INTERMITTENT POSITIVE PRESSURE RESPIRATION AFTER OPEN-HEART SURGERY

BY

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SUMMARY

(1) A description has been given of the use of tracheostomy and IPPR in the treatment of twenty-five cases suffering from respiratory and circulatory complications after open-heart surgery.

(2) In patients suffering from respiratory abnormalities, results were good. In those with a derangement of the circulation results were poor. However, it was concluded that therapy in this group is often justified.

(3) Although arterial blood gas determinations are of value, the decision to use artificial ventilation must be based on a consideration of the history and clinical state in addition to the biochemical data.

The use of intermittent positive pressure respiration (IPPR) in the treatment of ventilatory inadequacy after thoracic surgery is well documented (Björk and Engström, 1955, 1957; Sealy, Young and Hickman, 1957; Robson, 1958; Masson and Robertson, 1958; Björk, 1960; Norlander et al., 1961). Recently this technique has been used to treat patients recovering from open-heart surgery (Norlander et al., 1958, 1961; Spencer et al., 1959; Gilston, 1962). Although the respiratory inadequacy occurring in these circumstances is commonly due to depression of the respiratory centre or to lesions of the lungs and bronchi, it may also be associated with changes in the pulmonary blood vessels. These changes may be due to pre-existing pulmonary vascular disease, or to engorgement of the lung vessels during or after perfusion (Kolff et al., 1958; Kolff, Eiffler and Groves, 1960).

This paper analyses the results obtained in twenty-five patients who were treated with IPPR after open-heart surgery under total body perfusion.

MATERIAL

During the period March 1, 1960, to December 31, 1961, 176 patients were operated upon in this unit under total body perfusion. During the same period twenty-six patients (14.8 per cent) were treated on respirators.

Details of twenty-five cases are given in tables I and II. The records of one patient are not available.

INDICATIONS FOR TREATMENT

The patients have been classified into two groups according to the system primarily affected. The groups are (1) Respiratory and (2) Circulatory.

Group 1. Respiratory

There were fourteen patients in this group (table I). Two of these also had some degree of circulatory inadequacy.

Clinical.

All patients presented with some degree of respiratory distress. The term “distress” is used in its widest sense. Facial expression and general behaviour suggested that these patients were at the limit of endurance. Many were extremely restless. Cyanosis was obvious in almost every case and the respirations were usually shallow, fast and laboured. The respiratory rate was often above 50 per minute. The accessory muscles were active and a grunting type of respiration was common. In one case the respirations were slow and deep, but...
### Table I
Cases with primarily respiratory indications.

<table>
<thead>
<tr>
<th>Series No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Time Onset/Duration</th>
<th>Indications</th>
<th>Result</th>
</tr>
</thead>
</table>
| 1.         | I.F. | 18 years Pulm. stenosis        | 96 hours/4 days     | Respiratory distress and hypoventilation.  
Pco\(_2\)=55 mm Hg (oxygen saturation 70% in tent).  
Pulmonary consolidation.  
Re-operation for tamponade (27 hours).  
Neurological: Status epilepticus requiring sedation. | Died—15th day.  
Pulmonary embolism  
Improved on IPPR.                                    |
| 2.         | B.G. | 11 years p.v.r. 7 units. | 21 hours/16 days    | Respiratory distress—right haemothorax and patchy atelectasis.  
Re-operation for haemostasis (9 hours) and tamponade (23 hours). | Survived.  
Long, complicated course with bronchospasm and cardiac arrest. |
| 4.         | T.H. | 14 years a.v Canal with cleft mitral valve. | 40 hours/4 days | Respiratory distress and hypoventilation.  
Pco\(_2\)=62 mm Hg.  
Haemothorax and bilateral lower lobe atelectasis.  
Circulatory: Pulmonary oedema moderate, resulting from incompetent mitral valve. | Survived.  
Uneventful course. |
| 9.         | B.W. | 36 years a.s.d. with pulmonary and mitral stenosis. | 5 hours/4 days | Respiratory distress—blood in airways—cardiac arrest.  
Pulmonary artery thrombosis.  
Previous lung disease—bronchitis and tuberculosis. | Died.  
Increasing pulmonary consolidation due to pulmonary artery thrombosis. |
| 10.        | M.O. | 51 years Ruptured sinus of Valsalva. | 4 hours/2 days | Respiratory distress—bronchospasm and hypoventilation (Pco\(_2\)=59 mm Hg).  
Previous lung disease—chronic bronchitis. | Died.  
Acute anoxia airway obstruction. |
| 13.        | B.W. | 50 years Aortic stenosis.      | 24 hours/9 days     | Respiratory distress—retention of secretions; hypoventilation.  
Pco\(_2\)=68 mm Hg.  
Patchy atelectasis.  
Previous lung disease—infiltration of right base. | Survived.  
Uneventful course. |
| 15.        | C.G. | 7 years Fallot’s tetralogy with previous Blalock. | 18 hours/10 days | Respiratory distress, intrapulmonary bleeding.  
Left pneumothorax and laceration of lung (oxygen saturation—79% in tent).  
Re-operation for haemostasis (6 hours). | Died.  
Progressive pulmonary consolidation, aspiration of blood. |
| 16.        | J.A. | 34 years v.s.d. with pulmonary hypertension. | 16 hours/12 hours | Respiratory distress—minimal atelectasis.  
Cardiac arrest (16 hours) with delayed recovery. | Died.  
Prolonged unconsciousness and convulsions following arrest. |
| 19.        | F.M. | 55 years Mitral and aortic stenosis. p.v.r. 12 units. | 3 hours/4 days | Respiratory distress. Left haemopneumothorax and massive atelectasis.  
Re-operation for haemostasis (3 hours).  
Long history of chronic bronchitis and congestive failure. | Died.  
Sudden cardiac arrest during improvement.  
? Anoxia. |
### TABLE I—continued

<table>
<thead>
<tr>
<th>Series No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Time Onset/Duration</th>
<th>Indications</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>23. E.C.</td>
<td>13 years</td>
<td>Fallot's tetralogy.</td>
<td>29 hours/5 days</td>
<td>Respiratory distress (oxygen saturation on air 68%). Hyperventilation (Pco₂=27 mm Hg). Left lower lobe atelectasis and minimal effusion.</td>
<td>Survived. Uneventful course.</td>
</tr>
<tr>
<td>24. S.P.</td>
<td>6 years</td>
<td>Fallot's tetralogy.</td>
<td>54 hours/36 hours</td>
<td>Respiratory distress—re-operation for haemostasis (5 hours). Small left pleural effusion with left lower atelectasis. Neurological: Drowsy semiconscious state with inability to cough.</td>
<td>Survived. Uneventful course.</td>
</tr>
<tr>
<td>25. S.V.</td>
<td>8 years</td>
<td>Fallot's tetralogy.</td>
<td>48 hours/38 hours</td>
<td>Respiratory distress (oxygen saturation 88% in tent). Bronchospasm. Inability to cough. Wide mediastinum and left lower lobe atelectasis.</td>
<td>Survived. Uneventful course except for tracheostomy infection.</td>
</tr>
</tbody>
</table>

### TABLE II
Cases with primarily haemodynamic indications for IPPR.

<table>
<thead>
<tr>
<th>Series No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Time Onset/Duration</th>
<th>Indications</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. M.H.</td>
<td>36 years</td>
<td>Mitral incompetence following closed valvotomy (2 weeks prior).</td>
<td>11 hours/4 days</td>
<td>Prophylactic: Severe cardiac failure following closed valvotomy. Respiratory: Persistent right pleural effusion pre-operatively from thrombosis of right lower pulmonary vein.</td>
<td>Survived. Moderate residual incompetence with cardiac failure.</td>
</tr>
<tr>
<td>5. K.G.</td>
<td>5 years</td>
<td>Fallot's tetralogy.</td>
<td>36 hours/46 days</td>
<td>Low cardiac output and hypotension. Severe cyanosis, loss of consciousness, cardiac failure, acute respiratory distress.</td>
<td>Survived. Persistent cardiac failure. Incomplete relief of outflow tract and closure of v.s.d.</td>
</tr>
<tr>
<td>6. R.G.</td>
<td>13 years</td>
<td>Fallot's tetralogy.</td>
<td>5 hours/2 days</td>
<td>Complete heart block. Low cardiac output and hypotension. Severe cyanosis. Re-operation for haemostasis and tamponade (5 hours).</td>
<td>Died. Incomplete relief of outflow tract and closure of v.s.d.</td>
</tr>
</tbody>
</table>
TABLE II—continued

<table>
<thead>
<tr>
<th>Series No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Time Onset/Duration</th>
<th>Indications</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.</td>
<td>V.P.</td>
<td>8 years.</td>
<td>28 hours/8 hours</td>
<td>Complete heart block. Low cardiac output and hypotension.</td>
<td>Died. Small right ventricle resulted from repair.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a.s.d.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Common ventricle.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Corrected transposition.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fallot’s tetralogy.</td>
<td></td>
<td></td>
<td>(No necropsy.)</td>
</tr>
<tr>
<td>11.</td>
<td>K.H.</td>
<td>18 years.</td>
<td>28 hours/3 days</td>
<td>Low cardiac output and hypotension.</td>
<td>Died. Re-operation (48 hours) for severe tricuspid incompetence and closure of v.s.d.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>v.s.d.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>p.v.r. 5 units.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>P.E.</td>
<td>11 years.</td>
<td>20 hours/7 days</td>
<td>Pulmonary oedema—overdistension of l.v. and backward engorgement.</td>
<td>Survived. Good result.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>v.s.d.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>M.W.</td>
<td>45 years.</td>
<td>48 hours/2 days</td>
<td>Complete heart block. Low cardiac output and hypotension.</td>
<td>Died.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fallot’s tetralogy.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>B.C.</td>
<td>52 years.</td>
<td>4 hours/24 hours</td>
<td>Severe cardiac arrhythmias leading to cardiac arrest.</td>
<td>Died. Further cardiac arrest following persistent arrhythmias.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>v.s.d.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>following myocardial infarction.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>M.T.</td>
<td>19 years.</td>
<td>1 hour/8 days</td>
<td>Pulmonary oedema—collateral overfilling of lungs from undiagnosed p.d.a.</td>
<td>Died. Sudden death, following improvement.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p.d.a.</td>
<td></td>
<td>Complete heart block. Respiratory: kyphoscoliotic with severe impairment</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>p.v.r. 8 units.</td>
<td></td>
<td>of ventilatory function (m.b.c: 22-8 l/min).</td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>M.B.</td>
<td>14 years.</td>
<td>1 hour/10 days</td>
<td>Pulmonary oedema—overtransfusion and forward engorgement.</td>
<td>Survived. Sinus rhythm re-established on 3rd post-op. day.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fallot’s tetralogy.</td>
<td></td>
<td>Complete heart block (temporary).</td>
<td></td>
</tr>
</tbody>
</table>

**TABLES I AND II**

Times, apart from those under heading "duration", refer to time interval from end of operation.

**Abbreviations.**
- a.s.d. = atrial septal defect.
- v.s.d. = ventricular septal defect.
- p.v.r. = pulmonary vascular resistance.
- p.d.a. = patent ductus arteriosus.
- l.v. = left ventricle.
gasping in character. Coughing was feeble and the clearance of secretions ineffectual. In some cases clinical examination revealed an obvious cause for the respiratory distress, such as atelectasis, hemothorax, or severe bronchospasm.

No patient in this group had a depressed level of consciousness due to carbon dioxide retention. Three patients were semiconscious. In one case (No. 1) this was due to the heavy sedation used in the treatment of severe status epilepticus. In another (No. 16), it was due to anoxic cerebral damage following cardiac arrest. In the third (No. 24) increasing drowsiness responded dramatically to intravenous urea, although there was no obvious cause for cerebral damage.

Radiological.

At the time of instituting IPPR there were radiological signs which were considered sufficient to account for the clinical state of the patients in only six of the fourteen cases. In two cases there was no pulmonary abnormality in the postoperative films. In the remainder only minimal signs of interstitial pulmonary oedema or left lower lobe atelectasis were present. Two of these patients subsequently developed radiological and clinical signs of pulmonary infarction.

Biochemical.

It was not possible to perform arterial blood gas analyses routinely during the early part of this series. In the six cases in which estimations were performed immediately before commencing IPPR, a mild respiratory acidosis was found. The values for arterial carbon dioxide tension (Paco₂) ranged from 55 to 68 mm Hg. In one case there was evidence of hyperventilation, the Paco₂ being 27 mm Hg. Although the patient was in an oxygen tent the oxygen saturation at this time was 78 per cent.

In no case in this group was there a severe "non-respiratory" acidosis.

In seven patients in whom oxygen saturation was measured before instituting therapy, values between 68 and 93 per cent with a mean of 83 per cent were obtained. These patients were all in oxygen tents when measurements were made.

Group 2. Circulatory

Eleven patients had a severe circulatory disturbance resulting from perfusion or surgery (table II). Three of these also had complications originating in the respiratory tract. In one of these three (No. 18), severe pulmonary disease was present pre-operatively.

Clinical.

Six of the eleven patients in this group (Nos. 5, 6, 7, 8, 11 and 14) were treated with IPPR because of a low cardiac output syndrome. This was characterized by a decreased level of consciousness, severe peripheral cyanosis, severe systemic hypotension in the presence of a raised venous pressure, and signs of marked peripheral vasoconstriction.

Three cases presented with obvious pulmonary oedema. Two of these patients developed this during operation (Nos. 18 and 20). In the third patient (No. 12) the pulmonary oedema presented 12 hours after operation.

One patient in this group (No. 17) was treated because of severe hypotension and respiratory inadequacy after a cardiac arrest. This followed a period of multiple arrhythmias. The last case (No. 3) was in severe congestive failure pre-operatively and was placed on IPPR prophylactically following open repair of a mitral incompetence.

Radiological.

The cases with pulmonary oedema showed the typical appearances (fig. 3a). Those in the low output group showed no radiological changes initially, unless secondary pulmonary complications were present.

Biochemical.

In patients with low cardiac output states a marked "non-respiratory" acidosis was present. Two of these patients had standard bicarbonate values of 14 and 15 m-equiv/l. with pH readings of 7.18 and 7.17 respectively. The acidosis was corrected by intravenous sodium bicarbonate.

TECHNIQUE

There are certain aspects of technique which are of particular importance.

Tracheostomy.

This is usually performed under general anaesthesia with controlled ventilation. The use of such a technique facilitates surgery and reduces the metabolic demand for oxygen. The immediate application of IPPR is of immense value if any pulmonary oedema is present. At the end of the procedure vigorous physiotherapy and tracheal
suction can be carried out without discomfort to the patient and the lungs can be re-expanded. In addition, the PaO₂ can be lowered below normal levels by cautious hyperventilation, and the patient can be connected to the ventilator before consciousness returns. This facilitates synchronization.

There are, however, certain drawbacks to the use of general anaesthesia, particularly in those cases where tamponade is suspected, or where hypovolaemia is present. In these circumstances controlled ventilation may prove lethal. The induction should, therefore, be undertaken with great care. The patient is placed on the operating table and the wound dressings are removed. While the surgeon is scrubbing, the patient is allowed to breathe oxygen through a non-rebreathing system. An assistant monitors the pulse throughout. Anaesthesia is then induced with a minimal concentration of an inhalational agent such as cyclopropane. Intubation can often be performed without the use of relaxants, but in some cases a small dose of suxamethonium may be necessary. Spontaneous ventilation is then allowed to return and is maintained until the blood volume is restored or until the chest is opened to release the tamponade. Controlled ventilation can then be utilized for the rest of the procedure.

Ventilation.

Patient-triggered ventilators have not proved successful during the early stages of treatment. Synchronization with a time-cycled ventilator is achieved by ventilating the patient at a rate slightly in excess of that existing before IPPR. Large tidal volumes are used initially and gradually reduced over the next few hours. As pointed out elsewhere (Sykes, 1962), the minute volumes recorded during the postoperative period are commonly greatly in excess of those existing pre-operatively. It is, therefore, difficult to reduce the minute volume in an adult below 10 or 12 litres per minute in the early stages of treatment. Nevertheless, continual attempts are made to decrease the minute volume over the next few days by gradually reducing both rate and depth of respiration. Small quantities of depressant drugs may be necessary if the patient is distressed or in pain, and in some cases small “softening” doses of d-tubocurarine (5-10 mg intravenously) are required.

The ventilators used have included various models of the Radcliffe, Blease, Smith-Clarke, and Engström. We now prefer to use a ventilator that will deliver a fixed volume in the face of a changing lung compliance. This will largely prevent periods of hypoventilation resulting from accumulation of secretions, bronchospasm or desynchronisation, but it should be realized that the use of such a ventilator does not guarantee a constant tidal volume. There are two main causes of a variation in tidal volume; there may be a leak in the circuit, and there may be a change in the volume of gas compressed in the breathing tubes and humidifier. The volume of gas compressed into this space will depend on the volume of the space and on the pressure generated in overcoming the resistance to lung inflation. It is, therefore, essential to use tubes and humidifier with small internal volumes and to monitor both inspiratory pressure and expired volume at least every 15 minutes, and whenever the patient appears to be distressed. The chart on which these ventilatory parameters are recorded should also include instructions for the emergency treatment of acute respiratory distress. The relevant portion of the chart reads as follows:

NURSING RECORD FOR PATIENTS ON A VENTILATOR

Instructions.

At all times watch the colour of the lips and tongue and the movements of the chest. If secretions are heard at any time first inflate with 100 per cent oxygen, then suck out quickly. If the patient becomes distressed, if cyanosis appears or if the chest movements become impaired.

1. Check position of tracheostomy tube.
2. Disconnect the ventilator and inflate the patient with oxygen inflation unit.
   If the chest still does not move well:
   Deflate the cuff and bag—squeeze again using a large flow of more than 10 l./min of oxygen.
   If still no success:
   Remove the tracheostomy tube and insert a new one before inflating with the bag once more.

Humidification.

Hot water humidifiers (Marshall and Spalding, 1953) have been used. The inspired air temperature has been monitored continuously and the water temperature in the can has been adjusted to keep the inspired air temperature between 30 and 35°C; this ensures optimal humidification. The humidifier has been placed close to the patient to decrease heat loss from the tubing. This reduces the temperature differential along the inspiratory tube.
Tracheal aspiration is carried out at half-hourly intervals or more frequently if required. Full sterile precautions are used, great emphasis being placed on the brevity of the procedure and the continued provision of ventilation with 100 per cent oxygen. This is achieved by using a modified Nosworthy connector with a narrow chimney for suction. Physiotherapy is carried out at least three or four times each day, and while the patient is on the ventilator an anaesthetist attends. Vigorous manual inflation is carried out, the physiotherapist producing a forced expiration by compressing and vibrating the chest wall just as inflation ceases. in this manner secretions are expelled into the major bronchi and trachea, from which they can be aspirated by a soft sterile rubber catheter with terminal and lateral holes. Bronchoscopy is only used if there is persistent collapse of part of the lung or if it is necessary to investigate the origin of bleeding into the trachea. In this group of patients the procedure is considered to be extremely dangerous.

Daily bacteriological swabs are taken from the trachea and the tracheostomy wound, so that antibiotic sensitivities are available by the time a clinical infection becomes manifest.

Weaning is commenced by allowing spontaneous respiration for 5-minute periods during the day. This period is gradually increased until the patient is off the ventilator all day. Only then is the patient taken off the ventilator at night. The time required for weaning varies greatly, periods of 24 hours to 1 month having been required in this series. Despite the natural desire to curtail the period of IPPR it is important not to hurry weaning; this will result in exhaustion, increased secretions, and a deterioration in the clinical and radiological state.

Throughout treatment the greatest care is taken to prevent infection. All air entering the tracheostomy tube, either from a ventilator or from a blowertype humidifier, is filtered by passing it through a bacterial filter of non-absorbent cotton wool which is changed daily. Nursing and medical personnel wear masks and after use all mechanical ventilators and humidifiers are sterilized with formaldehyde vapour.

COURSE AND RESULTS

Group 1. Deaths.

Seven of the patients in Group 1 died. In case No. 1 death occurred 4 days after completion of IPPR. Death was due to multiple pulmonary infarcts but there was a small cerebral abscess from which pseudomonas pyocyaneus was grown.

Two children with Fallot’s tetralogy (Nos. 15 and 22) died due to overwhelmingly severe intrapulmonary haemorrhage associated with a low platelet count.

CASE No. 15.

An attempt to divide a previous Blalock anastomosis resulted in a laceration of the left lung with torrential haemorrhage into the left pleural cavity and lung. The aspiration of blood into the airways was confined to the left lung initially but spread later to involve the right lung. Compliance was markedly decreased. Various respirators were tried but none reduced the PaO₂ below 70 mm Hg. Eventually it was necessary to use half-hour periods of manual ventilation every 2 hours. The chest films revealed the severity of the pulmonary consolidation (fig. 1). Temporary improvement was achieved after almost continuous physiotherapy and suction over a period of 7 days. However, the platelet count steadily fell to a level of 8,000 per cu.mm by the 7th day, despite platelet transfusions. Profuse bleeding from the nose, mouth and respiratory tract and gut followed. The patient remained in a moribund state for a further five days during which time many complications occurred. These included renal failure, recurrence of heart block with circulatory failure, pulmonary oedema, and a left femoral vein thrombosis with gangrene of the left foot. Death occurred on the 10th day.

Pulmonary artery thrombosis caused the death of another patient (No. 9). The fifth patient (No. 19) had mitral and aortic stenosis and a pulmonary vascular resistance of 12 units. This patient was improving when acute respiratory distress occurred on the fourth postoperative day. (A similar episode of unexplained respiratory distress with a fatal result occurred in another patient in Group 2 (No. 18), and these deaths are discussed later.)

There were two other deaths. One (No. 16) occurred in a 3½-year-old child with a large ventricular septal defect. Sixteen hours postoperatively the blood pressure fell and cardiac arrest occurred. The patient was resuscitated but signs of cerebral damage were present and 12 hours later a further cardiac arrest occurred. The other death was due to a technical error. A silver tracheostomy tube with a detachable latex cuff was being employed in an elderly obese patient with bronchitis (No. 10). On the fourth day of treatment with IPPR, respiratory obstruction suddenly occurred, neither tracheal aspiration nor manual inflation being possible. The tracheostomy tube was removed but
1.4.61. On IPPR. Right lung clear. Patchy consolidation with some collapse in left lung. Some extrapleural fluid at the apex and a wide mediastinum on the left side.

22.4.61. Massive bilateral consolidation. Little aerated lung tissue.

8.4.61. Marked resolution of consolidation. Distended pulmonary vessels and probable early pulmonary oedema.

30.4.61. Severe bilateral pulmonary oedema.

Patient No. 15, aged 7 years. Operation (20.4.61) for complete correction of Tetralogy of Fallot and division of previous Blalock anastomosis. IPPR from 21.4.61 to 1.5.61.
the inflated cuff was left in situ. Before this could be removed cardiac arrest from anoxia occurred.

**Group 1. Survivors.**

There were seven survivors out of the fourteen patients in this group. Six of these had relatively uncomplicated courses, with a successful outcome. Four of these six patients (Nos. 13, 23, 24 and 25) were treated because of respiratory distress associated with retained secretions and varying degrees of atelectasis. The fifth case (No. 4) had an atrio-ventricular canal with pulmonary vascular disease. Postoperatively, widespread consolidation and collapse occurred and there was an associated haemothorax. The sixth patient (No. 21) was elderly and had an atrio-septal defect. The pulmonary artery pressure was raised before operation and a pulmonary artery thrombosis occurred post-operatively. These six patients were all in severe respiratory distress following operation and responded well to IPPR. The duration of therapy varied between 36 hours and 9 days.

**CASE NO. 13.**

The patient was an elderly, extremely anxious patient with aortic stenosis. Fourteen hours after operation she became cyanosed, restless and distressed and was unable to cough or co-operate. The respiratory rate was 50 b.p.m. and she was rapidly becoming exhausted. Tracheostomy alone failed to relieve her condition and 3 hours later IPPR was commenced. She synchronized easily with the ventilator and welcomed the rest. Apart from some distress on suction, the course was uneventful and weaning was completed by the 9th day of IPPR.

**Laboratory data.**

- Prior to IPPR: \( P_{a_{ox}} = 68 \) mm Hg; oxygen saturation = 84 per cent (in tent).
- First day: \( P_{a_{ox}} = 32.5 \) mm Hg; oxygen saturation = 96 per cent (with added oxygen), 82 per cent (on air).
- Third day: oxygen saturation on respirator = 97 per cent (with added oxygen), 90 per cent (on air).
- Eighth day: oxygen saturation on respirator = 96 per cent (on air), and oxygen saturation on spontaneous ventilation = 92 per cent (on air).

The X-rays are shown in fig. 2.

The seventh survivor in this group (No. 2) had a remarkably stormy course.

**CASE NO. 2.**

The patient was a girl, aged 11 years, who had a ventricular septal defect and severe pulmonary hypertension. Respiratory distress became marked following a second operation for excessive bleeding, 24 hours after total cardiopulmonary bypass. A tracheostomy was performed and IPPR was instituted 5 hours later. Bleeding continued and after 3 hours a further tamponade was diagnosed. Cardiac arrest occurred as this was being relieved. The heart was restarted and the patient quickly regained consciousness after operation. During the next 4 days on IPPR there was troublesome "bronchospasm". Weaning was slow, and on the 6th day of IPPR there was an acute attack of severe "bronchospasm" following tracheal aspiration. This led to anoxia and cardiac arrest. The...
chest was re-opened and the heart restarted. Bronchoscopy at this time revealed an extremely congested mucous membrane with a slit-like narrowing and kinking of the left main bronchus. The "bronchospasm" was treated by aminophylline and isoprenaline but, on several occasions during the next few days, it became so severe that only manual ventilation with extremely high pressures prevented a further anoxic cardiac arrest. Definite improvement coincided with the adoption of a sitting-up position and, after a further 10 days of IPPR, weaning was accomplished. This patient subsequently made an excellent recovery.

**Group 2. Deaths.**

There were eleven patients in this group, seven of whom died. Five of the patients who died (Nos. 6, 7, 8, 11, and 14) had a poor haemodynamic result following surgery. These patients suffered from complete heart block, incompletely corrected cardiac abnormalities, the production of another lesion such as tricuspid incompetence (No. 11) or abnormal narrowing of the right ventricle (No. 7).

The results of treatment with IPPR were disappointing, there being little improvement in the severe hypotension, restlessness, peripheral cyanosis and diminished level of consciousness. The severity of the postoperative haemodynamic complications was incompatible with prolonged survival.

In the sixth patient who died (No. 17), an acquired ventricular septal defect due to a myocardial infarction was closed. Following operation, she developed ventricular tachycardia and cardiac arrest. After successful external cardiac massage the patient was placed on a ventilator but further episodes of ventricular tachycardia led to repeated arrests during the next 12 hours.

The seventh death occurred in a poor-risk patient (No. 18), who had a ventricular septal defect with pulmonary hypertension. The pulmonary artery pressure was 90/50 mm Hg and the pulmonary vascular resistance was 8 units. There was also severe impairment of pulmonary function due to kyphoscoliosis and a history of repeated chest infections. Perfusion in the presence of an undiagnosed patent ductus arteriosus produced collateral engorgement with resulting pulmonary oedema during and after operation. This ductus was functionless since pressures in the pulmonary artery and aorta were equal: there was consequently no thrill or murmur (Cleland et al., 1958). IPPR proceeded uneventfully for 7 days, the blood-stained aspirate diminishing in quantity, with clearing of the physical signs and steady radiological improvement. Complete heart block was present, though a satisfactory heart rate and blood pressure were maintained with isoprenaline suppositories. On the eighth day of treatment, 2 hours after thorough physiotherapy, the patient coughed, tracheal aspiration was carried out, but she became extremely distressed and agitation and cardiac arrest occurred. Cardiac massage resulted in only a temporary response. The pulmonary oedema in this patient had been successfully treated by the use of IPPR when the death occurred.

These last two patients (Nos. 17 and 18) had extremely severe pre-operative lesions and complications.

**Group 2. Survivors.**

There were four survivors in this group. Two of these patients (Nos. 12 and 20), together with case No. 18 discussed above, had pulmonary oedema. These three patients illustrate the different mechanisms responsible for pulmonary vascular engorgement. These are forward, backward, and collateral overfilling as described by Kolff and his colleagues (1958, 1960).

**Case No. 12 (Ventricular Septal Defect).**

A period of left ventricular distension occurred, due to prolapse of an aortic cusp during perfusion which resulted in backward overfilling of the pulmonary circulation. The immediate postoperative clinical state was poor, the patient was drowsy, with severe hypotension, and an arterial oxygen saturation of 82 per cent in an oxygen tent. Obvious pulmonary oedema developed on the 1st postoperative day (fig. 3). IPPR resulted in marked improvement in the patient's colour and blood pressure. Radiologically, there was considerable resolution of the pulmonary oedema within 24 hours. Recovery was uneventful, weaning being complete by the 7th day.

**Case No. 20 (Fallot's Tetralogy).**

Pulmonary oedema occurred shortly after perfusion due to forward overloading of the pulmonary circulation with blood in an attempt to treat a persistently low blood pressure. Radiologically, the lungs were not clear until the 6th day of treatment. IPPR was required for 10 days.

The third survivor in this group (No. 5), who had Fallot's tetralogy, survived despite incomplete closure of the ventricular septal defect and incomplete relief of the outflow tract obstruction. The patient was in persistent congestive heart failure during the postoperative period and required mechanical ventilation for 46 days. Many attempts were made during this period to wean the patient but on each occasion there was a recurrence of right-heart failure with accumulation of pleural...
effusions and clinical distress. The survival of this child was dependent on the maintenance of the IPPR.

The other patient in this group (No. 3) underwent a prophylactic tracheostomy following open repair of mitral incompetence. The incompetence resulted from a closed valvotomy 2 weeks previously. Between the two operations the patient was in severe cardiac failure and after the second operation therapy with IPPR was required for 4 days. The patient survived but suffers from a moderate degree of heart failure.

DISCUSSION

Of the 176 patients submitted to open-heart surgery in this hospital 14.8 per cent (twenty-six) received IPPR. Fourteen of these died, a mortality rate of 54 per cent. Norlander et al. (1958) stated that mechanical ventilation was used in nineteen out of the first fifty-two patients undergoing open-heart surgery (37 per cent); there were seven survivors (mortality rate 68 per cent). These authors stated that treatment with a mechanical ventilator was strongly indicated in patients with pulmonary complications and the slightest degree of respiratory inadequacy. A second report (Norlander et al., 1961) concerned fifty-three patients treated by artificial ventilation. There were twenty-two survivors (mortality rate 58 per cent). The total number of patients undergoing surgery was not stated. Eight of these 53 patients suffered from pulmonary oedema and, despite mechanical ventilation, these all died by the third postoperative day. Norlander and associates report that the incidence of pulmonary oedema has decreased since the adoption of left atrial venting during operation. Buckley, van Bergen and Theil (1960), in a discussion of postoperative treatment following open-heart surgery, advise the use of IPPR in severe pulmonary congestion. On the other hand,
Lyons, DuShane and Kirklin (1960) state that whole body perfusion need not be accompanied by an undue incidence of pulmonary oedema or excessive tracheobronchial secretions. They are of the opinion that tracheostomy and mechanical assistance to ventilation are rarely required.

In an attempt to define the indications for this form of treatment more accurately, the present authors adopted a conservative approach to the use of IPPR in this first series of twenty-six cases. Despite this reluctance to employ the technique, it has been found necessary to employ IPPR in over 14 per cent of patients operated upon under total body perfusion. There are several possible reasons why mechanical assistance to ventilation has been utilized so often. Firstly, the incidence of postoperative respiratory complications following any form of surgery is undoubtedly higher in this country than in many others. This may be directly related to the high incidence of chronic bronchitis and upper respiratory tract infections (Stuart-Harris and Hanley, 1957). Secondly, many patients referred to this unit for surgery have pulmonary hypertension or complicated anatomical defects, both of which render them more liable to the development of postoperative complications. Thirdly, IPPR has been employed in the treatment of a group of patients with low cardiac output states and severe circulatory failure. The value of IPPR in these cases is debatable.

In the opinion of the authors, and also of Gilston (1962), the decision to use IPPR must be based on the patient's history and clinical condition. In patients whose main abnormality is in the respiratory system, determination of arterial Pco₂ is of great help. Serial oxygen saturations on 100 per cent oxygen and on air are also of great value in assessing the degree of shunt and venous admixture effect present. Finally, certain factors which are known to affect the prognosis unfavourably should be considered. These are: a poor haemodynamic state following operation; previous pulmonary disease, or other respiratory abnormality; moderate or severe pulmonary vascular disease; increasing age. Respiratory difficulties seem particularly likely to occur in patients over 45 years of age and in those who undergo a second operation shortly after the first. Nine of our twenty-five cases had been re-opened within 27 hours of the previous operation.

In the circulatory group of cases the most dramatic results are obtained in those suffering from pulmonary oedema. In patients suffering from other circulatory disturbances, it is felt that IPPR should be used if the oxygen saturation is low, if there is ventilatory inadequacy, if there is a severe metabolic acidosis which cannot be corrected adequately by intravenous sodium bicarbonate, or if the level of consciousness is decreasing rapidly. Under the last circumstance intravenous urea may also be of value. Recently Doberneck, Reiser and Lillehei (1962) have pointed out that IPPR is particularly valuable in the treatment of patients who have developed low cardiac output states and associated acute renal failure after open-heart surgery. In general, one may conclude that patients with good haemodynamic results rarely need ventilatory assistance unless they are elderly or have marked pre-operative chest disease. On the other hand, if the haemodynamic result is not perfect, and any of the above-mentioned complications are present, ventilatory inadequacy should be treated at the earliest possible moment by means of tracheostomy and IPPR.

There are many risks associated with the use of IPPR. There are additional hazards for patients treated with IPPR after open-heart surgery. These patients are extremely sensitive to oxygen lack. Anoxia may be induced either by a brief interruption in the supply of oxygen to the lungs or by an increase in the metabolic demand for oxygen due to undue restlessness, shivering, pyrexia or desynchronization with the respirator. It is, therefore, vital to ensure that the supply of oxygen is continuous and that desynchronization and restlessness are treated immediately. In one patient (No. 11) with severe circulatory failure and marked hypotension, disorientation, twitching and extreme restlessness followed every attempt at suction, even though this was of the briefest duration. Two other patients (Nos. 18 and 19) died during tracheal suction. Severe pulmonary vascular disease was present in both patients. Complete heart block was present in one. In the other only partial relief of aortic stenosis was obtained by valvotomy. In both cases the patient suddenly became distressed as tracheal aspiration was performed. Cardiac arrest occurred immediately. Postmortem examination in both cases revealed marked hypertensive pulmonary vascular changes, pulmonary oedema and
moderate amounts of sticky secretions, but no gross atelectasis. It is extremely difficult to be certain of the sequence and cause of these events. It seems most likely that they were due either to a sudden attack of severe "bronchospasm" or to an acute attack of left ventricular failure precipitated by the anoxic insult of tracheal suction.

Another hazard is infection. In this unit the most common organism is pseudomonas pyocyaneus. This has been isolated in five patients. Treatment with Polymixin insufflations via the tracheostomy has been effective in clearing this infection in three cases. In one patient (No. 1) unsuspected, small, scattered abscesses were found in both lungs and in the brain. In another very ill moribund patient (No. 22) a heavy infection with coliforms and P. pyocyaneus resulted in a purulent sputum which added to ventilatory difficulties. This infection was resistant to treatment and was present until death occurred from another cause.

It is concluded that this form of therapy is of great value in the treatment of some of the complications occurring after open-heart surgery. However, it is hoped that further research will define the nature of these complications more clearly and that improvements in perfusion and surgical technique will render the use of this form of treatment unnecessary in the great majority of cases.

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REFERENCES


BOOK REVIEW


"Focus" programmes are popular entertainments in these times. In science, focus had tended to become concentrated even more intensely on less and less—perhaps, more accurately, on the even more minute. Mere size, however, is no measure of importance and it is certainly a fact that the neuromuscular junction, small as it is, is of the greatest importance and of the most intense interest to the neurophysiologist and the clinician alike. Since the middle of the last century when Claude Bernard’s classical experiments elucidated the action of curare and pointed to the existence of a junctional tissue between nerve and muscle, great discoveries have been made in this field, and these for the most part during the last thirty years. The names of Dale, Eccles, Feldberg and Katz and Kuffler, to mention only a few, recall discoveries of the greatest significance in the elucidation of the mysteries of impulse transmission in this fascinating area.

In this little volume of 103 pages the focus is still more concentrated, almost entirely indeed on the relationship of drugs which block transmission across the junction to the anionic receptors on the molecules of the endplate plasma membrane. The book is a report of a Symposium held by the Ciba Foundation in its rooms at 41 Portland Place, London, which was attended by nineteen of the world’s foremost experts on this subject under the Chairmanship of Dr. W. Feldberg. The Symposium was held to honour Professor Chagas of Rio de Janeiro, who himself contributed the first paper.

As in the case of the best symposia, only a few papers were read, five in all, but the discussions were free, extensive, and have been carefully recorded. They form an accurate reflection of the personalities of the speakers whilst being free from the solecisms which are so often a feature of this form of reporting.

The subjects covered are “The fate of curare during curarization” by Professor Chagas; “Influence of curare on uptake and release of a neuromuscular blocking agent labelled with 131I” by Professor D. B. Taylor; “Drug receptor interactions at the neuromuscular junction” by Professor Paton and D. R. Waud; “Structure-action relations throwing light on the receptor” by Professor Cavallito; “Experimental hazards and artefacts in the study of neuromuscular blocking drugs” by Professor Zaimis. The data are, of course, very technical and specialized. The editing is scrupulous and calls for no comment except a plea for the inclusion of titles in the references quoted.

In opening the Symposium the Chairman remarked that, when he read the abstracts of the papers to be delivered, he could not help wondering “what Claude Bernard would think if he could see the new facts and ideas that have emerged since he discovered the action of curare”. What indeed! Most refreshing, however, were his closing remarks in which he drew attention to the similarity of the essential qualifications for a good pharmacologist to those suggested by Sir Winston Churchill for a good politician, “... the ability to foretell what will happen tomorrow, next month and next year—and to explain afterwards why it did not happen”. To find this eminent scientist appearing to advocate courage in speculation as a necessary precedent for experimental observation is a great relief, when so many lesser investigators have become almost strangulated in their thought by an unbalanced fear of contemplating anything other than that which is in their eyes completely proven as factual.

For all workers in this field this little book is a jewel to treasure. The knowledge herein may in time become superseded but the book will remain a memento of those workers taking part in the discussions.

Cecil Gray