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Continuous Positive Airway Pressure (CPAP) Using the Gregory Box

Gregory *et al.* (1971) in San Francisco reported treating 20 infants with severe respiratory distress syndrome (RDS) with continuous positive airway pressure (CPAP). Up to 12 mmHg pressure was delivered by endotracheal tube to 18 infants and via a head box to the remaining 2. Sixteen of the 20 infants survived, including 7 out of 10 weighing less than 1500 g at birth. In

October 1971 we adopted this form of treatment, and, in particular, the head chamber or Gregory box. Our early results have been reported elsewhere (Dunn *et al.* 1971, 1972, 1974). This paper deals with our first 15 months experience.

Management of RDS

Infants exhibiting signs of inspiratory retraction, expiratory grunting, oedema and poor alveolar air entry soon after birth, with or without tachypnoea and cyanosis, were initially given warmth to maintain a normal body temperature, oxygen as required to 35%, and carefully observed with minimal handling. Many such infants improved during the first few hours of life. They were classed as 'transitory RDS' and were not included in our RDS statistics. If, however, at the age of 4–6 hours the signs had not improved (or the infant had already required respiratory assistance) and characteristic radiological changes were present, a diagnosis of RDS was confirmed and the following standard treatment commenced.

Umbilical catheters were passed until their tips lay respectively in the inferior vena cava and lower aorta. Then, 5% dextrose/5% fructose solution was infused at a rate of 3 ml/kg per hour, increasing to 4 ml/kg per hour at 24 hours; oral feeding was rarely introduced before 48 hours. Monitoring of arterial blood gases and acid-base status was undertaken every 3–6 hours. The ambient oxygen concentration ($F_{I}O_2$) was adjusted

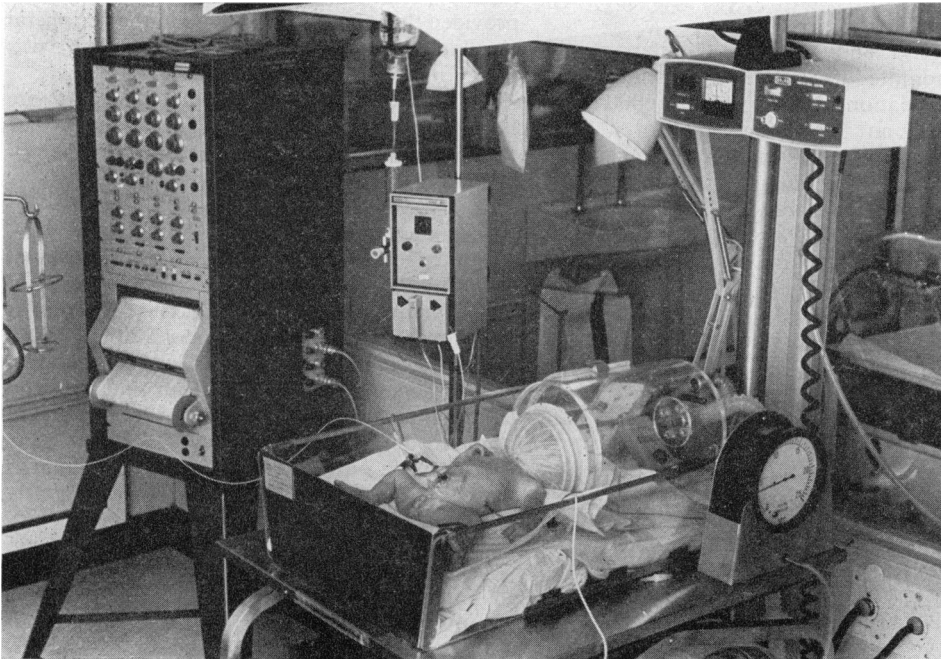


Fig 1 The Gregory box (by courtesy of Bernsen's International Press Service Limited)

until the arterial oxygen tension (P_{aO_2}) was between 50 and 70 mmHg. A course of antibiotics, usually ampicillin, was commenced at 24 hours. Small plasma transfusions were used when necessary to combat marked systemic hypotension. Occasionally $NaHCO_3$ was administered by slow i.v. infusion to counteract severe non-respiratory acidosis.

CPAP (Gregory box) was initiated, usually by the age of 6 hours, when the clinical and radiological signs of RDS were severe and an $F_{I_{O_2}}$ of 40–60% had failed to maintain the P_{aO_2} above 45 mmHg. Commencing with CPAP 6 mmHg and with $F_{I_{O_2}}$ unchanged, the P_{aO_2} was reestimated after 30 minutes. If, as was often the case, the P_{aO_2} had risen above 70 mmHg, the $F_{I_{O_2}}$ was reduced in stages until atmospheric levels were reached, usually over the following 24 hours; then the CPAP was reduced in 1 mmHg stages over a further 24 hours or so until discontinued.

Infants with severe respiratory depression, or having repeated apnoeic attacks, or remaining hypoxaemic in spite of the Gregory box (using maximum CPAP of 10 mmHg and $F_{I_{O_2}}$ of 80%) were treated by intermittent positive pressure respiration (IPPR). During the last 6 months of the study, the end expiratory pressure of such infants was usually maintained at 3–5 mmHg. It was often possible to then wean these infants off the ventilator onto CPAP, given first via an endotracheal tube and then via the Gregory box (Fig 1).

Results

Between 1.10.71 and 31.12.72, 47 infants with RDS were admitted to Southmead Hospital from a referral population of approximately 6000 newborn infants, an RDS incidence of 0.8%; 7 (14.9%) died (Fig 2). Post-mortem examination, undertaken on 6 of the 7 infants, revealed hyaline membrane and atelectasis in all, intraventricular haemorrhage in 5, subarachnoid haemorrhage in 2, bronchopulmonary dysplasia in 1, and pneumothorax in 1.

Twenty-eight infants (60%) received standard therapy alone and all survived; 3 infants (6%)

admitted in respiratory failure were treated with IPPR and all died; 7 infants (15%) were treated successfully with the Gregory box; the remaining 9 (10%) received a combination of CPAP and IPPR and 5 survived. Thus, of the 16 severely affected infants receiving CPAP, 4 died (25%).

A detailed analysis is so far only available for the 12 infants receiving CPAP during the first 12 months. There were 7 boys and 5 girls. They had a mean birthweight of 1810 g (range 1220–2330) and a mean gestational age of 32 weeks 3 days (range 29–35 weeks). Delivery was normal in 4, by forceps in 2, by the breech in 4, and by Caesarean section in 2. The mean Apgar score at 1 minute was 4, 6 infants requiring IPPR resuscitation at birth. Pregnancy complications included accidental antepartum haemorrhage in 4, multiple pregnancy in 2, premature rupture of the membranes in 2, and fetal distress in 4. Three infants were polycythaemic and received dilution exchange transfusion soon after birth.

Before commencing treatment with CPAP 6 mmHg (mean pressure) at an average age of 5 hours, these infants had a mean arterial pH of 7.09 and a P_{aO_2} of 46 mmHg in a $F_{I_{O_2}}$ of 49%. CPAP was used for a mean duration of 58 hours. In another report (Dunn 1974), we have shown that the introduction of CPAP 6 mmHg, while keeping the $F_{I_{O_2}}$ unchanged, resulted in an average 89% rise in P_{aO_2} from 53 to 100 mmHg. At the same time the arterial CO_2 tension usually fell rapidly. Metabolic acidosis was rarely troublesome provided the infant was kept warm, the peripheral circulation was maintained, and the P_{aO_2} was not allowed to fall below 45 mmHg.

All surviving infants treated with CPAP are being carefully followed up. Four are now over a year old. All appear to be physically and developmentally normal.

Discussion

The introduction of CPAP reduced our neonatal mortality from RDS from a previous level of approximately 33% to 14.9%. The reduction is all the more remarkable as at least 3 of the 7 fatal cases had almost certainly suffered intracranial haemorrhages before arrival in our unit. Also no less than half our deaths in the 15-month period occurred during one disastrous weekend in May (Fig 2) when our slender intensive care facilities were overwhelmed by a sudden influx of extremely ill infants, 6 of them requiring respiratory support. In addition, we undoubtedly had to learn from mistakes in the early months. It is interesting to note that neonatal mortality for the first 8 months was 20%, and for the last 7 months was 4.5%.

The success of CPAP was also evident in the striking clinical improvement that was frequently

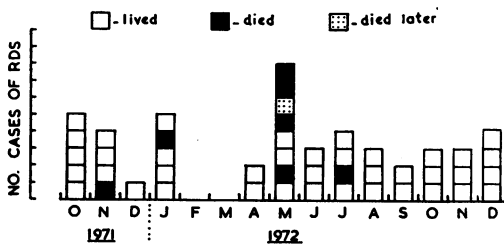


Fig 2 Histogram of RDS cases between October 1971 and December 1972

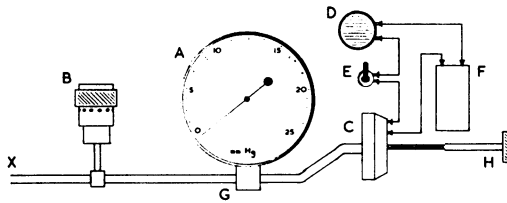


Fig 3 CPAP pressure-monitoring module with excess pressure dead-weight relief valve and adjustable low pressure alarm. A, manometer; B, safety valve (15 cm H₂O); C, constant pressure switch type QN; D, bleptone; E, on-off switch; F, battery; G, 3/16 inch T-joint and tubing; H, pressure alarm adjustment screw; X, inlet

observed. In most cases colour and peripheral circulation improved at once, activity increased, respiration deepened, and the signs of respiratory distress diminished. The radiological appearance of the chest X-ray also usually improved markedly. Such progress was rapidly reversed if CPAP was suddenly discontinued or the pressure reduced too quickly.

Because of the potential danger of sudden changes in Gregory box pressure, usually due to leaks around the neck, we developed a pressure monitoring module at the end of 1971 (Fig 3) which both warned of a fall in box pressure and also provided a relief valve in case the pressure rose above 15 cm H₂O.

A number of other potential hazards from the Gregory box deserve mention. Chilling is a danger if the gas blown into the Gregory box is not adequately warmed. Gastric dilatation is another theoretical hazard but has not been observed in our infants, perhaps because we have rarely exceeded CPAP 8 mmHg. Neck erosions from the edge of the diaphragm are liable to occur unless the skin of the neck is well protected by a collar of plastic foam or a firm pad of cotton wool and gauze; such a pad also improves the neck seal and diminishes the danger of obstructing venous return from the head. The potential hazards of decreased cardiac return and impaired pulmonary perfusion as a result of CPAP may be lessened by limiting the maximum pressure used and by steadily reducing the pressure as the infant recovers. Pneumothorax is also a theoretical danger and was encountered twice in our 16 cases; both infants had also received IPPR.

Many pædiatricians confronted with severe RDS counter any deterioration by raising the F₁O₂, and use CPAP or IPPR only as a last resort. We believe that a high F₁O₂ encourages atelectasis and may eventually produce bronchopulmonary dysplasia. Our experience also suggests that CPAP is much more effective if used before there is extensive atelectasis. For these

reasons we prefer to intervene relatively early in the course of the disease.

It may be argued that our results were influenced by selection of less severe cases for CPAP therapy. In fact, based on our previous experience, the expected mortality of the CPAP-treated infants might have been 80% rather than 25% as observed. As CPAP was begun nearly always before the age of 6 hours and there were no deaths among infants receiving standard therapy alone, it is possible to assess the severity of RDS at 4–6 hours from the gestational age, the clinical and radiological signs, the progression of the disease, and the blood gas findings.

In conclusion, CPAP using a Gregory box has proved to be an effective and relatively safe method of treating severe RDS, especially when used early in the course of the disease. The apparatus is cheap and the technique easy to learn and supervise. In many cases it dispenses with the need for an endotracheal tube and, by reducing the need for a high ambient oxygen, reduces the danger of oxygen toxicity. The apparent success of its use has induced a more positive and optimistic clinical attitude in our department to the management of very severe RDS.

Acknowledgments: I am grateful to pædiatric and nursing colleagues who assisted in the care of these infants and also to the Children's Research Fund, Liverpool, for financial assistance.

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Addendum (March 1974)

Since this paper was read our results for 1973 have become available. Six infants that required IPPR for respiratory failure from birth, due to intracranial bleeding, died. Thirty-three infants were admitted with RDS; 9 received CPAP and one also required IPPR. There was one death, an infant of 28 weeks' gestation, thought to be brain-damaged and not offered CPAP. The mean maximum CPAP used during 1973 fell to 4.7 mmHg. Further efforts were made to identify the most sick infants and to commence CPAP as early as possible (Dunn *et al.* 1973). Follow up of all CPAP survivors since 1971 has failed to reveal any significant pulmonary or developmental handicaps so far.—P M D

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