ANAESTHETIC MANAGEMENT FOR REPAIR OF OESOPHAGEAL ATRESIA IN THE NEW BORN

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ALTHOUGH the condition of atresia of the oesophagus has been recognized since the seventeenth century (Franklin, 1948) it was not until 1913 that the first attempt at relief by direct anastomosis was undertaken by Richter and it was a further thirty years before the first successful anastomosis was reported (Haight and Townsley, 1943). During the last decade the literature on the surgical management of oesophageal atresia has increased considerably but references to the anaesthetic techniques are still very few. In the United States Haight and Townsley (1943) have mentioned the use of open ether and Gross and Scott (1946) have recommended cyclopropane in a closed circuit without intubation, while, in South Africa, Roberts (1950) has described the anaesthesia he employed in a series of six patients. In this country Wilton (1951) has published the only detailed account of the management of anaesthesia for oesophageal atresia although Dinsdale (1954) has given a brief description of his method.

METHOD

Several days may elapse before a diagnosis of atresia of the oesophagus is made. Once it has been established, however, operation is undertaken without undue delay. In practice, this has meant that these young patients have come to the operating theatre within twelve hours of their admission to hospital. Before operation the child is nursed in a slight head down position and frequent suction is employed to keep the oral and nasal passages free from mucus. As a further precaution an intravenous drip of 1/5 N saline is established by the insertion of a fine polythene catheter into the medial saphenous vein over the medial malleolus. Atropine 1/200 grain (0.3 mg) is given intramuscularly thirty minutes before the operation is due to begin.

Anaesthesia is induced with intravenous thiopentone given slowly into the drip; a dose of 2.5–5 mg/lb (0.45 kg) of body weight in a 2.5 per cent solution is usually employed. This is followed by d-tubocurarine chloride 1 mg/5 lb (2.25 kg) of body weight. The pharynx and larynx are then cleared of secretions and the larynx intubated with a No. 00 armoured endotracheal tube. The latter is strapped in position and it is suggested that a soft pack or a dental sponge placed between the tongue and the hard palate will prevent movement. Respiration can then be readily controlled throughout the course of the operation. Controlled respiration is indicated because although

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originally access to the oesophagus was obtained by the extrapleural route, today the transpleural approach is the one most commonly adopted. The assistance of completely controlled breathing facilitates greatly the surgical technique. Before the operation is begun the anaesthetist should pass a fine rubber catheter, preferably through the nose, into the upper end of the oesophagus. This manœuvre helps the surgeon to localize more readily the tip of the upper segment.

When the incision is made, blood is substituted for the intravenous saline and allowed to drip slowly. Anaesthesia is maintained with a 50 per cent mixture of nitrous oxide and oxygen using a flow rate of 3–4 litres per minute through a Cope infant absorber. The inadvertent building up of pressure is avoided by having an opening in the catheter mount which can be sealed with the fingertip when required. Throughout the operation, at the end of each surgical manipulation, the retractors are removed and the lungs gently inflated.

If the baby still shows signs of curarisation at the end of operation neostigmine 0.03 mg per lb (0.45 kg) of body weight is given intravenously after a preliminary injection of atropine 1/200 grain (0.3 mg). When breathing is considered adequate the infant is returned to the ward in an oxygen tent.

RESULTS

Table I illustrates the main features of the patients in whom relief of an oesophageal atresia was undertaken during the latter half of 1954. It will be seen that of the six patients four are alive and well while two babies failed to survive. Both of these children had other congenital abnormalities.

Baby A. V. had previously been anaesthetised for the repair of an exomphalos but as far as was known she had no abnormality incompatible with life. Nevertheless, about 48 hours after operation she suddenly collapsed and died. The cause of death was never established as autopsy was refused.

Baby I. E. was a healthy child despite the congenital absence of his right arm. Induction and maintenance of anaesthesia were uneventful and he was breathing well when the endotracheal tube was removed. Immediately after withdrawing the tube, however, he appeared to be in some respiratory difficulty; he was consequently re-intubated and oxygen administered. There was no evidence of bronchial obstruction yet his colour became darker and he died within an hour of operation, apparently from asphyxia. At autopsy massive collapse of the right lung was found; it was further demonstrated that the normal pattern of lobes in the right lung was deficient. In retrospect it is almost certain that the collapse was due to a tension pneumothorax brought about by the combination of positive inflation of the lungs and a blocked drainage tube.

### Table I

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Birth weight</th>
<th>Other abnormalities</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.R.</td>
<td>F</td>
<td>34 hours</td>
<td>lb 8 oz</td>
<td>Deformity right ear with accessory auricles</td>
<td>Alive and well</td>
</tr>
<tr>
<td>R.R.</td>
<td>M</td>
<td>72</td>
<td>5 5</td>
<td></td>
<td>Alive and well</td>
</tr>
<tr>
<td>J.B.</td>
<td>F</td>
<td>20</td>
<td>4 4</td>
<td></td>
<td>Alive and well</td>
</tr>
<tr>
<td>A.V.</td>
<td>F</td>
<td>56</td>
<td>5 0</td>
<td>Exomphalos</td>
<td>Died 48 hours after operation</td>
</tr>
<tr>
<td>R.T.</td>
<td>F</td>
<td>56</td>
<td>5 3</td>
<td></td>
<td>Alive and well</td>
</tr>
<tr>
<td>I.E.</td>
<td>M</td>
<td>31</td>
<td>4 4</td>
<td>Absence right arm</td>
<td>Died one hour after operation</td>
</tr>
</tbody>
</table>
Fundamentally the anaesthetist’s responsibility in the management of anaesthesia for the repair of atresia is three-fold: (1) the provision of adequate anaesthesia and relaxation; (2) the prevention of anoxia and the elimination of carbon dioxide; (3) the replacement of blood loss and the restoration of disturbed fluid balance.

The hypnotic effect of thiopentone combined with the analgesic properties of nitrous oxide provide the required anaesthesia without undue depression. The provision of adequate relaxation for the surgeon is a most important feature of the management of these small patients. Breathing during the neonatal period of life is essentially diaphragmatic and abdominal, and this tends to put a variable degree of strain on the lower segment of the oesophagus. One of the dangers of operating under local anaesthesia is the tension placed on the sutures by coughing and straining. The use of a relaxant eliminates all tension and allows the surgeon to make his anastomosis deliberately and without hurry, thus reducing greatly the risk of sutures cutting out.

Although infants in the neonatal phase are remarkably tolerant of degrees of hypoxia which in the adult would produce irreversible changes, this is no justification for the mismanagement of their oxygen supply. The average new-born child requires about 25 ml of oxygen per minute (Smith, 1951) and as such a baby has a tidal air of approximately 20 ml (Deming and Hanner, 1936) a fairly rapid respiratory rate is needed to supply the necessary oxygen. This rate may be 40/min. When the chest is opened the tidal air is even further reduced and measures must be taken to ensure an adequate oxygen supply. The high oxygen content of the inspired gases contributes to satisfactory oxygenation. Breathing is further assisted by the removal of retractors at intervals throughout the operation to allow the collapsed segments of lung to be re-inflated. Patches of atelectasis are encouraged to expand by gentle massage with the tip of the operator’s finger.

In babies with oesophageal atresia a further embarrassment to respiration is offered by the presence of a tracheo-oesophageal fistula in 80 per cent (Franklin, 1948). In these patients, when controlled respiration is carried out distension of the stomach and intestines may follow. This can be avoided to a great extent by the surgeon closing the fistula as soon as possible after the chest is opened.

Any notable reduction in minute volume must lead to carbon-dioxide retention and it seems likely that many of the deaths reported following anaesthesia in young infants are due to this cause. The toxic manifestations of carbon dioxide are often masked during anaesthesia particularly when the patient is well oxygenated, and a narcotic concentration may be built up insidiously. Donald and Paton (1955) have pointed out that all anaesthetics and narcotics when given in larger doses finally cause medullary failure and they have commented that there is no reason to believe that carbon dioxide is any exception. Furthermore it has been shown (Bell et al., 1928) that in neonatal life a metabolic acidosis exists.
which may be regarded as physiological. If however a superimposed respiratory acidosis is added, this benign state may be converted into a lethal one.

The elimination of carbon dioxide is assisted by a high rate of gas flow, adequate controlled ventilation and the use of an absorber. Depressant gases such as cyclopropane and chloroform are best avoided.

Blood loss is not usually excessive in the repair of oesophageal atresia particularly if rib resection is avoided. Nevertheless when it is considered that the average 7 lb (3 kg) baby has a total circulating blood volume of approximately 270 ml (Mollison et al., 1950) it will be recognized that even slight haemorrhage needs replacement. In most cases 30-60 ml of blood are sufficient but occasionally 100 ml or more are required. Belsey and Donnison (1950) recommend the use of 200-300 ml of blood but their technique involves rib resection with presumably considerably more blood loss.

The amount of fluids administered will naturally depend on the degree of dehydration. If the child is only a few hours old dehydration will not be present. If however, several days have elapsed since birth it is advisable to establish a fluid intake. 1/5 N saline should be given but care must be taken to guard against over-transfusion. 10-15 ml of fluid per lb per day is adequate (Smith, 1951). Under-transfusion is undoubtedly safer than over-dosage and indeed complete starvation for the first 72 hours is a standard practice in the routine management of new-born premature babies. In infants, where evidence of dehydration is absent 5 per cent glucose intravenously is probably safer than 1/5 N saline.

No matter how well the surgical and anaesthetic techniques are developed there will be few successful results without the very highest standard of nursing care during the immediate postoperative period. The ultimate result is, in fact, absolutely dependent on the care and vigilance of the nursing staff.

Finally it may be said that the only chance of survival given to these small patients is operative repair. Surgical intervention is, however, dependent on the early recognition of the anomaly and as Franklin (1948) has pointed out, it cannot be emphasized too often the great responsibility resting on midwives and paediatricians to exclude atresia of the oesophagus in all new born infants who suffer from attacks of cyanosis and choking.

Morris and Heady (1955) have demonstrated that the decline in neonatal mortality has not kept pace with the fall during the remainder of the first year of life. They attribute this, among other things, to congenital malformations and mechanisms leading to "asphyxia" and "atelectasis". If as Belsey and Donnison (1950) claim the incidence of atresia of the oesophagus is 1 in 800 births, the early diagnosis and treatment of these unfortunate infants ought to reduce the incidence of neonatal deaths.

SUMMARY

The management of anaesthesia for the repair of congenital atresia of the oesophagus is described. A short series of six cases is presented. The problem of anaesthesia in such
cases is discussed and certain dangers emphasized.

ACKNOWLEDGMENTS

I am indebted to Mr. R. H. Franklin for the opportunity to anaesthetize these small babies and in particular I would like to thank him for permission to give the details in table I from his unpublished series.

REFERENCES