CLINICAL ANALYSIS OF CASES ADMITTED DURING THE FIRST YEAR'S EXPERIENCE IN THE RESPIRATORY RESUSCITATION UNIT OF THE JOHANNESBURG HOSPITAL

S. R. LYNCH, M.B., B.CH. (RAND); D. P. STABLES, M.B., B.CHIR. (CANTAB); A. W. W. VAN AS, M.B., B.CH. (RAND); A. H. RUBENSTEIN, M.B., B.CH. (RAND), F.C.P. (S.A.); AND G. A. ELLIOTT, M.D. (CAPE TOWN), F.R.C.P. (LOND.); *Department of Medicine, University of the Witwatersrand and the Johannesburg Hospital, and the Cardio-Pulmonary Research Unit of the Council for Scientific and Industrial Research*

The Respiratory Resuscitation Unit of the Johannesburg Hospital was formally established in December 1963. The administration of the unit has been described in a previous publication.[1] During the first year (December 1963 — December 1964) 70 cases were admitted to its 3 beds. Almost as many patients who in general were less severely ill were treated outside the unit on a consultative basis by the medical staff of the unit. They are not included in this survey. It would have been preferable to have transferred the more ill of these cases to the unit, particularly those on mechanical ventilation, but shortage of nursing staff has precluded the opening of the second specially designed 3-bed ward.

SOURCE AND NATURE OF MATERIAL

The Respiratory Unit of the hospital, together with a similar unit in the Department of Thoracic Surgery,[2]

TABLE I.

Diagnosis	*No. of cases*	*Average duration of stay in unit (days)*	*No. of survivors*
Intrinsic chest disease			
Medical	25	9	14
Postoperative pneumonia ..	3	6	1
Traumatic	16	21	13
Neurological disease with respiratory failure			
Drug coma	20	6	18
Head injury	1	1	0
Cerebrovascular accident ..	2	1	0
Muscular paralysis ..	3	14	0
Total	70	11	46

TABLE II. INTRINSIC CHEST DISEASE

Diagnosis	No. of cases	Average age and range (years)	Previous pulmonary disease (no. of cases)	Average duration of tracheostomy (days)	Type of respirator used: Bird — No. of cases	Bird — Average time required on respirator per patient (days)	Engström — No. of cases	Engström — Average time required on respirator per patient (days)	No. of survivors
(a) Medical									
(i) Status asthmaticus	6	48 (32–69)	6	10 (one permanent)	5	4	2	5	4
(ii) Chronic obstructive airway disease ..	10	59 (43–74)	10	16 (two permanent)	8	8	4	3	5
(iii) Severe pneumonia	2	52 (50–53)	1	18	2	14	0	–	1
(iv) Thoracic cage abnormality ..	5	40 (30–57)	1	15	2	2	4	11	4
(v) Miscellaneous ..	2	37 (18–56)	0	?	2	3	0	–	0
(b) Postoperative pneumonia	3	60 (56–63)	1	7	2	7	1	1	1
(c) Traumatic	16	50 (22–77)	3	18	10	4	14	15	13

handles most of the cases of acute respiratory failure in White adults in hospital practice in the Johannesburg area. Suspected or actual contagious paralytic cases are admitted to the Fever Hospital. Some of the patients are referred to the unit by the Accident Service of the Department of Surgery, others by the medical units, a smaller number coming from general surgical units or from neighbouring hospitals.

The types of conditions treated are recorded in Table I, and in a more detailed manner in Tables II, III and IV. The average age is 45 years, the youngest patient being 18 years and the oldest 77. The sex incidence is 2 : 1 (46 male and 24 female). The average duration of stay in the unit varied considerably from group to group; the over-all mean was 11 days. The majority of patients were transferred to another ward as soon as they could be managed without the assistance of a respirator.

TABLE III. NATURE OF THORACIC INJURIES

Nature of injury	No. of cases	No. of cases with paradoxus	No. of cases with significant haemo- or pneumothorax	No. of deaths
Cage fracture only	7	5	1	1
Cage fracture with lung injury	7	6	3	2
Cage fracture with lung and mediastinal injury	2	1	0	0

RESULTS

1. Intrinsic Chest Disease

This group included all cases where the primary abnormality was either in the respiratory system itself or in the thoracic cage.

(a) *Medical*

(*i*) *Status asthmaticus* (6 cases). The patients with status asthmaticus were referred after attempts to control their airway obstruction by conservative means had failed. They had all had asthma for many years. The length of stay in hospital before admission to the unit varied from 1 to 24 days (average 7 days). All of them required tracheostomy and mechanical ventilation. There were 2 deaths. Necropsy was performed on one, the cause of death being cerebral anoxia. This patient had suffered a prolonged period of respiratory failure before admission and was semi-conscious with a pCO_2 of 120 mm.Hg when first seen by the unit. She did not regain consciousness. The second death was due to peripheral vascular failure,

TABLE IV. PRIMARILY NEUROLOGICAL DISEASE

Diagnosis	No. of cases	Average age and range (years)	Average duration of unconsciousness (hours)	Average duration of tracheostomy (days)	Type of respirator used: Bird — No. of cases	Bird — Average time required on respirator per patient (days)	Engström — No. of cases	Engström — Average time required on respirator per patient (days)	No. of survivors
Drug intoxication ..	20	33 (18–56)	47	6 (14 cases)	18	3 (No respirator in two)	2	10	18
Head injury	1	32	24	1	0	–	0	–	0
Cerebrovascular accident	2	42 (25–60)	24	Tracheostomy not performed	2	1	0	–	0
Muscular paralysis ..	3	47 (31–64)	0	15	2	6	2	6	0

the electrocardiogram indicating probable myocardial infarction.

(*ii*) *Chronic obstructive airway disease* (10 cases). In these patients respiratory failure had been precipitated by pulmonary infection which in at least 3 cases was mild. All required tracheostomy together with mechanical ventilation. Five died in the unit. Two cases were left with a permanent tracheostomy because of the persistence of permanent severe respiratory decompensation after the acute infective episode had passed. Of the 5 patients discharged from the unit, 3 died within 4 weeks of discharge.

Necropsy was performed in 4 of the 5 patients who died in the unit. Two had evidence of thrombosis of the right main pulmonary artery. This probably precipitated death. One died from perforation of the bowel owing to steroid therapy; this patient whose record is published elsewhere[3] also had evidence of pulmonary aspergillosis. The fourth patient died following haemorrhage into a large bullus associated with congenital cystic lung disease. The fifth patient, on whom no necropsy was performed, was thought on clinical grounds to have died from myocardial infarction.

(*iii*) *Pneumonia without obstructive airway disease* (2 cases). One of these patients had had recurrent episodes of pneumonia, the responsible organism being a *Staphylococcus aureus*. She required tracheostomy and mechanical ventilation for a week, and recovered without any significant deterioration in her pulmonary status. The second patient denied any previous lung disease, but had a long history of alcoholism and evidence of liver damage. He died about 3 weeks after admission to the unit from bacterial endocarditis demonstrated at necropsy.

(*iv*) *Thoracic cage abnormality* (5 cases). In this group difficulty in fully expanding the thoracic cage was the common factor. Two were grossly obese, suffering from the Pickwickian syndrome, respiratory failure being precipitated by lung infection. One died, apparently from pulmonary embolism. No necropsy was done.

Of the remaining 3 patients, 2 had suffered recent burns of the trunk, restriction of chest movement by pain and gross oedema having resulted in respiratory failure. Both had severe tracheobronchitis from the inhalation of fumes. The third patient was a young man who had kyphoscoliosis owing to previous osteomyelitis of the ribs and presented on this occasion with a superadded bronchopneumonia.

(*v*) *Miscellaneous* (2 cases). One patient in this group had sustained fractured ribs and a pneumothorax during external cardiac massage for cardiac arrest owing to myocardial infarction. He required both mechanical ventilation and an intercostal drain. He succumbed soon after admission and necropsy showed the presence of a thrombus at the origin of the left main coronary artery.

The other patient died from cardiac arrest following penicillin anaphylaxis.

(b) *Postoperative Pneumonia* (3 *cases*)

Common factors in these cases, in addition to pneumonia, were a recent abdominal incision and the presence of intestinal ileus. All were in respiratory failure but in only one was this the major problem. In this male patient there was a long history of asthma; laparotomy was undertaken because of a massive gastro-intestinal tract haemorrhage. He recovered satisfactorily on tracheostomy and mechanical ventilation. The other 2 died, but death was not attributable to respiratory failure. One was found at necropsy to have a myocardial infarction sustained probably at the time of operation. The other died from acute renal failure owing to hypotension following the repair of a ruptured aortic aneurysm.

(c) *Traumatic* (16 *cases*)

Of the 16 cases, 13 survived. In 9 the major injuries were not confined to the thorax. Eight had a combination of abdominal and head injuries and limb fractures in addition to the chest injury. The ninth suffered from spinal injury with paraplegia.

Three of these patients had previous lung disease, but in only one was this primarily responsible for respiratory decompensation, the thoracic injury being relatively minor.

The nature of the thoracic injury for the whole group is recorded in Table III. The presence of mediastinal damage was diagnosed on the basis of electrocardiographic changes and a widened mediastinum on X-ray examination of the chest.

Abdominal surgery to repair damaged viscera contributed to inadequate ventilation in 3 patients. Thirteen of the patients developed pulmonary infection. This varied in severity but hampered recovery in only 2.

The cause of death in the 3 fatal cases in this group was systemic candidiasis with associated renal failure, extensive organization of pulmonary haematomas and pulmonary embolism respectively.

2. Primarily Neurological Disease

The patients with respiratory failure owing to impairment of the neuromuscular control of respiration can be classified into 4 groups (Table IV).

(a) *Severe Drug Intoxication* (20 *cases*)

About 300 patients with drug intoxications, the majority mild, are admitted to the Johannesburg Hospital in a year. Only the most severe are admitted to the respiratory unit. 70% (14) of those admitted to the unit remained comatose for over 24 hours and the majority displayed complications other than respiratory failure. Thus ileus occurred in most, hypotension requiring specific therapy in 8, and 'barbiturate' myocarditis and severe hypothermia each occurred twice. Barbiturates were responsible in 8 of the 20 cases, glutethimide in 2, and a combination of drugs in 10. The average age of the patients was considerably lower than in most other groups and none had experienced previous severe respiratory disease.

Intubation was initially endotracheal, being replaced by an elective tracheostomy at the end of 24 hours in the 14 cases remaining unconscious for this period. Eighteen required mechanical ventilation, 4 being totally apnoeic at some stage. The Bird respirator sufficed in all but 2 who developed severe pneumonia and required the more powerful Engström.

Forced diuresis was utilized in 16, peritoneal dialysis in 3 and exchange transfusion of 8 litres in one case. Pneumonia, responsive to antibiotics, occurred in 11 patients. One of the 2 deaths was due to respiratory failure. This patient was incorrectly assessed and became apnoeic after premature weaning from the respirator. The second death

followed persistent hypotension after a 7-day period of coma.

(b) *Head Injury and* (c) *Cerebrovascular Accident* (3 *cases*)

Probably none of these patients merited admission to a specialized respiratory resuscitation unit. One suffered from cerebral contusion and died about a day after admission in peripheral circulatory failure. The second suffered from a ruptured posterior fossa aneurysm and the third from a hypertensive cerebral haemorrhage; both were admitted to the unit in an apnoeic state and died within 24 hours.

(d) *Muscular Paralysis* (3 *cases*)

One case was an acute porphyric with generalized paralysis. He died of associated peripheral circulatory failure. Another had carcinomatous myopathy and died of the underlying disease. The third was a case of tetanus following burns. He suffered severe muscular spasms and was curarized and mechanically ventilated. He became accidentally disconnected from the Engström respirator and sustained irreversible cerebral anoxia in the few minutes before the technical fault was detected.

COMPLICATIONS OF TREATMENT

The complications of therapy encountered are those related to tracheostomy and mechanical ventilation *per se* and those related to cross-infection.

1. Complications of Tracheostomy and Artificial Ventilation

Tracheostomy was performed in 62 cases (89%) and a respirator was used at some time in all but 3 patients (96%). The complications directly attributable either to tracheostomy or to the respirator appear in Table V. Only one of these was fatal, namely, disconnection of a curarized patient from the respirator. The connection between the tracheostomy tube and the respirator has since been modified and we believe that accidental separation is now virtually impossible.

TABLE V. COMPLICATIONS OF TRACHEOSTOMY AND MECHANICAL VENTILATION

Complication	*No. of cases*
Accidental disconnection	1
Dislocation of the tracheostomy tube with obstruction to the airway	1
Severe subcutaneous emphysema	1
Severe haemorrhage requiring blood transfusion	1
Pneumothorax	1
Tracheal stenosis	1
Cellulitis	2
Failure of the tracheostomy wound to heal spontaneously	2
Total	10

Three other complications require mention:

(*i*) Pneumothorax is a recognized hazard of intermittent positive-pressure respiration. It occurred in one young patient who recovered and who surprisingly was easily managed on the Bird respirator with inspiratory pressures of only 15 cm. of water.

(*ii*) Extensive subcutaneous emphysema is uncommon, although minimal quantities of air enter the tissues after most tracheostomies. In one patient subcutaneous emphysema became extensive enough in 30 minutes to involve the whole trunk and both arms. The mechanism responsible appeared to be partial dislocation of the tracheostomy tube with the result that part of the air delivered by the respirator was blown into the tissue planes rather than down the trachea.

(*iii*) Tracheal stricture was undoubtedly due to tracheostomy in one case. In another there had been extensive tracheal and laryngeal injury, and stricture formation appeared to have occurred at both the sites of trauma.

2. Infection

Adequate facilities for the separation of 'clean' from infected cases are not at present available in the unit. The measures used to prevent cross-infection have been confined to (i) washing and sterilization of the respirator tubing and valves before and after use, (ii) the use of disposable Portex tracheostomy tubes which are changed every few days and discarded after being used once, and (iii) the practice of an aseptic technique and the wearing of a disposable mask during suctioning of the tracheostomy.

The patients without evidence of respiratory infection on admission were those suffering from chest trauma and those with neurological abnormality. Only 3 of the 16 patients with thoracic injury did not develop evidence of pulmonary infection while in the unit. This infection delayed recovery in 2 owing to the development of empyema. In one, the empyema was secondary to a perinephric abscess which ruptured through the diaphragm. Twelve patients in the group with neurological disease developed pneumonia, but in none of them did it significantly hinder recovery.

A *Staphylococcus aureus* was at some time cultured from the sputum of 17 patients. Phage-typing was routine. In only 3 was there definite evidence that the staphylococcus was the infecting organism. In one of these pneumonia was present at the time of admission. There was never a constant population of bacteria in specimens of sputum or tracheal washings from different patients concurrently in the unit.

DISCUSSION

The over-all mortality rate was 34%, an incidence similar to the experience of other writers where they have dealt with similar types of cases.[2,4-9] Table VI shows that only 5 of the 24 deaths could be attributed to respiratory failure. The remaining 19 died from other complications of their underlying diseases. Thus only 7% of the total admissions died of respiratory failure and of these 2 could have been avoided. One patient was incorrectly assessed and another was accidentally disconnected from the respirator. Outside of specialized units the incidence of error has been shown to be greater, particularly errors of a technical nature.

The survival rates in the various groups of patients treated warrant attention. The treatment of closed thoracic trauma has been revolutionized by the advent of intermittent positive-pressure respiration (IPPR).[2, 6, 10, 11] Closed thoracic injury carried a high mortality in the past. In the present series of 16 only 1 of the 3 deaths was due to respiratory failure. In the drug coma group of 20 cases there were 2 deaths, one of which was possibly avoidable.

Intensive care of these patients has brought about a reduction in the mortality rate of severe cases from 20% to about 4% as reported in a survey and review from Sweden.[12] IPPR plays a secondary role, but may be life-saving as in 4 of our patients who were apnoeic at some time. We employ a respirator when there is any doubt about the adequacy of spontaneous respiration, to safeguard against sudden cessation of spontaneous respiration, to prevent peripheral atelectasis in hypopnoeic patients, and to ensure adequate humidification of the inspired air. In keeping with the practice in other centres, we strenuously avoid the use of analeptic drugs.[12] IPPR has also improved the prognosis of respiratory failure owing to muscular paralysis as reported by Engström[13] and Spalding.[14] Our results in this group of 3 cases only are clearly not satisfactory, but the better prognosis cases, at least as far as respiration is concerned (poliomyelitis, Guillain-Barré syndrome) are treated at the Fever Hospital in the Johannesburg area. Lastly, the management of patients with post-operative pneumonia where intestinal ileus, postoperative pain and sometimes obesity hinder voluntary chest expansion and may precipitate respiratory failure, has been simplified and improved by IPPR.

TABLE VI. CAUSES OF DEATH

Respiratory failure	*No. of cases*
Organized pulmonary haematomas	1
Intrapulmonary haemorrhage	1
Status asthmaticus	1
Accidental disconnection of respirator	1
Error of judgement	1
Total	5
Other causes	
Myocardial infarction	4
Pulmonary embolism	2
Pulmonary artery thrombosis	2
Peripheral circulatory failure	2
Cerebral haemorrhage	2
Head injury	1
Peritonitis	1
Acute renal failure	1
Bacterial endocarditis	1
Systemic candidiasis	1
Carcinoma	1
Penicillin anaphylaxis	1
Total	19

Obstructive airway disease presents a different prognostic picture. IPPR, together with the all-important bronchial lavage to remove obstructing secretions, has a definite place in the treatment of status asthmaticus when conservative measures fail. Peripheral circulatory failure is relieved rapidly and recovery is the rule. Death from uncomplicated bronchial asthma is well documented.[15] In patients with respiratory failure owing to pneumonia superimposed on chronic obstructive chest disease, long-term improvement has followed recovery from the crisis only in those cases that have a good respiratory reserve.[16] In our series of 10 cases, the emergency of acute respiratory failure was readily and successfully treated by tracheostomy and IPPR. At the time of the emergency, however, it was often impossible to assess the degree of background permanent incapacity. If there has been prolonged respiratory incapacity and particularly in the presence of chronic cor pulmonale, tracheostomy and IPPR may alleviate the immediate crisis only to prolong a miserable existence.

Our experience of the management of apnoea owing to brain trauma is too limited for generalizations to be made. Here too is the threat that tracheostomy and IPPR will save the unfortunate patient for an unconscious vegetative existence on a respirator, unless the apnoea is due either to a reversible central nervous system lesion such as extradural haemorrhage or to a brief period of anoxia of central origin.

We have up to the present time used only 2 types of respirator, namely the pressure-cycled Bird Mark VII and the volume-time-cycled Engström. The experience gained with these machines supports the conclusions reached by Fairley and Hunter.[17] Patients in the neurological group with normal lungs can be adequately ventilated with any reliable equipment, provided it is used properly. In patients recovering from drug coma a patient-triggered machine proves useful. We are familiar with and have standardized on Bird machines for these types of cases. Chronic obstructive airway disease where air-trapping and poor alveolar mixing are major problems is often adequately managed with patient-triggered flow-adjustable machines such as the Bird, but the Engström has a better airflow-rate pattern for this type of patient, particularly in severe cases. The Engström has no device to allow for patient triggering. Consequently, since synchronization between patient and machine is vital in this group, continuous sedation is sometimes necessary.

Cases with pulmonary contusion and flail chests often require considerable hyperventilation against decreased lung and thoracic cage compliance. The respirator used must be able to deliver volumes of the order of 20 litres per minute and must possess sufficient power to maintain this in the face of varying resistance. Severe cases are usually satisfactorily managed only with a volume-cycled machine. The Engström is in our experience by far the better machine for these patients. Although the Bird is capable of pressures up to 40 cm. of water, the volume of gas delivered is unpredictable at this pressure.

For pH and pCO_2 measurements we use the method described by Siggaard-Andersen *et al.*[18] using arterialized capillary blood, and find it adequate in the majority of cases. Only when there has been marked peripheral vasoconstriction has arterial puncture been necessary.

Finally, attention should be drawn to the fact that in spite of what we consider to be our inadequate anti-infective measures, dangerous or disabling cross-infection has not yet occurred. Nevertheless, all the tracheostomies and many lungs became infected to some degree, but serious sequelae were inconspicuous.

SUMMARY

The first year's experience of the Respiratory Resuscitation Unit at the Johannesburg Hospital is reviewed. Seventy cases were treated during this period. The average duration of stay in the unit was 11 days. The over-all mortality rate was 34%, but of the 24 deaths only 5 (7%) were due to respiratory failure. The best results were obtained in non-penetrating chest trauma and drug intoxication. The

treatment of status asthmaticus gave satisfactory results, but in chronic obstructive lung disease tiding the patient over an acute crisis of respiratory failure usually did no more than restore him to his previous chronic ill-health, and the long-term prognosis was very poor indeed.

REFERENCES

1. Elliott, G. A., Rubenstein, A. H., Stables, D. P. and Van As, A. W. W. (1965): S. Afr. Med. J., **39**, 517.
2. Van Hasselt, H. and Marchand, P. (1965): South African Journal of Surgery, **3**, 1.
3. Jacobs, P., Friedberg, E. C. and Van Niekerk, V. (1965): Cent. Afr. J. Med., **11**, 155.
4. Hercus, V., Johnston, J. B., Rollison, R. A. A. and Hackett, R. E. (1964): Lancet, **1**, 1265.
5. Norlander, O. P., Björk, V. O., Crafoord, C., Friberg, O., Holmdahl, M., Swensson, A. and Widman, B. (1961): Anaesthesia, **16**, 285.
6. Harrison, W. H., Gray, R. A., Couves, C. M. and Howard, M. J. (1960): Amer. J. Surg., **100**, 715.
7. Woolf, C. R. (1961): Canad. Med. Assoc. J., **84**, 466.
8. Fairley, H. B. (1961): Anaesthesia, **16**, 267.
9. Safar, P., De Kornfeld, T. J., Pearson, J. W. and Redding, J. S. (1961): *Ibid.*, **16**, 275.
10. Maloney, J. V. and McDonald, L. (1963): Amer. J. Surg., **105**, 484.
11. Mörch, E. T., Avery, E. E. and Benson, D. W. (1955): Surg. Forum, **6**, 270.
12. Nilsson, E. (1951): Acta med. scand., suppl. 253.
13. Engström, C. G. (1954): Brit. Med. J., **2**, 666.
14. Spalding, J. M. K. and Crampton Smith, A. (1963): *Clinical Practice and Physiology of Artificial Ventilation*, Appendix A, p. 133. Oxford: Blackwell.
15. Earle, B. V. (1953): Thorax, **8**, 195.
16. Munck, O., Kristensen, H. S. and Lassen, H. C. A. (1961): Lancet, **1**, 66.
17. Fairley, H. B. and Hunter, D. D. (1964): Canad. Med. Assoc. J., **90**, 1397.
18. Siggaard-Andersen, O., Engel, K., Jorgensen, K. and Astrup, P. (1960): Scand. J. Clin. Lab. Invest., **12**, 172.