Patient triggered ventilation using a flow triggered system

Michael F Hird, Anne Greenough

Abstract

The role of patient triggered ventilation (PTV) for the newborn was assessed using a new patient triggered ventilator, the Draeger Babylog 8000, which incorporates significant improvements in both ventilator performance and the triggering system. Thirty three infants, median gestational age 30 weeks and postnatal age 2-5 days, were entered into the study to compare blood gases obtained during conventional and patient triggered ventilation. Oxygenation did not improve with PTV in the group overall but increased significantly (median change 7%) in infants greater than 28 weeks' gestation. Arterial carbon dioxide tension (Paco2) decreased during PTV in the majority of infants (median reduction 7%), this was not related to the gestational or postnatal age, but was greatest in infants previously on a low conventional ventilation rate. Blood gases (both Paco2 and Paco2) deteriorated in infants requiring the highest inspired oxygen concentration. We conclude that patient triggered ventilation is most useful in infants with mild respiratory distress.

Patient triggered ventilation (PTV) was reintroduced as a method of respiratory support for neonates in 1986.1 Since that time there have been a series of studies which have assessed both the possible short1-3 and long term benefits of PTV.4,5 In the majority of infants oxygenation acutely improves on transfer from conventional ventilation to PTV1-3 but PTV fails to provide a successful method of respiratory support in infants of less than 28 weeks' gestation6 or if it is started in the first 24 hours of life.5 A predictor of failure of PTV was an asynchronous respiratory interaction, with inflation occurring either late in inspiration or even in expiration, as a result of a long trigger delay.5 All these studies have employed a modified conventional neonatal ventilator (model HV 2000, SLE) with a variety of triggering systems.

Recently, purpose built patient triggering ventilators have appeared. We have demonstrated that one such ventilator (Draeger Babylog 8000) has a performance superior to the SLE, with a much shorter trigger delay: a median of 80-100 ms (Draeger) compared with a median of 200-250 ms (SLE).8 The Draeger Babylog 8000 also has the advantage of no inadvertent positive end expiratory pressure (PEEP) at high flow rates9 or the fast ventilator rates which are frequently triggered by very immature infants.8 Thus it seems likely that this ventilator could be used with success in a more varied group of infants. Our aim, therefore, was to use the Draeger ventilator to redefine the optimal role of PTV.

Methods

Infants were entered into the study when they had been stable on conventional ventilation for at least two hours and when a ventilator became available. All infants had previously been ventilated using Sechrist ventilators and had blood gases within the clinically acceptable range (pH 7.25-7.4, arterial oxygen tension (Pao2) 5-33-9-33 kPa, arterial carbon dioxide tension (Paco2) 4-67-6-67 kPa).

The patients were then changed over to the Draeger Babylog 8000 ventilator and ventilated conventionally for one hour at the same settings as were previously in use. The ventilator was then switched into patient triggered mode for at least one hour. The Draeger Babylog 8000 utilises an airflow triggering system, a hot wire anemometer, which has the advantage of not increasing the dead space of the patient circuit.

We have demonstrated that the median trigger delay of this system is between 80 and 100 ms at inflation times between 0-24 and 0-4 seconds.8 On switching into the patient triggered mode the peak inspiratory pressure, PEEP, gas flow rate, and inspired oxygen concentration were not changed. An inflation time initially of 0-4 seconds was used, reducing in steps of 0-02 seconds, if necessary, until the spontaneous respiratory effort was no longer visibly distinct from the positive pressure inflation.

Throughout the study infants were monitored continuously using a Searle intra-arterial electrode or transcutaneous oxygen electrode. Infants were withdrawn from the study if, during PTV, their continuous monitoring demonstrated that the Pao2 was below 5-33 kPa. Arterial blood gas samples were obtained from the indwelling arterial line one hour after changing to the Draeger ventilator used conventionally and then again after one hour of trigger ventilation.

ANALYSIS

Differences in Paco2 and Pao2 between study periods were assessed for significance using a paired Wilcoxon rank sum test. The change in Pao2 and Paco2 over the first hour of PTV was expressed as a percentage of the values obtained immediately before PTV; these were then related to the infant's gestational and postnatal age and the severity of the infant's respiratory
disease. Relationships were assessed for significance using a Wilcoxon rank sum test or Fisher's exact test.

Patients
Thirty three infants, all less than 35 weeks' gestational age, were recruited into the study. Their median (range) birth weight was 1508 (592–2700) g and gestational age 30 (24–34) weeks; they were studied at a median (range) postnatal age of 2.5 (0.25–50) days. The infants were all ventilated because of respiratory distress associated with prematurity. Immediately before the study their median (range) ventilator rate was 25 (4–90) breath/minute, peak (range) inspiratory pressure 17 (12–25) cmH2O and inspired oxygen concentration 35 (21–80)%.

Ethical permission for this study was granted by the King's College Hospital ethics committee.

Results
No infant had to be withdrawn from the study because of low oxygen concentrations demonstrated by continuous monitoring. On analysis of the arterial blood gas concentrations at one hour, however, one infant (26 weeks' gestation) had a PaO2 of 4·80 kPa, although her continuous monitoring had suggested her oxygenation to be at the higher level of 5·33 kPa.

No significant change was found in PaO2 when blood gases were compared between the period of conventional ventilation and PTV; PaO2 increased in 17 patients, decreased in 13 and there was no change in the remaining three infants (fig 1). Oxygenation significantly improved in infants greater than 28 weeks' gestational age (p<0.05), with a median (range) improvement in PaO2 of 7 (−28 to +42%)%. Fifteen of the 23 infants of gestational age greater than 28 weeks had an improvement in oxygenation, compared with only two of the 10 infants less than 28 weeks gestational age (p<0.05). Oxygenation also significantly improved in the subgroup of infants of greater than 28 weeks' gestation who were ventilated at a rate of more than 15 breaths/minute (p<0.05). There were no significant relationships between changes in oxygenation and postnatal age, fractional inspired oxygen concentration (FiO2) or peak inspiratory pressure (table 1).

Paco2 showed a significant decrease over the PTV study period, decreasing in 21 patients and increasing in 12 patients, medium (range) change −7 (−43 to +29) % (p<0.05). This change in Paco2 was neither significantly related to gestational nor postnatal age (fig 1). In the 12 patients whose PaCO2 increased, the median ventilator rate on conventional ventilation was significantly higher than in the 21 patients in whom PaCO2 decreased (p<0.05)(fig 2, table 2).

No significant relationships were found between changes in PaCO2 and either the peak inspiratory pressure or improved oxygen concentration (FiO2) (table 2).

The improvement in oxygenation was associated with a reduction in PaCO2 in 12 patients, but a rise in PaCO2 in five others. These five infants had been previously ventilated at a significantly higher rate than the rest of the study group (p<0.05). A deterioration in PaO2 was accompanied by a fall in PaCO2 in nine patients (these infants were significantly less mature, but older than the rest of the study group; p<0.05) and a rise in PaCO2 in seven others. Infants in whom there was no improvement in either PaO2 or PaCO2 required a higher inspired oxygen concentration (p=0·054) and peak inflating pressure than the remaining infants (table 3). Infants whose blood gases improved on PTV were maintained on this mode for up to several days or until extubation; no complications were experienced.

![Figure 1 Percentage change in PaO2 and PaCO2 during PTV related to gestational age. Individual patient data is demonstrated by separate bars.](http://adc.bmj.com/)

Table 1 Changes in oxygenation. Results are median (range)

<table>
<thead>
<tr>
<th>Increase or decrease in PaO2</th>
<th>No change in PaO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of infants</td>
<td>Ventilator rate during conventional ventilation (breaths/min)</td>
</tr>
<tr>
<td>No of infants</td>
<td>FiO2 (%)</td>
</tr>
</tbody>
</table>
when compared with similar pressure settings on conventional ventilation. This improvement in alveolar ventilation on PTV compared with conventional ventilation is particularly likely with the short inflation times used in this study for infants of all gestational ages. Thus, it is not unexpected that changes in Paco₂ were not related to the maturity of the infant. Oxygenation, however, is determined by mean airway pressure (MAP). Inflation time during PTV was always shorter than conventional ventilation, particularly in the smallest infants; this would reduce MAP and hence oxygenation.

The only significant relationship of change in Paco₂ was with the ventilator rate previously used on conventional ventilation. At low rates during conventional ventilation only a small proportion of spontaneous breaths can be supported by a positive pressure inflation. In contrast, during PTV, all spontaneous breaths may trigger the ventilator, resulting in both an increase in tidal and minute volume. Thus Paco₂ will be affected to a greater extent by changing to PTV from conventional ventilation at a low ventilator rate than a high one. The greater reduction in Paco₂ at lower ventilator rates suggests PTV may have a useful role in weaning when infants are recovering from respiratory distress syndrome and are usually supported on intermittent mandatory ventilation.

We have previously demonstrated that failure of oxygenation to improve after one hour is an accurate predictor of a lack of successful respiratory support by PTV. The results of this study, therefore, indicate that despite improvements in ventilator design and performance there remains a substantial proportion of infants for whom PTV is not a useful alternative to conventional ventilation. The failure to improve oxygenation in the most immature infants, the greater reduction in Paco₂ at previously low ventilator rates and the combination of failure of oxygenation and increase in Paco₂ in infants at the highest inspired oxygen concentration all suggest PTV is more suitable for infants with mild, not severe, respiratory distress.

Dr M Hird is supported by Children Nationwide Medical Research Fund. We thank Ms Sue Williams for secretarial assistance.

Discussion

Initial inspection of the results might suggest that PTV appeared to confer no advantage in improving oxygenation over conventional ventilation, as there was no significant improvement in the study population overall. Oxygenation, however, improved in infants of gestational age of more than 28 weeks, with a similar improvement in the subset of that group ventilated at rates of more than 15 breaths/minute. There was a deterioration in oxygenation in the group of infants of gestational age 28 weeks or less, which confirms our preliminary findings in five other immature infants. Thus, despite using a superior ventilator design and performance, PTV is still unsuitable for infants of extreme prematurity.

In contrast, changes in Paco₂ were not significantly related to gestational age. During PTV, tidal volume and minute volume are increased

Table 2 Changes in Paco₂. Results are median (range)

<table>
<thead>
<tr>
<th>Change in</th>
<th>Increase in Paco₂</th>
<th>Decrease in Paco₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of infants</td>
<td>12</td>
<td>21</td>
</tr>
<tr>
<td>Ventilator rate during conventional ventilation (breaths/min)</td>
<td>35 (15–90)</td>
<td>20 (4–65)</td>
</tr>
<tr>
<td>Pao₂ (%)</td>
<td>35</td>
<td>20</td>
</tr>
<tr>
<td>Peak inspiratory pressure (cmH₂O)</td>
<td>16 (12–25)</td>
<td>17 (12–22)</td>
</tr>
<tr>
<td>Postnatal age (days)</td>
<td>2 (0–25–50)</td>
<td>2.5 (0–25–46)</td>
</tr>
</tbody>
</table>

Table 3 Relationships of the changes in blood gases. Results are median (range)

<table>
<thead>
<tr>
<th>Change in PaO₂</th>
<th>Increase in PaO₂</th>
<th>Decrease in PaO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational age (weeks)</td>
<td>31 (25–34)</td>
<td>32 (25–34)</td>
</tr>
<tr>
<td>Postnatal age (days)</td>
<td>2 (0–25–6)</td>
<td>3 (0–25–4)</td>
</tr>
<tr>
<td>Ventilator rate before PTV (breaths/min)</td>
<td>25 (0–25–7)</td>
<td>20 (0–25–4)</td>
</tr>
<tr>
<td>Peak inspiratory pressure (cmH₂O)</td>
<td>17 (12–22)</td>
<td>16 (12–18)</td>
</tr>
<tr>
<td>Pao₂ (%)</td>
<td>30 (21–80)</td>
<td>30 (21–50)</td>
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Arch Dis Child 1991 66: 1140-1142
doi: 10.1136/adc.66.10_Spec_No.1140

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