral disease. She was catheterized, hourly urine volumes measured and frequent central venous pressure readings made. Her venous pressure fell rapidly from its initial high level. The urinary output was good and was maintained at over 50 ml/hour for the next 5 days by means of infusion of fluid and electrolytes to maintain a central venous pressure of 5-15 cm H2O. The fluids used are shown in table I. Even though the urine became discoloured with haemoglobin there was never any suggestion of oliguria.

A nasogastric tube was passed because of the nasal burn and the possibility of oesophageal burn damage. However, on March 5 an orogastric tube was passed and feeding commenced with 40 ml half-strength milk hourly, gradually increasing to a high protein liquid diet, in an effort to avoid a Curling's ulcer which seemed threatened. Thereafter oral feeding commenced and when her oral intake reached adequate amounts the orogastric tube was removed.

The treatment of her superficial burns and prevention of infection.

The burns to face, neck and shoulders were treated by exposure and the oedema settled. The hands, being worse, were dressed. Swabs were taken daily from her burns, which were sprayed with Polymyxin. Her nares rapidly became very purulent and culture revealed Staphylococcus aureus (penicillin sensitive). This was treated by local cleansing and application of topical framycetin. The urine remained sterile. From the tracheal secretions, which became purulent in 24 hours, no organisms were isolated on smear or culture, but there were many carbon particles and these continued to be coughed up even when she was discharged from the unit. No steroids were given systemically or by inhalation and the conscious decision was taken to give no antibiotics systemically or per trachea until an organism was isolated in significant numbers. Every effort was made to isolate potential pathogens. Daily cultures were made from nasal swabs, tracheal secretions, catheter specimens of urine and frequent blood cultures. In addition the bacteriologist, with whom early contact was established and who took special interest in the case, laid out culture plates daily in the room to try to isolate any organisms in the air. In the event no systemic antibiotics at all were given while she was in the unit and it was only after the endotracheal tube had been removed that S. aureus was isolated in the sputum and even then in numbers of doubtful significance. Pneumococcus and Haemophilus influenzae were isolated on one occasion each.

As her physical condition improved so her mental state deteriorated, confusion and disorientation developing each evening. This was considered to be intensive care psychosis, the more likely since she was completely unconscious each evening. The respirator was turned off for an hour each evening and ventilatory assistance withdrawn to allow the patient a period of ventilator-free ventilation.

The management of the respiratory distress and the respiratory damage.

She was admitted to the Artificial Ventilation Unit where at first it was found impossible to ventilate the lungs mechanically despite depression of her own respiratory drive by means of phenoperidine 3 mg i.v. followed in an hour by papaveretum 10 mg i.v. However, aminophylline given i.v. on several occasions did ease what was by now predominantly bronchospasm with no audible crepitations. After hand-ventilation through a Waters canister for an hour she was connected to a Cape ventilator with water bath humidifier which was now able to ventilate the lungs at an inflation pressure of 28 cm H2O. Ventilation was continued with 100 per cent oxygen at a minute volume of 9 l/min; her blood gases at 1150 hours were pH 7.39, PaO2, 56 mm Hg, St. bicarb. 33 m.equiv/1. O2 sat 100 per cent. At this point the patient's management could be broken down into several facets.

1. The management of the respiratory distress and the respiratory damage.
2. The prevention of oliguria and renal failure and maintenance of fluid, electrolyte and nutrition.
3. The treatment of her superficial burns.
4. The prevention of infection.

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FIG. 3
Day 2, 9 a.m. The cardiac shadow is enlarged with biventricular and left atrial enlargement. The pulmonary arteries are also prominent. These findings are in keeping with chronic mitral disease. There is an area of well developed consolidation in the left mid-zone. In addition there is coarse interstitial oedema in the right mid-zone shown as horizontal septal lines. These are not present at the lung bases nor are they present in previous films or ones taken after discharge and are therefore unlikely to be due to the mitral disease.

FIG. 4
Day 4, 9 a.m. The area of consolidation in the left mid-zone has extended to involve the lingula and become more dense. There are now signs of pulmonary oedema at the bases with septal lines visible at the right base. The interstitial oedema is still present at the right mid-zone but is partially obscured by the development of an area of consolidation in the same area. In the upper zones of both lungs there is the development of a mottled appearance which may be due to small areas of atelectasis or alveolar oedema.

FIG. 5
Day 6, 9 a.m. The areas of consolidation in the right and left mid-zones are starting to clear. There is still interstitial oedema at the right base and to a lesser extent at the right mid-zone. The mottling in the upper zones has, however, increased in the area of each opacity.

FIG. 6
Day 7, 12 noon. There is further clearing of the areas of consolidation and the mottling in the upper zones is also clearing. There is still interstitial oedema at the right base and there is also now evidence of right basal collapse; this subsequently cleared.
RESPIRATORY TRACT DAMAGE IN BURNS

Table I
The intravenous fluid treatment administered to Case No. 2.

<table>
<thead>
<tr>
<th>Date</th>
<th>Ringer lactate</th>
<th>Lactulose 5%</th>
<th>Lactulose 10%</th>
<th>Normal saline</th>
<th>Plasma</th>
<th>Rhamnulose</th>
<th>Sodium bicarbonate</th>
<th>Potassium chloride</th>
<th>Total vol.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>2500</td>
<td>350</td>
<td>500</td>
<td>800</td>
<td>400</td>
<td>500</td>
<td>150</td>
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<td>4,800</td>
</tr>
<tr>
<td>2</td>
<td>2000</td>
<td>1000</td>
<td>400</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>3,400</td>
</tr>
<tr>
<td>3</td>
<td>1800</td>
<td>2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>3,000</td>
</tr>
<tr>
<td>4</td>
<td>500</td>
<td>1500</td>
<td>1500</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>3,500</td>
</tr>
<tr>
<td>5</td>
<td>1500</td>
<td>1000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>2,500</td>
</tr>
</tbody>
</table>

isolated in a single room and barrier-nursed. Since ventilation care was no longer needed she was transferred to a surgical ward in the hospital from where, 48 hours later, she was transferred to the Regional Burns Unit at Bangour General Hospital. During this short period, in spite of frequent vigorous physiotherapy, her condition deteriorated and by the time she reached the Burns Unit she was semi-conscious and cyanosed due to retention of secretions and dehydration. However, intensive treatment over several days with oxygen, antibiotics, frequent vigorous physiotherapy and oral rehydration reversed the deterioration and she became conscious, rational and able to do things for herself while the burns healed. Her subsequent recovery was uneventful.

DISCUSSION

On November 28, 1942, a fire started in the Cocoanut Grove, a Boston night club; 491 people lost their lives; 114 casualties were taken to the Massachusetts General Hospital and of these 75 were either dead on arrival or died of anoxia within minutes (Aub, Pittman and Brues, 1943); 39 survived long enough to be treated and of these only 3 were entirely without respiratory symptoms (Aub, Pittman and Brues, 1943). This seems to have been the first time that it was realized that patients suffering burns could die, not from the burn, but from associated damage to the respiratory tract (Beecher, 1943). At this time the pulmonary lesions were unexpected (Cope and Rhinelander, 1943). This disaster was very fully documented (Aub, Pittman and Brues, 1943; Beecher, 1943; Cannon, B., 1943, 1944; Cannon, L., 1943; Cobb and Lindemann, 1943; Cope, 1943a, b, c; Cope et al., 1943; Cope and Rhinelander, 1943; Faxon, 1943; Faxon and Churchill, 1942, 1943; Lyons, 1943; Mallory and Brickley, 1943; Moore, 1943; Editorial, 1943; Schatzki, 1943; Soutter, 1943; Watkins, 1943).

Therefore a steady stream of reports were published citing respiratory damage as a major cause of death in burns (Baxter and de Crosse, 1958; Foley, 1969; Jackson and Lee, 1963; Phillips and Cope, 1960, 1962a, b; Phillips, Tanner and Cope, 1963; Shook, MacMillan and Altemeier, 1968; Socher and Mallory, 1963; Yates et al., 1958) and the term "pulmonary burn" crept into the literature. In 1945 Moritz, Henriques and McLean published experimental work on dogs in which they showed that when hot air was blown into the larynx at 270°C the temperature of the air fell to 50°C in the trachea. Hot air and flame could burn the larynx and trachea but failed to produce pulmonary damage. Stone and his colleagues (1967) while endeavouring to cause pulmonary burns had to resort to the totally unrealistic means of insufflating steam through a chilled, plastic, intratracheal tube. These experiments and others (Shook, MacMillan and Altemeier, 1968; Aviado, 1959; Aviado and Schmidt, 1952) tend to disprove the theory that pulmonary burns are caused by heat.

Details of the pathological findings were published after the Cocoanut Grove fire (Mallory and Brickley 1943), the main findings being a diffuse membranous bronchitis (similar to that found in inhalation of nitrogen dioxide and the war gases, phosgene, mustard gas and chlorpicrin) and massive pulmonary oedema. These findings are the same as found in Case 1. Subsequent reports from other centres (Foley, 1969; Klyachkin et al.,
amplified the findings associated with death due to "pulmonary burns" and it seemed that all the pathological ramifications had been explored when Taylor and Gumbert (1965) took a critical look at the "typical pulmonary burn pathology" and found that all the features could be found in other terminal states, such as peritonitis or status asthmaticus, or could be caused by the treatment, for example tracheostomy or inhalation of high concentrations of oxygen. This recalled the original Cocoanut Grove findings that were reported as being similar to the changes caused by noxious gases (Mallory and Brickley, 1943). This was a very shrewd observation since most authors (Editorial, 1967; Ganzoni, 1968; Jackson and Lee, 1963; Klyachkin et al., 1962; Liljedahl, 1965; Mounier-Kuhn et al., 1963; Müller, 1968; Phillips, Tanner and Cope, 1963; Reed and Camp, 1969; Stone, 1968; Travis, Armbrister and Heironimus, 1967) but not all (Ollstein, Symonds and Crikelair, 1968) now agree that the damage distal to the trachea and main bronchi is caused not by heat but by smoke or the incomplete products of combustion carried in the smoke. Widdicombe, Kent and Nadel (1962) found that bronchospasm could be induced by the inhalation of granulated charcoal dust. They also showed that, though this was a reflex spasm mediated through the vagus, chemical irritants caused decrease in airway size, even when the vagus was blocked. In a fire in an enclosed space there are probably two mechanisms causing bronchospasm, namely the carbon particles in the smoke and the chemical irritant.

The terms "pulmonary burn", "inhalation burn" or "tracheobronchial burn" are all misnomers and should be replaced by "respiratory tract damage in burns" (RTDB). The problem then arises as to how respiratory tract damage is diagnosed in a burned patient. The first obvious factor is for the clinician to have a high index of suspicion (Stone, 1968). Three criteria have been given for the diagnosis of RTDB. These are:

1. flame burns involving the face, principally the mouth and nose (Ganzoni, 1968; Klyachkin et al., 1962; Moritz, Henriques and McLean, 1945; Mounier-Kuhn et al., 1968; Phillips and Cope, 1962b; Phillips, Tanner and Cope, 1963; Reed and Camp, 1969; Stone, Martin and Claydon, 1967; Stone and Boswick, 1968);

2. singed nasal vibrissae (Stone, Martin and Claydon, 1967);


Stone, Martin and Claydon (1967), however, found that patients satisfying two of the three criteria had clinical courses similar to those with all three, albeit with a lower mortality: 44.8 per cent compared with 71.2 per cent of those satisfying all three. The mortality found by various authors is shown in table II.

Only about 5 per cent of patients admitted to hospital in Britain with domestic burns have received them in conflagrations, and pulmonary complications being uncommon, may be missed (Editorial, 1967). Since the history of how the burns were sustained is not always immediately available and, since singed nasal vibrissae may be missed on initial examination, the presumptive diagnosis of RTDB must be made on any patient having a burn of the face which includes the mouth or nose, until time has proved otherwise. If, however, the history contains any reference to "a smoke-filled room" RTDB is probable.

Clinical signs of damage to the respiratory tract may develop early (Beal, Lambeth and Conner, 1968; Reed and Camp, 1969; Stone and Martin, 1969) or late (Reed and Camp, 1969; Stone, Martin and Claydon, 1967) and are very variable, the most common being bronchospasm and rales due to pulmonary oedema. In RTDB, as in severe asthmatics (Riding and Ambiavagar, 1967), a decrease in the adventitial sounds may be a sign of deterioration due to a decrease in tidal volume and not an improvement (Phillips, Tanner and Cope, 1963). This was undoubtedly the situation in Case 1.

The radiological changes which have been reported as specific for RTDB are:

1. Linear densities which are supposed to be due to local atelectases. These densities, however, are "flitting", i.e. they may shift from lobe to
TABLE II

Mortality in patients with respiratory tract damage in burns (RTDB) reported by various groups.

<table>
<thead>
<tr>
<th>Numbers with RTDB</th>
<th>Treated with IPPV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
</tr>
<tr>
<td>Shook, MacMillan and Altemeier (1968)</td>
<td>65</td>
</tr>
<tr>
<td>Travis, Armbriester and Heironimus (1967)</td>
<td>1</td>
</tr>
<tr>
<td>Donellan, Poticha and Holinger (1965)</td>
<td>1</td>
</tr>
<tr>
<td>Cox et al. (1955)</td>
<td>14</td>
</tr>
<tr>
<td>Mournier-Kuhn et al. (1968)</td>
<td>3</td>
</tr>
<tr>
<td>Beal, Lambeth and Conner (1968)</td>
<td>29</td>
</tr>
<tr>
<td>Stone and Martin (1969)</td>
<td>197</td>
</tr>
<tr>
<td>Klyachkin et al. (1962)</td>
<td>31</td>
</tr>
<tr>
<td>Karrushenko (1965)</td>
<td>62</td>
</tr>
<tr>
<td>Schenk et al. (1955)</td>
<td>4</td>
</tr>
<tr>
<td>Pollard (1970)</td>
<td>1</td>
</tr>
<tr>
<td>Gorman et al. (1968)</td>
<td>2</td>
</tr>
</tbody>
</table>

A large number ventilated; no detailed figures given
Some ventilated; no detailed figures given
No information given on ventilation; probably none treated by IPPV
Treated with hyperbaric oxygen with good results; no IPPV

lobe or vanish with effective coughing (Phillips, Tanner and Cope, 1963; Travis, Armbrister and Heironimus, 1967). In addition to the densities there is vascular crowding, elevation of the diaphragm and displacement of the hilum (Phillips, Tanner and Cope, 1963; Travis, Armbrister and Heironimus, 1967).


(3) Miliary mottling (Schatzki, 1943).

(4) Pulmonary oedema (Phillips, Tanner and Cope, 1963; Schatzki, 1943).


(6) Areas of “drowned lung” (Schatzki, 1943). These are areas with homogeneous ground-glass appearance.


A single normal radiograph is of no significance (Editorial, 1967; Klyachkin et al., 1962; Mournier-Kuhn et al., 1968; Stone, Martin and Claydon, 1967; Stone and Boswick, 1968) since radiological signs are variable in onset (Reed and Camp, 1969). It may be only in retrospect that the radiological features described in RTDB become apparent. This means that all these patients must be observed carefully and blood-gas analysis carried out on the first sign of restlessness, confusion, disorientation which may be due to respiratory failure (Editorial, 1967; Phillips, Tanner and Cope, 1963; Reed and Camp, 1969) or the development of clinical signs of respiratory distress. Cyanosis is an unreliable sign since it may be masked by anaemia or carbon monoxide poisoning (Editorial, 1967; Phillips, Tanner and Cope, 1963; Pollard, 1970).

Once the diagnosis of RTDB has been made there comes the problem of management. When respiratory failure is evident and confirmed by blood-gas analysis, oxygen must be administered. Most authors recommend 40 per cent oxygen with 80–100 per cent humidity given by means of a head tent (Cox et al., 1955; Editorial, 1967; Reed and Camp, 1969; Stone, 1968; Stone, Martin and Claydon, 1967; Stone et al., 1967). One author treated his cases with hyperbaric oxygen and reported good results (Gorman and Regent, 1965; Gorman et al., 1968).

If, however, there is severe bronchospasm, as in the cases reported here and by Donellan, Poticha and Holinger (1965) and Travis, Armbrister and Heironimus (1967), further treatment is required. Since the bronchospasm is reported to be refractory to treatment with normal bronchodilators (Donellan, Poticha and Holinger, 1965; Stone, Martin and Claydon, 1967; Stone and Martin, 1969), some authors have recommended intravenous injection of steroids in high doses (Donellan, Poticha and Holinger, 1965; Shook,
MacMillan and Altemeier, 1968; Stone et al., 1967) as in the treatment of status asthmaticus, or via the nebulizer (Donellan, Poticha and Holinger, 1965; Shook, MacMillan and Altemeier, 1968). Steroid treatment may relieve bronchospasm (Beal, Lambeth and Conner, 1968; Stone et al., 1967), decrease the inflammatory oedema of small bronchi (Beal, Lambeth and Conner, 1968; Donellan, Poticha and Holinger, 1965), maintain surfactant (Beal, Lambeth and Conner, 1968) and prevent atelectasis (Donellan, Poticha and Holinger, 1965) and pneumonitis (Donellan, Poticha and Holinger, 1965). Steroids have also been given as a last resort in desperate circumstances (Stone and Boswick, 1968). However, many workers have noted that steroids increase the incidence, rate of onset, and virulence of infection (Donellan, Poticha and Holinger, 1965; Shook, MacMillan and Altemeier, 1968; Stone, Martin and Claydon, 1967), may increase the damage caused by tracheostomy, and do not prevent chronic pulmonary changes (Beal, Lambeth and Conner, 1968; Donellan, Poticha and Holinger, 1965).

Since, in status asthmaticus, the reponsiveness to bronchodilator treatment may be restored by correcting the acidosis by means of intravenous bicarbonate (Mithoefer, Porter and Karetzky, 1968), in cases with RTDB with bronchospasm, this should be done before resorting to steroids. (The responsiveness of the bronchospasm to bronchodilators was restored by this means in Case 2.)

Stone's recommendation (Stone, 1968; Stone and Martin, 1969; Stone, Martin and Claydon, 1967) that a single "shot" of a massive dose of corticosteroid be given intravenously in a final effort to relieve bronchospasm is logical since corticosteroid "weaning" is not necessary and the hazards of infection are decreased.

In status asthmaticus IPPV is recommended (Rees, 1967; Rees, Millar and Donald, 1968) if the PaO₂ is greater than 60 mm Hg on admission, or if it is between 50-60 mm Hg on admission and does not fall below 50 mm Hg in the first 8 hours. With tracheal intubation, as distinct from tracheostomy, the indications were widened to include patient exhaustion in the presence of normal blood gases (Rees, 1967; Rees, Millar and Donald, 1968). It would seem logical in RTDB to recommend the institution of IPPV (Beal, Lambeth and Conner, 1968) when there is evidence of pulmonary insufficiency, which some believe to be almost uniformly fatal (Reed and Camp, 1969), and to apply the same criteria as in status asthmaticus in deciding when and if to undertake passive ventilation.

IPPV in patients with RTDB has been condemned on four grounds. First, that it may cause "shock" (Beecher, 1943; Griffiths, 1960) by decreasing the venous return to the heart. Secondly, that it does not help to control pulmonary oedema, to overcome bronchospasm (Travis, Armbrister and Heironimus, 1967) or to overcome hypoxia. Thirdly, that ventilators are heavily contaminated by virulent hospital organisms and merely serve to inoculate the lungs with bacteria (Stone, 1968; Stone Martin and Claydon, 1967). It has been stated that the most severe pneumonias occurred in cases where IPPV was instituted early (Stone and Martin, 1969; Stone et al., 1967). Fourthly, that oxygen toxicity (congestion, oedema, collapse, tracheal irritation and decrease of diffusing capacity) is increased (Foley, 1969).

The first argument was used against IPPV therapy of crushed chest injury but Griffiths (1960) found that this was only true if the pressure in the trachea was excessive, if the inflation phase of the cycle was prolonged or if the patient was breathing out of cycle with the machine. The same argument was encountered when IPPV was suggested for severe status asthmaticus, but Riding and Ambiavagar (1967) in resuscitating moribund asthmatics found that the high inflation pressures (+70 to -20 cm H₂O) needed were dissipated in overcoming the bronchial obstruction and were therefore not transmitted to the vascular bed. They also found that hypotension was not produced, provided fluid replacement was adequate as judged by central venous pressure. IPPV also acted by reducing the grossly abnormal intrathoracic pressure fluctuations which occur in the asthmatic during spontaneous breathing.

The second part of the argument may, in part, be supported by the fact that the "oedema" is partly protein exudate (Müller, 1968), due to the chemical pneumonitis, and is not merely transudate. Nevertheless IPPV is now recognized as being the only useful treatment if pulmonary oedema has developed (Beecher, 1943; Editorial,
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1967; Reed and Camp, 1969; Stone, 1968; Stone and Martin, 1969; Stone et al., 1967) and Riding and Ambiavagar (1967) found that IPPV could overcome the bronchospasm sufficiently to correct hypoxia and hypercarbia in the severe asthmatic. Travis, Armbrister and Heironimus (1967) found that volume-cycled IPPV was the only means of alleviating the bronchospasm in their case.

The third objection is the most valid and it has been suggested that ventilators should be changed every 6–8 hours and cleaned or preferably sterilized with ethylene oxide (Stone, 1968). In Case 2 there was no evidence of pulmonary infection during ventilation. This may in part be due to the use of a water bath humidifier in this case and not nebulizers, as described in other reports (Donellan, Poticha and Holinger, 1965; Shook, MacMillan and Altemeier, 1968). (Nebulizers are more prone to carry infection than are water bath humidifiers (Chamney, 1969). This point is discussed later.) The fourth point, namely that oxygen toxicity is increased by IPPV, has been disproved by careful measurement by de Lemos and associates (1969).

Once the decision has been made to institute IPPV the next problem is the route of administration. EPPV via face mask, though recommended for occasional use (Stone, Martin and Claydon, 1967), can rarely be of value because of the impossibility, in the presence of laryngeal or bronchial obstruction of inflating the lungs without inflating the stomach and because of the difficulty of achieving an airtight seal on a burned face.

There remains the choice between endotracheal intubation and tracheostomy. While some authors recommend early tracheostomy as essential (Beal, Lambeth and Conner, 1968; Epstein et al., 1963; Jackson and Lee, 1963; Klyachkin et al., 1962; Liljedahl, 1965; Mounier-Kuhn et al., 1968; Muir, 1965; Stone and Boswick, 1968) others feel that, at least in the initial stages, this procedure carries more dangers than benefits. Thus it may precipitate pulmonary oedema, sometimes fatal (Reed and Camp, 1969; Stone and Martin, 1969; Stone, Martin and Claydon, 1967; Stone et al., 1967) or bronchospasm (Donellan, Poticha and Holinger, 1965). In addition a tracheostomy provides an ever-open route for infection to enter the lungs (Donellan, Poticha and Holinger, 1965; Ganzoni, 1968; Reed and Camp, 1969; Stone and Boswick, 1968; Teplitz et al., 1964). Amongst complications mentioned are necrotizing tracheitis (Teplitz et al., 1964), respiratory obstruction with the cannula impinging on the carina or intubating a bronchus (Epstein et al., 1963; Travis, Armbrister and Heironimus, 1967), erosion or perforation of the trachea (Moncrieff, 1959) or displacement of the tracheostomy tube due to oedema of the neck (Moncrieff, 1959).

It has generally been found that prolonged endotracheal intubation in adults is certainly safe for up to 3 days (Lindholm, 1969) and there is no absolute time limit after which endotracheal intubation must be discontinued. Lindholm (1969) also found that the complications of prolonged endotracheal intubation were no greater than those of tracheostomy. In Case 2 a tracheostomy was not considered because the site of election for tracheostomy lay within the burned area and it was felt that the combination of raw burn plus tracheal secretions would offer an ideal culture medium for bacteria immediately adjacent to the damaged trachea (Stone and Boswick, 1968). (Nasotracheal intubation, used by some (Reed and Camp, 1969; Stone, Martin and Claydon, 1967) was not considered because of the nasal burn.) An orotracheal tube was therefore passed. Aspiration of the tracheal secretions is more difficult and cannot be as vigorous as via a tracheostomy but this may be to the patient’s benefit, since over-vigorous tracheal aspiration can remove remaining islets of undamaged mucosa from which repair occurs (Phillips, Tanner and Cope, 1963). Some authors recommend bronchoscopy as an essential part of the removal of secretions (Klyachkin et al., 1962; Mounier-Kuhn et al., 1968; Walder et al., 1967) but other authors make scant use of it, relying instead on adequate humidification and endotracheal suction. In Case 2 bronchoscopy was considered in the initial period but rejected because of the danger of further damage to the trachea and because it was felt that even a brief period of hypoxia during the procedure might prove fatal.

The ventilator used was a volume-preset time-cycled machine (Cape). This was the only ventilator in the unit capable of reaching the pressures required to ventilate the patient’s lungs. Other authors have used pressure- and volume-cycled machines (Shook, MacMillan and Altemeier,
One problem in patients with RTDB appears to be progressive atelectasis. Shook, MacMillan and Altemeier (1968) suggest that this is due to increased metabolic demands, with apprehension causing rapid shallow breathing resulting in early atelectasis. This may be mediated through loss of pulmonary surface active forces. Matsuura, Najib and Lee (1966) found that lung surfactant function was decreased after any burn, that this decrease started 1 hour after a burn and was significant after 72 hours. It was also decreased by a transfusion of plasma and this effect was enhanced after a burn. Surfactant function is also decreased by circulating fatty acids, especially oleic and linoleic (Matsuura, Najib and Lee, 1966). It is tempting to speculate whether the fat embolism present in some burned patients (Shook, MacMillan and Altemeier, 1968) may contribute to the respiratory difficulties by an effect on the surface active forces as well as by direct effect on the lungs.

Other factors found to decrease surfactant function were respiratory acidosis, exposure to oxygen and the presence of intra-alveolar fluid, whether pulmonary oedema or administered per trachea (Avery and Said, 1965). Constant-volume ventilation has been suggested as a cause of progressive atelectasis causing decreased compliance and a fall in arterial oxygen tension (Avery and Said, 1965; Bendixen, Hedley-Whyte and Laver, 1963; Bernstein, 1957; Egbert, Laver and Bendixen, 1963; Ferris and Pollard, 1960; Mead and Collier, 1959) but other authors failed to confirm this finding (Askrog et al., 1964; Emerson, Forres and Lyons, 1960; Lumley, Morgan and Sykes, 1969; Morgan, Lumley and Sykes, 1970; Nunn, Bergman and Coleman, 1965; Sykes, Young and Robinson, 1965; Theye and Tuohy, 1965) and the present position is still controversial. All these factors may be present as part of the symptomatology of RTDB or as part of the treatment of the burn itself or the RTDB. The only methods available to improve the surfactant function and increase the pulmonary compliance are reduction of the P\textsubscript{1\textsubscript{02}} (Avery and Said, 1965), the administration of steroids (Beal, Lambeth and Conner, 1968) and periodic hyperinflation (Avery and Said, 1965; Bendixen, Hedley-Whyte and Laver, 1963; Bernstein, 1957; Egbert, Laver and Bendixen, 1963; Ferris and Pollard, 1960; Mead and Collier, 1959). It may not be possible initially to reduce the P\textsubscript{1\textsubscript{02}} but this may not be so important in these cases since Ohlson (1947) found that rabbits, which normally develop oxygen toxicity when the inspired oxygen content is 80–90 per cent, are protected by the prior inhalation of diphosgene which causes respiratory tract damage pathologically similar to RTDB (Mallory and Brickley, 1943). Periodic hyperinflation may be provided by hand-ventilation (Shook, MacMillan and Altemeier, 1968; Travis, Armbrister and Heironimus, 1967), automatically by the ventilator, or by voluntary effort by the patient (Lindholm, 1969; Phillips, Tanner and Cope, 1963). High ventilation volumes may also prevent the decrease in P\textsubscript{a\textsubscript{02}} (Hedley-Whyte et al., 1964, 1965) and this has been recommended in the treatment of RTDB (Travis, Armbrister and Heironimus, 1967). However, the value of periodic hyperinflation and high ventilation volumes have been disputed (Morgan, Lumley and Sykes, 1970). In Case 2 periodic hyperinflation appeared to be the most satisfactory single factor in treatment.

Humidification is another important factor in preventing drying of the secretions. Most reports refer to the use of nebulizers, gas-driven and ultrasonic, with the addition of various medicaments. However, they have many disadvantages. They are difficult to sterilize and often produce a cloud of bacteria (Chamney, 1969). Paradoxically their design (to produce droplets of 1–5 \textmu for adequate passage into the alveoli) will in these circumstances allow the carriage of organisms (American Thoracic Society, 1967b; Edmondson et al., 1966; MacPherson, 1958; Moffet and Williams, 1967; Pierce and Sanford, 1966; Reinarz et al., 1965) to areas beyond the range of the normal ciliary mechanism of removing foreign matter (Chamney, 1969). The concomitant problem is that fluid passing to the alveoli may diminish surfactant function (Matsuura, Najib and Lee, 1966) or alter the surface tension there by other means (Modell, Heinirsh and Gianna, 1969). Normal saline may cause severe haemorrhagic pneumonia in 72 hours (Shook, MacMillan and Altemeier, 1968) in puppies (Modell, Giannma and Davis, 1967) and arterial desaturation in tracheotomized adult dogs (Modell et al., 1968). Distilled water causes much less severe changes in animals (Chamney,
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Fluid overload is also possible with nebulization (Glover, 1965; Harris and Riley, 1967; Herzog, Norlander and Engstrom, 1964; Smith, 1966; Pratt, 1967) and it has been suggested that organisms may be disseminated beyond the immediate environment of the patient (Corn and Flynn, 1967). The water bath humidifier on the Cape ventilator provides less efficient humidification but there was no problem with drying of the secretions. Further, there was no infection in the respiratory tract while ventilation was in progress. Several authors have found that the output from water bath humidifiers is no more highly contaminated than is the ambient air (Edmondson et al., 1966; Moffet and Williams, 1967; Reinarz et al., 1965; Schultze et al., 1967) though there is one report of heavy contamination of tubing and ventilators with Pseudomonas aeruginosa (Phillips and Spencer, 1965).

It has been suggested that antibiotics should be administered prophylactically by nebulizer (Shook, MacMillan and Altemeier, 1968) and/or systemically (Beal, Lambeth and Conner, 1968; Ganzoni, 1968; Liljedahl, 1965; Mounier-Kuhn et al., 1968; Stone and Boswick, 1968; Travis, Armbrister and Heironimus, 1967). Others condemn prophylactic treatment on the grounds that it predisposes to the emergence of resistant organisms (Reed and Camp, 1965; Stone, 1968; Stone et al., 1967). Since the latter view is held in this centre no antibiotics were given in Case 2 and none were needed until well after the 7 days by which time the development of bronchopneumonia is considered to be inevitable (Ganzoni, 1968; Katrushenko, 1965; Klyachkin et al., 1962; Muir, 1966; Müller, 1968; Reed and Camp, 1965; Stone, 1968; Stone and Martin, 1969; Stone, Martin and Claydon, 1967; Stone et al., 1967). However, it is most important to search for any infection by culturing tracheal secretions, urine, swabs from burned surfaces, and blood cultures, which last has been shown to be a valuable method of isolating pathogens in burned patients (Foley, 1969; Stone, Martin and Claydon, 1967). A variety of ingredients have been instilled down the trachea: steroids (Donnellan, Poticha and Holinger, 1965; Phillips and Cope, 1969), antibiotics (Shook, MacMillan and Altemeier, 1968), heparin (Liljedahl, 1965; Shook, MacMillan and Altemeier, 1968), proteolytic enzymes (Liljedahl, 1965; Travis, Armbrister and Heironimus, 1967) and isoprenaline (Donnellan, Poticha and Holinger, 1965) but none has been shown to have a definite beneficial effect.

Some authors think that late tracheostomy is essential (Reed and Camp, 1965; Stone, 1968; Stone et al., 1967) to deal with the bronchorrhoea which appears after 4–8 days (Pollard, 1970; Reed and Camp, 1965; Stone, 1968; Stone, Martin and Claydon, 1967) but adequate intensive respiratory care with vigorous chest physiotherapy may eliminate the need (Travis, Armbrister and Heironimus, 1967). In case 2 the probable reason for the deterioration before and after her transfer from the AVU was the unrecognized onset of bronchorrhoea, although the transfer took place on the 10th day. In view of this experience it may be necessary to keep patients with RTDB in an area of intensive respiratory care for at least 14 days.

Fluid needs to be given to compensate for that lost by ooze from the burn and tracheal exudates (Katrushenko, 1965) and also to compensate for the fluid sequestered in the skin and subcutaneous tissues. Enough fluid should also be given to maintain a high urinary output to deal with the haemolysis (Stone and Boswick, 1968; Travis, Armbrister and Heironimus, 1957) which occurs after burns—more extensive in RTDB than skin damage (Aviado and Schmidt 1952)—and which, in the presence of oliguria, may precipitate anuria and/or renal failure. However, these needs have to be balanced against the risks of pulmonary oedema which is very likely to occur in patients with RTDB, especially those who also have some cardiac disorder (Stone, Martin and Claydon, 1967). A number of methods of balanced fluid replacement (Schenk et al., 1955), or “rules of thumb” (Ganzoni, 1968; Phillips, Tanner and Cope, 1963; Stone, Martin and Claydon, 1967) have been recommended but the most rational would seem to be to insert a central venous catheter and to balance the fluid input by means of frequent central venous pressure measurements (Shook, MacMillan and Altemeier, 1968; Stone, 1958; Stone and Boswick, 1968) and hourly urine
output measurements (Schenk et al., 1955; Stone and Boswick, 1968) since this eliminates guesswork. If there is oliguria, mannitol may be used to promote an adequate urine volume (Stone and Boswick, 1968). Stone (1968), incidentally, recommends that the central venous pressure catheter should be removed after 48 hours on the grounds that the majority of them are contaminated and may lead to septicemia and pulmonary embolism. The risk of infection must, in each case, be balanced against the risk of pulmonary oedema.

RECOMMENDATIONS
(1) Any patient who suffers burns of the face, especially round the nose or mouth, should be observed for signs of respiratory distress. A history of the patient being found in a smoke-filled room makes some degree of RTDB almost inevitable.

(2) At the first suggestion of respiratory distress arterial blood-gas analysis should be undertaken.

(3) Hypoxia should be treated by giving high inspired percentages of oxygen with adequate humidification.

(4) An arterial carbon dioxide tension exceeding 60 mm Hg, or rising tensions, is an indication for the institution of IPPV which should be via endotracheal tube whenever possible. Sterile precautions must be strictly observed.

(5) Pulmonary ventilation, if required, should be volume-controlled, using high volumes.

(6) Water bath humidifiers may be safer than nebulizers for humidification.

(7) Periodic hyperinflation of the lungs may be valuable.

(8) Physiotherapy to the chest should be both vigorous and frequent.

(9) Careful control of fluid balance is necessary, particularly if there is co-existing cardiac disease.

(10) Antibiotics need not be given prophylactically but blood cultures should be taken and cultures made of secretions. If an organism is cultured the appropriate antibiotic should be given without waiting for clinical signs of infection to develop.

(11) Patients with RTDB should be kept in an area of intensive respiratory care for 14 days to avoid trouble with the late onset of bronchorrhoea.

ADDENDUM
Since preparation of this article, Case 2 has died. She collapsed at home on July 9, 1970, and died 14 hours after admission to hospital. Postmortem examination showed:

(1) Blockage of the mitral valve orifice with massive thrombus of the left atrium.

(2) Rheumatic heart disease with mitral stenosis and incompetence.

(3) Right lower lobe pneumonia with tracheo-bronchitis.

(4) Pulmonary oedema.

(5) Brown induration of the lungs.

Findings (4) and (5) were both due to the chronic mitral disease and there were no pathological findings in the lungs which could be due to the previous RTDB.

ACKNOWLEDGEMENT
We would like to thank Dr A. S. Crawford for permission to publish details of Case 1.

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