Patient triggered ventilation in premature neonates

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SUMMARY Patient triggered ventilation using oesophageal pressure changes was assessed in eight premature neonates. Respiratory activity was reliably recorded and positive pressure inflation occurred synchronously with inspiration. Peristalsis resulted in only minimal interference. During patient triggered ventilation, inflating volume and oxygenation increased significantly compared with periods of conventional ventilation.

Synchronous respiration with artificial ventilation in premature neonates improves oxygenation,[1] but attempts to achieve such a response by imposing a standard rate are not always successful.[2] Patient triggered ventilation—the delivery of positive pressure inflation initiated only by respiratory efforts—ensures synchrony but until recently was not practical in preterm neonates. A recent report suggested that it was possible to modify a conventional neonatal ventilator so that it could be triggered even by preterm infants' respiratory efforts.[3] Changes in abdominal expansion were used to detect respiration,[3] but these signals may be confused by gross body movements. During mechanical ventilation, oesophageal pressure changes were shown to be reliable in detecting respiratory efforts, and they were not affected by ventilator pressure changes or by postural movements.[1 4] The aims of the present study were to investigate the apparent success of triggered ventilation in preterm neonates and to assess the effect of oesophageal pressure changes as the trigger.

Patients and methods

Eight patients with the respiratory distress syndrome were studied; all were less than 1 week old with a mean gestational age of 27 weeks (range 24–31). Ventilatory support was given by an SLE Newborn 250 ventilator. Ventilator settings were determined before the study by the clinician in charge of the infant; rates varied from 50–80/minute, and peak inspiratory pressures from 14–24 cm H_2O. No infant was either paralysed or sedated, but those on ventilators with rates of less than 30/minute were treated with a methylxanthine. Permission for the study was given by the hospital's ethics committee.

Infants were entered into the study as soon as they had been stable on conventional ventilation for at least two hours. They were then switched to continuous positive airways pressure trigger ventilation for at least 30 minutes. Trigger ventilation was discontinued if the infant was either apnoeic for longer than 20 seconds, or failed to trigger a ventilator pulse during that time.

Patient triggered ventilation was delivered by a modified SLE Newborn 250 ventilator. The ventilator has a manual breath control which, when set for continuous positive airways pressure, permits delivery of a single positive pressure inflation of the same magnitude and duration as that predetermined during intermittent positive pressure ventilation. Only if a critical change in oesophageal pressure is exceeded (increase in negative pressure swings) is the manual breath control triggered (a systems delay of up to 100 milliseconds is possible).

Throughout periods of patient triggered ventilation and the preceding and succeeding 30 minute periods of conventional ventilation we recorded transcutaneous oxygen tensions, inflating volume, and ventilator and oesophageal pressure changes using the techniques previously described.[1 2 4] From the oesophageal pressure record we calculated the change in pressure necessary to trigger the ventilator, the infant's respiratory rate, and the number of periods of apnoea and peristalsis. Ventilator rate and inflating volume during different types of ventilation were compared, as was the mean transcutaneous oxygen tension during the final 10 minutes of each 30 minute period.

Statistical analysis was by paired Student's *t* test.

Results

Oesophageal pressure changes of 0·3–0·5 cm H_2O triggered the ventilator; these were greater than the pressure change from the cardiac artefact. Only one infant who had previously been apnoeic had periods of respiratory efforts that were not strong enough to trigger the ventilator. Apnoeic periods were infrequent despite the fact that three of the infants had previously been fully ventilated. The longest period of apnoea was 18 seconds and occurred in an infant

Table *Comparison of conventional and patient triggered ventilation. Figures are mean (range)*

Measurement	*Conventional ventilation*	*Triggered ventilation*
Tidal volume (ml)	6·8 (5–11)	8·2 (5·6–12·3)
Delivery rate of inflating pressure:		
Conventional rate ≥60/minute (n=3)	72 (60–80)	78 (72–90)
Conventional rate <60/minute (n=5)	17 (15–30)	45 (30–52)

previously on a rate of five breaths/minute. Otherwise, apnoeic episodes varied in length between two and 10 seconds and were less than one per minute. Peristaltic waves did not trigger the ventilator, but did interfere with the signal. This was only a problem in three infants, and then only for the first two minutes. The table shows that in seven of eight infants the inflating volume increased significantly during triggered ventilation ($p<0·02$). Delivery of inflating pulses increased in all previously ventilated infants at a rate of less than 60 breaths/minute. Trancutaneous oxygen tensions increased during triggered ventilation in all infants when compared with both periods of conventional ventilation (mean increase 12 mm Hg, range 5–30 mm Hg, $p<0·01$).

One infant remained on triggered ventilation for a total of three hours. After only 15 minutes his PaO_2 rose from 67 to 87 mm Hg with an increase in inflating volume of 11–12·3 ml when compared with conventional ventilation. Peak inspiratory pressure was gradually reduced from 14–10 cm H_2O with maintenance of arterial blood gases (pH 7·27, $PaCO_2$ 36 mm Hg, and PaO_2 67 mm Hg at 14 cm H_2O on conventional ventilation; and pH 7·27, $PaCO_2$ 38, PaO_2 84 mm Hg at 10 cm H_2O after one hour of triggered ventilation).

Discussion

Our results support previous findings that patient triggered ventilation, even in very premature infants, can be successful[3]: oxygenation improved, partly due to an increase in inflating volume during triggered ventilation. This increase reflected the difference in the infants' respiratory efforts—synchrony during trigger mode, but asynchrony and limitation of inflation during 'conventional' ventilation. In infants who had been ventilated at a rate of less than 60 breaths/minute the improvement in oxygenation was also due to the increase in the number of inflations delivered during triggered ventilation.

Small changes in oesophageal pressure successfully triggered positive pressure inflation. To decrease the frequency of ventilator breaths trigger sensitivity had to be reduced. The variation in the strength of respiratory efforts was insufficient to be certain that positive pressure inflation was delivered only on a certain number of respiratory efforts. Ideally—to facilitate weaning—triggering from a preselected number of breaths (regardless of size or frequency) should be incorporated into the ventilators as with 'adult' servo-ventilators.

The oesophagus has the advantage as a trigger site that gross body movements are not registered as pressure changes which could trigger the ventilator. Peristaltic activity, a theoretical disadvantage of this site, caused relatively few problems but these could be increased by the pooling of saliva after prolonged use. Both trigger sites studied to date—oesophagus and abdomen—[3] have a major disadvantage in that they fail to differentiate between obstructive and non-obstructive respiratory efforts and fail to set off the alarm during obstructive episodes despite the absence of air flow.[5]

We conclude that patient triggered ventilation can be successful, particularly during weaning. By conversion to synchronous respiration oxygenation was improved and this might reduce the incidence of pneumothoraces.[2] Using changes in air flow as the trigger could be advantageous, as this is a more accurate indicator of both respiratory efforts and apnoea. This is currently being investigated.

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References

1 Greenough A, Pool J, Greenall F, Morley C, Gamsu H. Comparison of different rates of artifical ventilation in premature neonates with RDS. *Acta Paediatr Scand* 1987;**76:**706–12.
2 Greenough A, Morley CJ, Pool J. Fighting the ventilator – are fast rates an effective alternative to paralysis? *Early Hum Dev* 1986;**13:**189–94.
3 Mehta A, Callan K, Wright BM, Stacey TE. Patient triggered ventilation in the newborn. *Lancet* 1986;**ii:**17–9.
4 Greenough A, Morley CJ. Oesophageal pressure measurements in ventilated preterm babies. *Arch Dis Child* 1982;**57:**851–5.
5 Southall DP, Richards JM, Lau KC, Shinebourne EA. An explanation for failure of impedence apnoea alarm systems. *Arch Dis Child* 1980;**55:**63–5.

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