Discontinuation of continuous positive airways pressure in infants with respiratory distress syndrome

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SUMMARY Unexpected stability of arterial oxygen and carbon dioxide tensions occurred in infants recovering from respiratory distress syndrome (RDS) on the abrupt withdrawal of CPAP at 6 cmH₂O pressure. 30 preterm infants (birthweights 880–3200 g gestational ages 29–38 weeks) were treated at a mean age of 10 hours and for a mean duration of 62 hours. CPAP was stopped when F₁O₂ requirement fell below 0·3, and stable Po₂ was maintained for 4 hours. Discontinuation of CPAP at 6 cmH₂O resulted in a mean change in Po₂ from 66 to 64 mmHg (8·8 to 8·5 kPa) and a mean change in PCO₂ from 41 to 40 mmHg (5·4 to 5·3 kPa). We conclude that this population of infants suffering from moderate RDS tolerated the abrupt withdrawal of CPAP, and that gradual reduction of pressure was unnecessary.

It is now over 7 years since CPAP was recognised as an effective mode of treatment for infants suffering from respiratory distress syndrome (RDS) (Gregory et al., 1971). However there are still many unanswered questions before the maximum benefit-risk ratio is attained: the best time and mode for instituting treatment, the most efficient pressures, the techniques for monitoring complex physiological processes, and the optimal time and method for stopping treatment. With regard to this last, a prospective trial was designed to determine the effect on arterial blood-gas tensions of removing CPAP without the generally accepted weaning procedure (Llewellyn and Swyer, 1973; Krouskop et al., 1975; John et al., 1976; Stark and Taueusch, 1977).

Patients and methods

During the 7-month period from 1 July 1977 to 31 January 1978, 41 infants suffering from RDS were admitted to the neonatal intensive care unit. Of these infants, 30 required treatment with CPAP as the sole form of support, while 11 were treated with mechanical ventilation. The former group of infants comprised the study group.

CPAP was delivered by a nasal prong device (Wung et al., 1975) at a pressure of 6 cmH₂O. This applied pressure measured at the nares was kept constant and F₁O₂ was altered throughout the course of treatment in order to maintain Po₂ in the 50–80 mmHg range. The criteria for stopping CPAP were: ambient oxygen requirement of 0·3 or less to maintain Po₂ above 50 mmHg, a stable Po₂ at this level for support for 4 hours, and the absence of clinical signs of respiratory distress. Arterial blood samples were drawn from the umbilical catheter about 20 or 30 minutes before and after the withdrawal of CPAP and measured on an IL oxygen analyser (Instrumentation Laboratories: Lexington, Mass.).

The mean birthweight of the infants was 1920 g (range 800 to 3200) and the mean gestational age 34 weeks (range 29 to 38). Nine infants weighed <1500 g, 9 were between 1500 and 2000 g, and 12 were >2000 g.

RDS was determined by x-ray and clinical evaluation to be of moderate severity. A-aDo₂ values were calculated at the time that treatment with CPAP was started for a precise assessment of the disease. The A-aDo₂ before treatment was 217 ± 110 mmHg (mean ± SD). 70% of the A-aDo₂ values were between 100 and 300 mmHg, and 23% were between 300 and 600 mmHg.

Results

CPAP was begun at a mean age of 10 ± 10 hours (mean ± SD). The mean duration of CPAP was 62 hours, SD 36. 14 infants reached the criteria for withdrawal by 48 hours whereas the remainder required 48 to 160 hours to reach this goal.

The response of the arterial blood-gases to the removal of CPAP was evaluated at a F₁O₂ of 0·26 ± 0·04 at an A-aDo₂ of 71 ± 28 mmHg. The Po₂ on CPAP was 66 ± 9 mmHg and off CPAP it was 64 ± 10 mmHg (P>0·5). The Pco₂ was 41 ± 7 mmHg before CPAP withdrawal and 40 ± 8 mmHg after (P>0·5). The distribution of Po₂ and Pco₂ values before and after CPAP was comparable (Figure). None of the infants needed to return to CPAP after it had been stopped.

Examination of the individual responses showed that 10 Po₂ values increased and 20 decreased, while
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Before this prospective trial, the procedure for stopping CPAP in the treatment of infants with RDS had not been critically assessed, although textbooks (Llewellyn and Swyer, 1973; Pierog and Ferrara, 1976) and papers (Krouskop et al., 1975; Stark and Taehus, 1977) have suggested that weaning is the optimal method of CPAP removal.

The results of this study show that the abrupt removal of CPAP did not place any infant in a state of hypoxia or hyperoxia. The possibility that the criteria for stopping CPAP resulted in some infants being treated longer than necessary is indicated by the correlations. The positive correlation between duration and PCO₂ before withdrawal as well as the negative one between P0₂ before withdrawal and the P0₂ response, suggest that towards the end of treatment CPAP may have had a deleterious effect. These results suggest that in some infants recovering from RDS, prolonged use of constant distending pressure can cause slight P0₂ decrease and CO₂ retention. This observation has previously been reported by Nelson et al. (1977). Therefore abrupt withdrawal could have been completed earlier in the course of recovery.

Several hypotheses may be considered as possible explanations for the blood-gas responses to CPAP withdrawal. The removal of a constant distending pressure may result in a new ventilation perfusion relationship or in the initiation of compensatory mechanisms by the infant.

We conclude that our population of infants recovering from moderate RDS tolerated the abrupt removal of CPAP, so that gradual reduction of pressure was unnecessary. In some infants prolonged use of CPAP had deleterious effects.

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References


Table Comparison of the P0₂ response subgroups

<table>
<thead>
<tr>
<th></th>
<th>Increased (n = 2)</th>
<th>Unchanged (n = 20)</th>
<th>Decreased (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight (mean ± SD g)</td>
<td>2240 ± 790</td>
<td>1805 ± 558</td>
<td>2080 ± 348</td>
</tr>
<tr>
<td>Age at start (hours)</td>
<td>6 ± 4</td>
<td>10 ± 8</td>
<td>16 ± 16</td>
</tr>
<tr>
<td>A-aDO₂ start (mmHg)</td>
<td>210 ± 90</td>
<td>238 ± 123</td>
<td>197 ± 70</td>
</tr>
<tr>
<td>P0₂ prior (mmHg)</td>
<td>57 ± 4*</td>
<td>67 ± 8</td>
<td>76 ± 4</td>
</tr>
<tr>
<td>Change in P0₂ (mmHg)</td>
<td>–4 ± 6</td>
<td>–1 ± 5</td>
<td>3 ± 4</td>
</tr>
</tbody>
</table>

Conversion: traditional to SI units—A-aDO₂, P0₂, and PCO₂ 1 mmHg = 0.133 kPa.

*P < 0.05.
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