SURVIVAL AFTER PULMONARY HAEOMORRHAGE IN IDIOPATHIC RESPIRATORY DISTRESS SYNDROME OF THE NEWBORN

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SUMMARY

Pulmonary haemorrhage is a commonly fatal condition which may complicate mechanical ventilation of the premature neonate with the respiratory distress syndrome (RDS). The survival of one such patient is recorded.

CASE REPORT

The child, born at 34 weeks gestation by spontaneous vaginal delivery, weighed 2 kg at birth. The Apgar score at 1 min was 5, and on examination there was no evidence of any congenital abnormality. Auscultation of the chest did not reveal any murmurs (Kitterman et al, 1970).

At 8 hours old the infant began to exhibit signs of RDS, with expiratory grunting, intercostal recession, and an increased respiratory rate (80 breaths per minute). On chest X-ray an air bronchogram and the ground glass appearance in the lung fields, typical of RDS, were noted. An umbilical arterial catheter was inserted, and the blood-gas values were: pH 7.19, Pco₂, 34 mm Hg; Po₂, 70 mm Hg (the inspired oxygen concentration was 70%), base excess (BE) = -14 m.equiv/l. Ten m.equiv of sodium bicarbonate were given, and the inspired oxygen concentration was increased to 100%.

The infant suffered two periods of respiratory arrest during the next 12 hours and was intubated with a 3.5 mm Portex nasotracheal tube. Thereafter he was attached to an Engstrom 300 ventilator, and ventilated with 100% oxygen at a rate of 35 b.p.m. Five cm H₂O positive end-expiratory pressure (PEEP) was applied to the machine. The peak inflation pressure was 40 cm H₂O. At this time, the arterial blood-gas values were: pH 6.80; Po₂, 35 mm Hg; Po₁, 160 mm Hg; BE = -20 m.equiv/l. Fifteen m.equiv of sodium bicarbonate were given, and the inspired oxygen concentration was reduced to 70%.

Subsequent arterial blood-gas values were: pH 7.60; Pco₂, 20 mm Hg; Po₁, 75 mm Hg; BE = -2 m.equiv/l. In order to maintain satisfactory oxygenation, the PEEP had to be increased to 10 cm over the next 6 hours, but this resulted in the chest becoming considerably over-inflated. However, over the subsequent 12 hours, oxygenation improved (Pao₂ was 100 mm Hg; inspired oxygen concentration was 50%), and the PEEP was reduced to 5 cm.

It was now felt that the risk of pulmonary damage from mechanical ventilation with PEEP was becoming serious (Northway, Rosan and Porter, 1967). However, the infant still had apnoic episodes when breathing spontaneously. The apnoic attacks ceased at 60 hours old, and thereafter the child breathed spontaneously 100% oxygen via a humidified constant positive airway pressure (CPAP) circuit with 3 cm pressure. At this stage the arterial blood-gas values were: pH 7.50; Pco₂, 50 mm Hg; Po₁, 140 mm Hg; BE = +1 m.equiv/l. The chest X-ray showed a small right-sided pneumothorax, but this was not drained. On this regime the clinical improvement continued, and after a further 6 hours the Pao₂ was 100 mm Hg with 1 cm CPAP (30% oxygen). The infant now exhibited a marked metabolic alkalosis (pH 7.55; Pao₂, 40 mm Hg; BE = +12 m.equiv/l), which was possibly iatrogenic in origin.

After breathing spontaneously for 8 hours on the CPAP circuit, at 72 hours old the infant suddenly became cyanosed, and developed a bradycardia. These changes were followed by the appearance of bright red blood in the nasotracheal tube. The volume was not estimated, but the bleeding was brisk for 5 min, and it continued intermittently for another 30 min. Mechanical ventilation was immediately re-instituted, and with an inspired oxygen concentration of 70%, arterial blood-gas values were: pH 7.40; Pco₂, 38 mm Hg; Po₁, 100 mm Hg; BE = -1 m.equiv/l. Twelve hours later the infant was again allowed to breathe spontaneously via the CPAP circuit with 5 cm pressure and 100% oxygen. The following arterial blood-gas values were obtained: pH 7.40; Pco₂, 53 mm Hg; Po₁, 180 mm Hg; BE = +4 m.equiv/l. The general condition of the baby improved rapidly, and the endotracheal tube was removed 24 hours later with no further difficulty, apart from a lower motor neurone lesion of the left seventh cranial nerve, which lasted for 7 days, and was thought to have been the result of pressure. The infant was discharged home 3 weeks later.

DISCUSSION

The mortality from pulmonary haemorrhage in the newborn being mechanically ventilated is extremely high. In a recent report of 15 patients there were no survivors (Cole et al., 1973). Although the exact aetiology of the condition is not known, several theories have been advanced. These include:

Left heart failure with haemorrhagic pulmonary oedema.

Cole and her fellow workers postulate that the left ventricle fails because of a combination of acidosis and hypoxia, and that this, combined with haemorrhagic necrosis of the alveolar walls as a result of hypoxia, cause the familiar sanguineous exudate.

This child did not exhibit the marked acidosis (mean BE = -12 m.equiv/l) described as being an integral part of the syndrome. However, he must
have had an acid shift during or immediately preceding the haemorrhage, as the blood, 6 hours previously, had shown a gross metabolic alkalosis. Unfortunately the urine pH was not tested. In addition there was no clinical or radiological evidence to suggest left ventricular failure in this case. It also seems unlikely that hypoxia played a significant part; until the haemorrhagic episode, oxygenation was improving rapidly, and immediately following the haemorrhage the PaO₂ was 100 mm Hg with an inspired oxygen concentration of 70%.

Direct pulmonary oxygen toxicity.
High inspired oxygen partial pressures are known to cause pulmonary damage, with capillary proliferation and exudation into the alveoli (Pratt, 1958, 1965; Kistler, Caldwell and Weibel, 1967; Nash, Blennerhassett and Pontoppidan, 1967; Robinson et al., 1967). Oxygen toxicity has been suggested as a cause of pulmonary haemorrhage in neonates by Shanklin and Wolfson (1967) and Boothby and DeSa (1973) and incriminated in the aetiology of bronchopulmonary dysplasia, a condition which includes alveolar necrosis and pulmonary fibrosis (Northway, Rosan and Porter, 1967; Hawker, Reynolds and Taghizadeh, 1967).

This particular child had received inspired oxygen concentrations of greater than 70% for 60 hours, which is well into the range at which oxygen toxicity is known to occur, and this may have been a factor in causing the haemorrhage in this case.

Diffuse intravascular coagulation (DIC).
Cole, Normand and Reynolds (1973) demonstrated that DIC was an irregular feature in their series. Coagulation studies were not performed on this particular case, and so its contribution cannot be ascertained.

Infection.
The child developed a Klebsiella chest infection in the latter part of his illness, but did not have active infection at the time of the haemorrhage.

Rhesus incompatibility.
There was no evidence to suggest this.

It is not possible to ascribe the haemorrhage in this patient to any definite cause, but it does seem likely that oxygen toxicity was a contributory factor, with DIC possibly contributing also. The metabolic alkalosis exhibited by the infant prior to the haemorrhage could conceivably have contributed to his survival, by buffering the undoubtedly large increase in hydrogen ions which occurred; this was reflected in the BE decreasing from 12 m.equiv/l. to —1 m.equiv/l. Therefore, when presented with the prospect of ventilating a neonate with RDS one is faced with the dual problem of ensuring adequate oxygenation and preventing long term lung damage; often these two interests conflict. For example, the provision of an adequate PaO₂ may necessitate high inflation pressures with a high inspired oxygen concentration which could contribute to the development of pulmonary fibrosis (Hawker, Reynolds and Taghizadeh, 1967). The use of PEEP increases the inflation pressure and increases the risk of pneumothorax. Reynolds (1971) has shown that the use of inspiratory/expiratory ratios (I:E) of the order of 3 or 4 to 1 is associated with a marked increase in PaO₂, with no increase in inflation pressure. The ventilator used for this particular case had a fixed I:E ratio of 1:2, and PEEP had to be employed to provide an adequate PaO₂. As a result this particular type of ventilator is not ideal for use with cases of severe RDS due to the high inflation pressures which may result. A ventilator with a variable I:E ratio would seem a more logical choice in the acute stage of the disorder. However, Reynolds (1971) was careful to point out that this could result in a decreased venous return and air trapping if used in the resolving stages of RDS.

REFERENCES
8 cm distal to this, the oesophagus appeared normal. A radiograph of the oesophagus revealed a pin-sized stenosis 5 mm in length situated 3 cm below the cricopharynx; 1 cm distal to this, the oesophagus appeared normal.


Sir,—I wish to report the history of a boy who received 46 general anaesthetics including halothane between the ages of 18 and 32 months.

At the age of 18 months, the boy suffered from a corroded oesophagus after having accidentally swallowed vinegar essence (25%).

Four weeks later he was admitted to the ENT department with severe dysphagia. Radiography of the oesophagus revealed a pin-sized stenosis 5 mm in length situated 3 cm below the cricopharynx; 8 cm distal to this, the oesophagus appeared normal.

These findings were confirmed on subsequent oesophagoscopy. The anaesthetic technique for this procedure comprised preoperative medication with atropine 0.3 mg im. and rectal thiopentone 25 mg/kg. In the operating room, anaesthesia was maintained with oxygen, nitrous oxide and halothane and oro-tracheal intubation was performed under deep halothane anaesthesia. The course of anaesthesia and postoperative recovery were uneventful.

From the findings, it was obvious that extensive treatment would be necessary before the stenosis could be cleared. Furthermore the boy's age and psychological condition would demand general anaesthesia and in order to permit oesophageal dilatation, intubation would be mandatory to avoid the risk of compression of the trachea.

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After a few days treatment was commenced using the same anaesthetic techniques as had been employed previously. After 5 dilatations within the next 13 days, a 17-gauge bougie could be passed easily and the patient was able to swallow solid food. He was sent home in good condition. One week later readmission was necessary because of renewed dysphagia. Again, dilatations were undertaken using the same anaesthetic techniques. Within the next 20 weeks 35 anaesthetics were given during the regime of treatment. Thereafter a 30-gauge bougie could be passed through the oesophagus without any difficulty. The boy was able to eat any kind of food and was discharged home in excellent condition.

Within the next 8 months 5 operations for either oesophagoscopy or dilatation were undertaken. As before, the same anaesthetic technique was used. When the patient was first admitted to hospital, hepatic function tests were not performed. Careful observation during the subsequent treatment revealed no signs of reaction to the premedication or the agents used during anaesthesia, indicative of hepatic malfunction. Slight pyrexia occurring on a few occasions on the day of treatment and lasting for not more than 3 days, was interpreted as reaction normally seen after such procedures. After the 24th anaesthetic he was sent to the paediatric department for his first hepatic function test (table I). No abnormal changes were found. During the following months he was examined thoroughly 3 more times. Except for a very slight increase in bromualothalaine retention in the second test, and of the α1 globulin on the third test, all laboratory results were within normal limits. A final examination was undertaken 17 months after the patient's last exposure to halothane. There was no indication of liver damage.

Altogether, during almost 14 months of treatment and surveillance 46 anaesthetics were given in the same manner (table II). The time of each individual exposure to halothane varied from 12 to 46 min (mean time 24.4 min) and the cumulative exposure time was 18 hours 44 min.

CORRESPONDENCE

A LEAD TOOTH-BRIDGE

Sir,—During laryngoscopy, it may be desirable to bridge gaps formed by absent or damaged maxillary teeth to prevent the blade of the laryngoscope from sinking into the breech. This may be effected by the use of malleable lead strips, cut from an old diathermy plate or "lead hand" and covered with adhesive strapping. A range of three sizes will be found to be adequate. They should be cut to a pointed oval shape, the long axes varying from 5 to 3 cm, the short from 3.5 to 2 cm. The plate may be bent across its short axis into an "L" shape. One limb lies across the upper incisor teeth bridging any gap. The other rests upon the upper lip. Alternatively, to protect a loose or damaged tooth or delicate conservation work, the plate may be bent across its long axis so that no pressure lies upon that particular tooth.

After use the adhesive tape is stripped off and replaced by fresh strapping prior to use again. The lead plate may be washed or autoclaved.

E. N. S. Fry
Stockton

RESPIRATORY MASS SPECTROMETER USERS' GROUP

Sir,—A preliminary meeting of a number of respiratory mass spectrometer users was held recently at the Clinical Research Centre. Because of the complexity and the range of potential medical applications of these instruments, it was agreed that a mass spectrometer users' group should be formed to provide a forum for discussion of technical problems and an exchange of ideas. We have prepared a preliminary list of mass spectrometer users in this area and would like to extend this to people who have experience of respiratory mass spectrometry who would be interested in participating in further meetings of this group.

M. J. Halsey
J. G. Jones
Clinical Research Centre, Harrow

MULTIPLE EXPOSURE TO HALOTHANE

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