

THE MANAGEMENT OF RESPIRATORY FAILURE IN A COUNTRY HOSPITAL :

with special reference to crushed chests

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RESPIRATORY FAILURE, which may be defined as a "condition in which the amount of oxygen and carbon dioxide in the blood stream is altered by an abnormality of the respiratory system" (Arnott, 1960), may arise from many causes – among them diseases of, injury to, or intoxications of the central or peripheral nervous systems, thoracic skeleton, diaphragm and other respiratory muscles, and lung parenchyma. Many of these lesions are reversible, and if the patient is adequately resuscitated during the period of temporary respiratory inadequacy, a full recovery may be expected.

The use of artificial respiration to maintain life in animals unable to breathe was described by Hooke (1667), and in curarized man by Waterton (1879). These descriptions were primarily of experimental work. The application of prolonged artificial respiration in clinical medicine was described by Drinker and McKhann (1929) and Eve (1932). Since then the development of efficient compact lung ventilators, and the widespread use of curariform drugs in anaesthesia following their use by Griffith and Johnston (1943) has increased the use of artificial respiration in clinical medicine.

It has been suggested that patients requiring artificial or intermittent positive ventilation (I.P.V.) should be treated in the intensive care or respiratory failure unit of a large hospital (Windsor and Dwyer, 1961). From the point of view of the economy of use of medical staff (Dundee and Gray, 1963) and availability of laboratory facilities this system has much to commend it. It has, however, two disadvantages :

(1) Patients with crushing chest wall trauma, which commonly causes reversible respiratory failure, frequently have multiple injuries. They may have lost much blood, or be shocked and do not tolerate movement well. Furthermore, the presence of internal haemorrhage, arising from a ruptured spleen, liver or kidney can be most easily diagnosed by close observation of changes in pulse rate and blood pressure. A rising pulse rate or fall in blood pressure will often be regarded as an indication for laparotomy. Movement of an injured patient may easily provoke cardio-vascular changes simulating those of internal haemorrhage. Unnecessary movement may thus render the diagnosis of associated internal injuries more difficult and result in delay in their treatment.

(2) Ventilatory failure may occur in some patients with overdosage of or abnormal response to drugs, but if treated immediately and effectively the patient's life may be saved. Since the period of I.P.V. required is often a matter of hours rather than days, the time spent on the journey to a respiratory unit may be disproportionate to the total period of resuscitation required. The management of a case of this type in a country hospital has been described by Barron and Milliken (1960).

This paper presents a series of patients who received I.P.V. in the treatment of respiratory failure arising in the surgical practice of Daisy Hill Hospital, Newry, during the period August 1961–December 1965. A case of respiratory failure which was treated by means other than I.P.V. is reported elsewhere (Nicholl et al., 1967).

CASE REPORTS

Case 1

A 57 year old female had a hydronephrotic right kidney removed in Daisy Hill Hospital on 29th August, 1960. Operation, anaesthesia and convalescence were uneventful. She was discharged on 7th September and failed to attend the follow-up clinic.

She was re-admitted on 22nd October 1961 with a history of abdominal pain and vomiting; there was a progressive rise in temperature and she became anuric. X-ray of the left renal tract showed an opacity near the lower end of the ureter. After atropine premedication, under thiopentone, nitrous oxide, oxygen and halothane anaesthesia, cystoscopy was performed and an attempt made to pass a ureteric catheter. This was unsuccessful, so the patient was intubated under topical (lignocaine 4 per cent.) anaesthesia and the lower end of the ureter was explored. During this latter procedure, as relaxation was inadequate, the halothane was turned off and the patient was given gallamine (Flaxedil) 80 mg. Manual ventilation of the lungs was performed by compression of the bag in the anaesthetic circuit. Twenty minutes later the patient began to breathe more vigorously so she was given a supplementary dose of gallamine 20 mg. The wall of the lower end of the ureter was found to be calcified. The bladder was opened and the ureter dilated. This caused the escape of some purulent urine which had been held up by the obstruction. During closure of the wound relaxation was again insufficient, so she was given a further injection of gallamine 10 mg. intravenously approximately 15 minutes after the second injection of this drug. Throughout the operation the patient's respiratory efforts were not completely abolished by the dose of relaxant employed, which totalled 110 mg. of gallamine.

At the end of the operation diaphragmatic breathing and tracheal tug were present and the intercostal muscles were paralysed. Administration of atropine and neostigmine caused an improvement in her condition lasting about half an hour, after which the signs of partial curarization recurred.

A tracheotomy was performed and the patient was respired for nine hours with a Smith-Clarke ventilator, following which she recovered. A sample of blood taken at the time of operation had a urea content of 275 mg. per 100 ml. Four weeks later the blood urea had fallen to 20 mg. per 100 ml.

The patient was re-admitted on 6th December 1961 with airway obstruction. This was found to be due to the formation of a web in the trachea near the site of the

tracheotomy. The web was broken down and the airway re-established but it re-occurred about three weeks later. The tracheal stricture was again dilated, but tracheal resection was subsequently required to effect a permanent cure. This complication of tracheotomy has been noted by other authors (Bargh and Slawson, 1965).

Case 2

A 34 year old female was admitted on 14th January 1965 with multiple injuries following a motor car accident. These included fractures of the shafts of both femora, pelvis and orbital margin, sixth right rib, left third to ninth ribs and she had a left pneumothorax. On admission she was severely shocked. After transfusion with two pints of plasma, four pints of blood and administration of cortisol and metaraminol (Aramine), the legs were placed in Thomas' splints under general anaesthesia (methohexitone 10 mg. nitrous oxide and oxygen).

During the next three days "traumatic wet lung" gradually developed and on the 18th January a tracheotomy was performed because of the profuse amount of purulent sputum which was being aspirated from the tracheo-bronchial tree. A size 10 Oxford tracheotomy tube was inserted. Her condition continued to deteriorate and by evening the respiratory rate had risen to 44 per minute and there was paradoxical movement of the chest wall. I.P.V. was started using an Adelaide Mk. 2 ventilator (Kenny and Lewis, 1960), a Garthur condenser-humidifier being used to prevent drying of the bronchial tree. Since the tube was a close fit in the trachea, with a negligible leak of air, the cuff was not inflated. At first the patient resisted mechanical ventilation, but she readily became apnoeic after being hyper-ventilated manually using an Ambu bag. Thereafter it was possible to ventilate her with the Adelaide machine at 28 cycles per minute. A similar routine was necessary to get the patient re-established on the ventilator after tracheal suction, which was carried out at half-hourly intervals.

By the fifth day of ventilator treatment (23rd January) it was possible for her to stay off the ventilator for up to 30 minutes at a time, but she became very restless on the slightest physical exertion. On the 25th the ventilator was changed to a Smith-Clarke machine. Although the Garthur condenser-humidifier had proved adequate to prevent drying of the bronchial tree, it did nothing to replace water vapour which was removed when tracheal suction was performed. It was felt that the hot moist air provided by the Smith-Clarke ventilator would be more beneficial.

Between 27th and 31st January the patient developed acute gastric dilatation and it was necessary to give up to five litres of fluid intravenously daily in order to balance the patient's body water loss.

On the first of February artificial ventilation was discontinued (after 14 days) but the tracheotomy was kept patent for a further four days in order to perform adequate bronchial toilet. By the 2nd February the gastric aspiration was reduced to 90 ml. in 24 hours and the stomach tube was withdrawn. Thereafter convalescence was uneventful in so far as her respiratory and alimentary function was concerned, although prolonged because of her fractured femora. She was discharged on 21st July.

The aetiology of the acute gastric dilation in this patient is obscure. It may arise in patients with tetanus who are curarized and given I.P.V., possibly due to the action of curare on the sympathetic ganglia ; but this patient did not receive any

myo-neural blocking agent at any time. It may also arise in patients who are hypokalaemic, but during this period of loss of alimentary function the patient's serum potassium level was 4.4 m. eq/l, 3.6 m. eq/l and 3.8 m. eq/l on three occasions.

Case 3

A 64 year old male underwent laparotomy for gastric carcinoma on 2nd February 1965, operation revealing an inoperable growth with extensive metastases. Anaesthesia (thiopentone, suxamethonium, nitrous oxide, oxygen, d-tubocurarine chloride) was uneventful, but there was difficulty in re-establishing adequate respiratory movement post-operatively. The patient displayed clinical signs of partial curarisation which was unaffected by intravenous injections of atropine and neostigmine. He was ventilated with air through an oro-tracheal tube from a Smith-Clarke ventilator for eight hours after which he was extubated.

His convalescence was thereafter uneventful and he was discharged home on 21st February. It was thought that the causation of the prolonged paralysis may have been a mixed type neuromuscular block (Paton, 1956).

Case 4

A 75 year old male was admitted to hospital on 23rd November 1965, following a motor accident in which he sustained fractures of the right first to seventh ribs and of both rami of the pubic bones. There were also multiple bruises and some minor lacerations.

He had been in hospital two months previously with a history of chest pain, when an electrocardiograph showed evidence of right bundle branch block.

Signs of a lung infection developed and by 8th December he was cyanosed and sweating, with paradoxical movement of the chest wall deep to the right pectoralis major. The trachea was intubated under topical (lignocaine 2 per cent.) anaesthesia and about 20 ml. purulent mucus sucked out of the trachea. He was ventilated through the endotracheal tube with an Adelaide ventilator with a considerable improvement in his colour and a diminution in sweating. Because of his respiratory inadequacy and the copious amounts of pus which were being obtained from the trachea, tracheotomy was performed and a size 10 Oxford tracheotomy tube was inserted. Ventilation was carried out with a Smith-Clarke machine delivering a tidal volume of 700 ml. at a respiratory frequency of 22 cycles/minute, a supplementary oxygen flow of 2 litres/minute being added to the inspired air.

Five hours later there was a considerable improvement in his condition and it was possible to turn off the supplementary oxygen. He tended to resist inflation, but settled well after receiving morphine 10 mg. intramuscularly. Ventilation was continued on the following day, but on the 10th December he was able to breathe adequately for two hours.

During the next two weeks there was a very gradual improvement in his condition, although he was very frail throughout. Faecal incontinence developed on the 12th and it was difficult to prevent bedsores occurring. A tracheal swab taken on the 13th grew coagulase-positive *Staphylococcus pyogenes*, coliforms, *Clostridium welchii* and *Proteus*. The tracheotomy was cleaned with hydrogen peroxide, gentian violet and "Hibitane" and he was given systemic antibiotics. It was possible to clear the wound of three of these organisms, but the staphylococcus remained to the end.

After two and a half weeks of ventilator treatment he was able to dispense with I.P.V. for three days, during which he was breathing adequately through the tracheotomy. Unfortunately, he now developed signs of venous thrombosis in the left leg. Anti-coagulant therapy was started on the 27th but on the 31st there was a marked deterioration in his condition and it became necessary to restart ventilator treatment. It was thought that he had developed a pulmonary embolism. The deterioration in his condition continued and he was transferred to the Royal Victoria Hospital on the 6th January 1966. He died four days later.

Post mortem examination showed a thrombosis of the left femoral vein with bilateral pulmonary emboli.

A fifth case has been reported previously in detail in this journal (Nicholl & Pillow, 1964) and is summarised briefly below :

Case 5

A woman of 22 years was admitted, following a motor car accident, with multiple injuries which included a fracture of the middle fossa of skull, multiple bilateral rib fractures and a right pneumothorax. On admission she was unconscious with periodic (Cheyne-Stokes) breathing ; she had a pulse rate of 140 beats per minute, but the blood pressure was within normal limits.

Twenty-four hours later there was a deterioration in her condition. The pulse rate, which had earlier fallen, had again started to rise and the blood pressure had fallen. Because of these signs of internal haemorrhage laparotomy was performed following blood transfusion. Operation revealed a ruptured spleen which was removed. The patient's post-operative condition was satisfactory, but twelve hours later it began to deteriorate ; cyanosis developed and secretions pooling in the pharynx began to "spill over" into the larynx, causing intermittent laryngeal spasm. Tracheotomy was performed and the patient's condition remained satisfactory for a further 36 hours ; after which a further deterioration took place. Cyanosis returned, the respiratory rate increased and paradoxical movement of the sternum and anterior ends of the ribs was marked. I.P.V. was given continuously for the next four days and intermittently for a further seven days. After this she was able to breathe adequately but only through a tracheostome. The tracheotomy was removed on the thirty-third day and she was discharged home forty-five days after admission to hospital.

DISCUSSION

Since the use of a tank respirator to resuscitate a patient with multiple rib fractures by Hagen (1945), and more so since the use of I.P.V. in the management of reversible respiratory failure in acute poliomyelitis by Lassen (1953) and Ibsen (1954), artificial respiration has acquired a wide application in therapeutics. The principles of treatment are well established (Spalding and Smith, 1963) although there are differences in detail among different authors.

The management of individual patients with severe crushing chest wall injury has been described in some detail (Avery et al., 1955 and 1966 ; Clarkson and Robinson, 1962 ; Garden and Mackenzie, 1963). Some describe series of patients with respiratory failure of diverse origins (Pearce, 1961) ; including chest wall injury as one of the causes (Safar et al., 1961 ; Norlander et al., 1961 ; Fairley, 1961 ; Robbie and Feldman, 1963 ; Bargh and Slawson, 1965). Other authors have

published details of larger series of patients with crushing chest injury and analysed the results (Griffiths, 1960 ; Whitwam and Norman, 1964 ; Reid and Baird, 1965 ; Lloyd, et al., 1965; Campbell, 1966).

Many of these papers have come from teaching hospitals or other large units which usually have a highly developed laboratory service. This is reflected in the complexity of the investigations which several of the authors seem to regard as routine (Campbell, 1966) or even obligatory (Bates, 1964) if a patient with respiratory failure is to be resuscitated. Since "patients with conditions so desperate as to require intensive care withstand movement extremely badly" (Robinson, 1966) a difficult decision may face the clinician in charge of such a case, if the patient is admitted to a provincial hospital in which facilities for blood gas analysis are not always available.

In spite of the difficulties attending the management of patients with major chest wall injury in respiratory failure in provincial hospitals, Sandor (1963) has reported a series of 9 patients with paradoxical chest wall movement due to rib fractures treated in two provincial hospitals in five years. None of his patients received I.P.V. as a therapeutic measure and only one patient required a tracheotomy. Two of his patients underwent thoracotomy for associated intra-thoracic injuries.

This series is presented to illustrate the feasibility of treating patients in respiratory failure with I.P.V. in a country hospital with limited laboratory facilities and no methods for blood gas analysis. The assessment of the adequacy of ventilation was on purely clinical grounds :

(1) As a general rule patients were ventilated with air only (i.e. without additional oxygen). Patients might easily be underventilated (with a resulting retention of CO₂) if an oxygen enriched atmosphere were employed for a long time. By reducing or eliminating alveolar nitrogen this also predisposes to pulmonary, lobar or alveolar collapse if there is much retained sputum.

(2) If patients become cyanosed, oxygen is added to the inhaled air and the minute volume is increased until the patient's colour improves. As soon as this happens the oxygen is gradually reduced until the patient is again being ventilated with air only.

(3) The early treatment of post-haemorrhagic anaemia is important because wounds and fractures are slow to heal in anaemic subjects. Cyanosis as an important sign of hypoxaemia, and of possible underventilation, is lost on these patients.

(4) If a patient feels comfortable when on a ventilator, respiration is probably adequate.

(5) Heavy sedation is avoided, because in the presence of respiratory depression inadequate ventilation may be tolerated. Experience shows that patients require most sedation when due to be weaned off the ventilator or when there is foreign material in the bronchial tree.

It may be suggested that, while it is possible to treat patients in respiratory failure due to crushing chest injury in a country hospital, the superior results which may be expected in a larger unit justify the risks of an otherwise unnecessary journey. The table gives an estimate of the mortality rate among patients receiving ventilator treatment for crushing chest injury in ten teaching hospitals. Where an author gives the number of patients requiring I.P.V. for chest injury, this is quoted but some have not differentiated between patients with crushed chests who required

TABLE

Centre	Author	No. of cases admitted to intensive care unit	No. with severe chest injury	No. of deaths among patients with severe chest injury
Baltimore	Safar et al.	3931*	8	1
Belfast	Gray, Dundee and Clark	350	19	9
Edinburgh	Bargh and Slawson	160	51	7
Glasgow	Campbell	51	28	14
Leeds	Whitwam and Norman	9	9	3
Liverpool	Robinson	168	11	3
Oxford	Lloyd et al.	121	33	9
Stockholm	Norlander et al.	522	14	4
Toronto	Fairley	204	20	4
Westminster	Robbie and Feldman	50	2	0
Total			195	54

NOTE: * The Baltimore figure includes 3370 post-operative cases with no complications, admitted to the intensive care unit.

The results of treatment of crushed chests by I.P.V. in ten teaching hospitals.

ventilator treatment, and those less ill who did not require this. Here the total number of cases with major chest injury managed by that unit is quoted. It will be seen that of a total of 195 patients treated for crushing chest wall injuries, 54 died; a mortality rate of approximately 27 per cent.

A patient admitted with multiple rib fractures to a small hospital may face the risk of transport to a larger unit which may aggravate his state of shock and render more difficult the diagnosis and treatment of associated injuries. By treating the patient in the hospital to which he is first admitted the risks of movement are avoided and the diagnosis of concomitant injuries may be facilitated, but the risks associated with ventilator treatment remain.

The results obtained by the anaesthetic service in Daisy Hill Hospital, Newry, in the management of such patients during the period August 1961 to December 1965 support this view.

SUMMARY

The possibility of treating patients in respiratory failure by mechanical lung ventilation in a country hospital is demonstrated. Case histories are given and some of the complications of treatment are described.

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