

# Patient triggered ventilation in chronically ventilator-dependent infants

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#### Summary

Abstract. Patient triggered ventilation (PTV) has been assessed as a method of respiratory support in infants remaining ventilator-dependent beyond the 1st week of life. Sixteen preterm infants were studied who had a median gestational age of 26 weeks and postnatal age of 22 days. PTV was delivered using a ventilator incorporating an airway pressure trigger. PTV was only successfully maintained until extubation in 3 infants, failing to provide a satisfactory method of respiratory support in the remaining 13 infants after a median of 1 h (range 1-10). One of the 13 infants was persistently asynchronous at 1 h despite manipulation of inflation time. The other 12 infants, at failure of PTV, were making respiratory efforts which were inadequate to consistently trigger the ventilator. Infants in whom PTV was successful were older, more mature and of greater birth weight; the trigger delay at 1 h was significantly shorter in this group (P < 0.05). A predictor of failure of PTV was asynchrony in the 1st h after commencing PTV (P < 0.02). We conclude PTV incorporating an airway pressure trigger infrequently provides a useful method of respiratory support in infants who are chronically ventilator-dependent.

**Key words:** Patient triggered ventilation – Chronic lung disease – Preterm infants

# Introduction

Minimisation of barotrauma is a priority in infants ventilated beyond the 1st week of life and at high risk of developing chronic lung disease. In infants suffering from acute respiratory disease, patient triggered ventilation (PTV) can improve oxygenation when compared to conventional ventilation [3, 6], enabling peak inspiratory

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Abbreviation: PTV = patient triggered ventilation

pressures to be reduced. Further evidence suggesting PTV might be associated with a reduction in barotrauma is the finding of a lower incidence of pneumothorax in infants ventilated by this method, although comparison was made only with historical controls [1]. The aim of this study was to determine if PTV was a useful method of respiratory support in infants ventilated beyond 1 week of age and to determine factors influencing its success.

### Methods

Infants were eligible for the study if they remained ventilated beyond 1 week of age. They were entered into the study according to the availability of the ventilator, a modified SLE Newborn 250 ventilator (SLE Ltd, Croydon, UK). An airway pressure trigger system was employed. Airway pressure changes were measured from a small T-piece attached just proximal to the endotracheal tube. A change of more than  $0.5 \text{ cmH}_2\text{O}$  triggered the ventilator, resulting in delivery of a single positive pressure inflation; this represented the maximum sensitivity of the triggering device. If the critical airway pressure change was not exceeded by the infant within an 8s period, the ventilator automatically switched back into conventional mode for 60 s at the ventilator rate previously used during conventional ventilation.

Patients had all been initially ventilated using the Sechrist ventilator (EME Ltd, Brighton, UK). They were transferred to the SLE ventilator with the settings unchanged. If, after 2 h, their condition remained stable with blood gases within the clinically acceptable range (pH 7.25–7.4, PaCO<sub>2</sub> 35–50, PaO<sub>2</sub> 50–70 mm Hg) they were then switched to patient triggered mode. No change was made in the ventilator pressures, gas flow rate (81/min) and inspired oxygen concentration immediately on commencement of PTV. The inflation times, however, were altered after observation of the inflation times, however, were altered after observation of the inflation time (J1, appropriate compensation for the systems delay being made. Inflation times ranged from 0.20-0.4 s. The inflation time was progressively reduced until the inflat's respiratory efforts were indistinct from the ventilator inflation [11].

Respiratory measurements were made using the system that has been previously described [4]. Flow, volume, oesophageal and airway pressure changes were simultaneously recorded onto a Gould Polygraph chart recorder (Model 2800 S, Gould Electronics Ltd, Ilford, UK). Recordings were made over a 20min period. The first recording was usually made after 1 h of PTV to allow the infant time to adapt to the new mode of ventilation, and only earlier if the infant remained obviously asynchronous despite manipulation of the inflation time. Recordings were subsequently made twice daily and, in addition, if the infant's respiratory efforts became distinct from the positive pressure inflation. From the recordings the infant's respiratory interaction with the ventilator inflation was determined. The infant's interaction was classified as that seen on at least 80% of the positive pressure inflation [5]. Synchrony was defined as ventilator inflation commencing early in inspiration and terminating prior to spontaneous expiration. Asynchrony was defined as ventilator inflation extending into spontaneous expiration [7].

The trigger delay was also calculated from the respiratory recordings. This was defined as the delay between the onset of spontaneous inspiration (denoted by the negative deflection in the oesophageal pressure trace) and the commencement of positive pressure inflation. The trigger delay was calculated as the mean of the delays seen over the respiratory recording made at 1 h.

Blood gases were measured from an indwelling arterial catheter sited for clinical purposes. Arterial samples were obtained immediately prior to the commencement of PTV. Further samples were obtained after 1 h of PTV unless PTV was considered to have already failed (see later) and subsequently as clinically indicated. During PTV, peak inspiratory pressures and the inspired oxygen concentration were altered accordingly to maintain blood gases within the clinically acceptable range until extubation [11].

PTV was considered to have failed to provide a successful method of respiratory support if the infant had to be returned to conventional ventilation before extubation. Indications for the infant to be returned to conventional ventilation were:

1. Persistent appoea in the 1st h of commencing PTV.

2. Persistent asynchrony despite manipulation of inflation time

[11] with respiratory efforts remaining distinct from positive pressure inflation.

3. Irregular triggering, such that the ventilator was automatically switching back to conventional mode on at least 10 occasions in the previous 20 min.

4. Irregular triggering, although less frequent than in category (3), was associated with the development of acidosis (pH < 7.25).

#### Patients

Sixteen preterm infants with a median gestational age of 26 weeks (range 24–30 weeks), birth weight of 802 g (550-2704 g) and postnatal age of 22 days (range 8–88 days) were studied. Thirteen of the infants had a birth weight of less than 1500 g and 10 were less than 28 weeks gestational age. All had been ventilated from birth and had suffered from respiratory distress syndrome. Ventilators support had initially been via Sechrist IV ventilators employing rates equal to or in excess of 60 breaths/min during the acute stage of the respiratory illness. At entry to the study the median ventilator rate was 30 breaths/min (range 5–56); peak inspiratory pressure 17 cm H<sub>2</sub>O (range 12–28) and inspired oxygen concentration 35% (range 21–90). All infants were receiving theophylline (4 mg/kg per day) during the study.

The study was approved by the King's College Hospital Ethics Committee.

#### Statistical analysis

Differences between infants in whom PTV was successful and those in whom it failed were assessed for statistical significance using the Wilcoxon rank sum test or Fisher's exact test.

Table 1. Factors influencing outcome of PT	Table 1	. Factors	influencing	outcome	of PTV
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Median (range)	Success	Failure	
n	3	13	
Birth weight (g)	996 (592-2704)	802 (550-1644)	NS
Gestational age (weeks)	29 (24–29)	26 (24–30)	NS
Postnatal age (days)	31 (9–50)	18 (8–72)	NS
Trigger delay (ms)	180 (150-300)	325 (225-500)	P<0.05

NS, Non significant

#### Results

PTV was successfully maintained until extubation in only 3 of the 16 infants. The median duration of PTV in these three patients was 60 h (range 5-75 h). PTV failed in the remaining 13 infants. In 2 infants failure of PTV was due to the nature of the infant's respiratory efforts. One infant became apnoeic and the other was persistently asynchronous in the 1st h of PTV, despite manipulation of the inflation time. The remaining 11 infants failed to consistently generate the critical airway pressure change necessary to trigger the ventilator. Six of these 11 infants were triggering the ventilator so irregularly (category 3) that they had to be returned to conventional ventilation within 1 h of commencing PTV. The remaining 5 infants, although initially successful on PTV, subsequently made irregular respiratory efforts and an acidosis developed (category 4). The median duration of PTV in the 13 infants in whom PTV failed was 1 h (range 0.3–10 h).

PTV tended to be successful in more mature infants and those of greater birth weight and postnatal age (Table 1). The trigger delay was significantly shorter in this group (P < 0.05). Analysis of the respiratory recordings made after 1 h on PTV revealed all three infants in whom PTV had succeeded were synchronous compared to only 1 of the 13 in whom PTV failed (P < 0.02).

#### Discussion

In this study we have demonstrated that PTV is infrequently a useful method of respiratory support for infants ventilated after the 1st week of life. Failure of PTV was particularly common amongst the least immature infants. We had included infants of less than 28 weeks gestational age as, although PTV had been unsuccessful in this group in acute respiratory distress [11], it was hoped that with advancing postnatal age these immature infants might have improved respiratory effort and hence triggering ability. Such immature infants represent a large proportion of those who remain chronically ventilatordependent and thus it is important to determine if this form of ventilation is useful in this group.

It is possible that ventilator malfunction may have explained failure of PTV in some of the infants. Infants ventilated after 1 week of age frequently maintain a relatively rapid spontaneous respiratory rate [8] which is likely to result in a fast triggering rate. Unfortunately, the SLE ventilator is known to malfunction at very fast rates [2] and this could have impaired gas exchange.

Infants with chronic lung disease have a high airways resistance [9], overcoming this high resistance could have increased the time necessary to generate the airway pressure changes critical to trigger the ventilator. This would explain the long trigger delay in certain infants we studied. The long trigger delay meant that inflation only commenced late in inspiration and thus the majority of inspiration was unsupported by positive pressure inflation, reducing its efficiency. The long trigger delay caused asynchrony [11] which, when demonstrated at 1 h, as shown previously [7], was an accurate predictor of failure of PTV.

The airway pressure trigger used in this study had been demonstrated to be the superior triggering system when used in infants with acute respiratory distress. It had been associated with a shorter trigger delay and greater sensitivity than the system incorporating a Graseby capsule [10]. Infants with chronic lung disease may make considerable respiratory effort but these efforts, because of the high airways resistance, may not create large airway pressure or flow changes. Thus the critical airway pressure necessary to trigger the ventilator is not always generated. Such respiratory efforts may be more successfully detected by body movement sensors.

We conclude that PTV incorporating an airway pressure triggering system is rarely a useful method of respiratory support in infants ventilated beyond the 1st week of life. Alternative triggering systems are being explored. Acknowledgements. Dr. M. F. Hird, Research, Fellow, is supported by the Children Nationwide Medical Research Fund. We thank Ms Sue Williams for secretarial assistance.

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