

Manipulation of ventilator settings to prevent active expiration against positive pressure inflation

D FIELD, A D MILNER, AND I E HOPKIN

Department of Neonatal Medicine and Surgery, City Hospital, Nottingham

SUMMARY Recent publications have suggested that in infants receiving artificial ventilatory support a particular pattern of interaction between spontaneous breaths and ventilator inflations (active expiration against each ventilator inflation) may be important in the production of pneumothoraces. We have looked at patterns of interaction from 47 preterm infants studied on 51 occasions. We found that active expiration against the ventilator occurred on a total of 16 occasions. This pattern was prevented on 14 occasions by altering the ventilator settings. In two other babies, the pattern persisted but neither baby developed a pneumothorax.

Recently, Greenough *et al*¹ described various patterns of breathing they have observed in infants of less than 34 weeks' gestation who were receiving artificial ventilation. They related one specific pattern of breathing, occurring during artificial ventilation, with the development of pneumothorax.² This pattern they describe as 'Active expiratory effort against each ventilator inflation'.¹

During the past 15 months we have developed a system that allows us to optimise the ventilatory support given to our sickest preterm infants. To do this we have been recording ventilator inflation pressure, oesophageal pressure, tidal volume, and transcutaneous oxygen tension (TcPo₂) and carbon dioxide tension (TcPCO₂) during a series of changes in ventilator settings. In the course of our studies we have become aware of the deleterious effect that the infants' own respiratory efforts can have on the applied ventilation. We also observed that the infants' own pattern of respiration could often be modified by changing the ventilator settings. We decided to determine how often active expiration against the ventilator occurred and how often we could abolish this pattern by changes in ventilator settings.

Classification

Greenough *et al*¹ described five patterns of interaction:

- (1) Apnoea.
- (2) Synchrony—spontaneous respiration synchronised with ventilator inflations.

- (3) Hering-Breuer—each ventilator inflation induces a temporary cessation of the infant's respiratory efforts.
- (4) Active expiration against the ventilator.
- (5) Augmented respiration—this was seen only at rates below 15 breaths per minute and therefore need not be considered here.

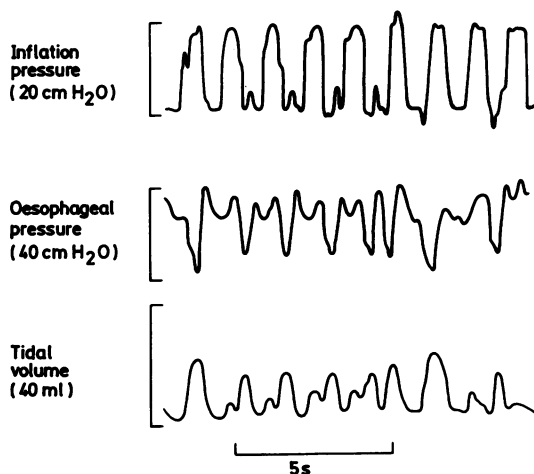


Figure Inflation pressure, oesophageal pressure, and tidal volume trace from an infant showing a mixed pattern of interaction between spontaneous breaths and ventilator inflations. It can be seen that the infant's own respiratory efforts are occurring at various phases of the ventilator cycle.

We wish to add a sixth category. In this the infant produces a mixed pattern of interaction with spontaneous breaths occurring throughout the ventilator cycle (Figure).

Subjects and methods

Subjects. Forty seven infants with a clinically based diagnosis of the idiopathic respiratory distress syndrome, were studied on 51 occasions during the first week of their illness. The mean birthweight was 1.47 kg, and the range 0.68 to 2.73 kg. The mean gestation was 30.4 weeks, and the range 24 to 40 weeks. Mean age at the time of study was 2.2 days. All babies were receiving intermittent positive pressure ventilation (IPPV) with ventilator rates of between 20 and 100 breaths per minute at the time of the study. Draeger Babylog ventilators (time cycled pressure limited) were used exclusively during these studies.

None of the babies had a pneumothorax at the time of study, and each was studied in his incubator.

Apparatus. Measurements were performed as follows:

(1) Inflation pressure—a wide bore needle was inserted into the infant's endotracheal tube at the point where it entered the mouth and connected to an SE Labs 4.86 pressure transducer. Calibration was performed using a water manometer. Frequency response, measured by bursting balloons in a sealed bottle to which the pressure transducer was connected, was satisfactory with a 63% rise time of 6.0 msec.

(2) Oesophageal pressure—an oesophageal balloon was passed using standard techniques and connected to a pressure transducer calibrated as above. Signals obtained were used only to obtain a qualitative estimate of the infant's respiratory efforts.

(3) Tidal volume—a Fleisch type 0 pneumotachograph was connected between the patient manifold of the ventilator circuit and the endotracheal tube. A constant bias flow through the pneumotachograph was achieved by connecting a suction pump (Airshields Diapump) via a 21 gauge needle between the distal portion of the pneumotachograph and the infant's endotracheal tube. As the pressure gradient across the needle was in the order of 900 cm H₂O, the bias flow was not affected by the ventilator cycling pressure. Provided that the ventilator circuit was compensated for the loss of the bias flow, ventilator performance was not affected. The flow signal was converted to a volume signal by electronic integration against time. Calibration was performed by injecting and withdrawing known volumes of air.

The pneumotachograph was found to give a linear response to flows up to 30 l per minute. The 63% time of the pneumotachograph, tubes, and differential pressure transducer was 10.3 msec.

(4) Blood gases—blood gas trends were recorded with a Radiometer TcPO₂ and TcPCO₂ apparatus. These were used over the period of the study to provide a measure of change; however, values were always checked against an arterial blood gas sample taken during the course of the study.

All signals were recorded on paper (Devices recorder) and retained on tape (RACAL tape recorder). Visual display was also available on an SE Labs oscillograph.

Technique. All babies were clinically stable at the time of study and on ventilator settings determined by the medical staff, who were guided by blood gas values. A period of 15 minutes was allowed for stabilisation.

Our initial measurements were made on the babies original settings (that is, those chosen by the clinical staff) during a 14 minute study period. Transcutaneous PO₂ and PCO₂ values were recorded at two minute intervals while the predominant pattern of interaction was defined using the classification above. At the end of 14 minutes the first of a series of ventilator changes were made. Each change comprised an alteration of the rate, or inspiratory:expiratory (I:E) ratio, while peak inspiratory pressure and positive end expiratory pressure remained constant. An attempt was made to study each baby at fast (60 or more breaths per minute) and slow (30 or less breaths per minute) rates, with data on physiological and reversed I:E ratios at each rate. Thus, we aimed to study each individual baby at I:E ratios of 2:1, 1:1, and 1:2—rates of approximately 30 and 100 breaths per minute: six settings. Each change was followed by a 14 minute period of stabilisation.

If any ventilator change was accompanied by an appreciable deterioration, measurements at that particular setting were abandoned. In addition, the need to perform routine care for the baby often restricted the number of ventilator changes that could be performed on each infant.

Results

Data were collected on 230 settings, with at least four readings on each baby. The mean (range) for the initial ventilator setting over the 51 studies, that is that chosen as optimal by the clinical staff, were: peak inspiratory pressure 19 cm H₂O (12 to 28); positive end expiratory pressure 1.4 cm H₂O (0 to 4); inspiratory time 0.6 seconds (0.3 to 1.5);

expiratory time 0.7 seconds (0.2 to 2.8); ventilatory rate 56.4 breaths per minute (20 to 100); inspired oxygen 63.2% (21 to 100). Mean and range of TcPo₂ and TcPco₂ on these settings were 58 mmHg (33 to 103) and 50.1 mmHg (29 to 101), respectively. Throughout our studies only two infants were recorded as having a blood pH of 7.2. The frequencies of the various patterns of interaction occurring on these settings are given in Table 1.

On each occasion, the predominant pattern of interaction was recorded. Active expiration against the ventilator was documented, however, if we observed this pattern over three consecutive breaths or during five inflations in any 30 second period. The baby's initial pattern of interaction was recorded as active expiration against the ventilator on eight occasions. The mean (range) of the initial ventilator settings for these infants were: peak pressure 19.6 cm H₂O (12 to 28); positive end expiratory pressure 1.1 cm H₂O (0 to 3); inspiratory time 0.6 seconds (0.4 to 1.0); expiratory time 0.55 seconds (3 to 1.0); ventilatory rate 63 breaths per minute (45 to 100); inspired oxygen concentration 78% (42 to 100), TcPo₂ 72 mmHg (65 to 103), TcPco₂ 57 mmHg (33 to 101).

Active expiration against the ventilator was prevented by changing the settings in six of these eight babies. This was achieved by increasing the ventilator rate in four studies and by changing from a reverse to a physiological I:E ratio (1:1 or less) in a further two studies. Details are given in Table 2. It can be seen that these changes caused transcutaneous gas readings to remain stable or to improve despite the reduction in mean airway pressure that occurred in those studies where a physiological I:E ratio was used. The remaining two babies who showed active expiration against the ventilator on their initial settings continued to show this pattern through all combinations of ventilator settings. In these, the pattern occurred for at least 25% of the study period. Neither baby developed a pneumothorax.

On eight other occasions the baby's original pattern of interaction was altered to that of active expiration against the ventilator during the series of ventilator changes. On five occasions this occurred as a result of reducing the ventilator rate and on three occasions after the use of a reversed I:E ratio.

Details of these eight events are given in Table 3.

The TcPo₂ and Pco₂ values given in Tables 2 and 3 for infants displaying active expiration against the ventilator indicate that blood gas values were well maintained. During active expiration against the ventilator, however, and when there was a mixed pattern of interaction, TcPo₂ values showed noticeable fluctuation both above and below the baseline, which on occasions did not become evident for sometime during observation.

Two infants included in the study subsequently developed a pneumothorax: neither were making respiratory efforts when studied. A further baby actually suffered a pneumothorax during a period of monitoring when making synchronised respiratory efforts.

Discussion

Our results show that in most babies active expiration against the ventilator can be prevented by changing the ventilator setting, either by the use of faster ventilator rates (60 breaths per minute) or more normal I:E ratios (1:1 or less). This may explain the findings in a previous controlled trial which showed a decreased risk of air leak associated with the use of a ventilator rate of 60 breaths per minute compared with 30 breaths per minute.³ Active expiration against the ventilator is an extremely disruptive pattern of interaction, and is certainly a marker of poor ventilator adjustment. In our studies, it did not seem to be a consistent pattern, and was usually accompanied by episodes in which the baby and the ventilator intermittently synchronised to produce large inflations in a chaotic fashion. It seems likely that these excessive and rapid fluctuations in lung volume are more dangerous than consistent active expiration against the ventilator, where tidal exchange is reduced and the transpulmonary pressure change is small. Similar fluctuations, but of smaller magnitude, were seen during the mixed pattern of interaction.

In our study, we made no attempts to study every baby who developed respiratory distress within the first few hours of birth. Our aim was merely to investigate the possibility of controlling babies' patterns of interaction with the ventilator. In this work we have used Draeger Babylog ventilators

Table 1 *Frequency of observed patterns of interaction on original ventilator settings between ventilator inflations and spontaneous respiration*

Pattern	Synchrony	Apnoea	Hering-Breuer	Active expiration against the ventilator	Mixed	Total
Number	30	4	4	8	5	51

Table 2 Ventilator parameters and transcutaneous gas values for infants showing active expiration against the ventilator on their original settings and after manipulation of the ventilator to produce an acceptable pattern of interaction

Pattern of interaction	No	Peak pressure (Cm H ₂ O)	PEEP (cm H ₂ O)	Inspiration time (secs)	Expiration time (secs)	Ventilator (rate/min)	Inspired O ₂ concentration (%)	TcPo ₂ (mmHg)	TcPco ₂ (mmHg)
(a) *Active expiration against the ventilator Synchrony (4)	4	17.8	1	0.6	0.6	50	66	75.8	53.3
(b) *Active expiration against the ventilator Synchrony (2)	2	15	2	0.7	0.6	46	60	66	72
		15	2	0.4	0.9	46	60	67	67

* (a) Studies in which active expiration against the ventilator was abolished by an increase in ventilator rate.

(b) Studies in which active expiration against the ventilator was abolished by the use of a normal I:E ratio.

PEEP=positive end expiratory pressure.

Conversion: traditional units to SI—1 mmHg=0.076 kPa.

Table 3 Ventilator parameters and transcutaneous gas values associated with the baby's original pattern of interaction with the ventilator, and subsequently when the pattern of interaction has been altered to one of active expiration against the ventilator

Pattern of interaction	No	Peak pressure (Cm H ₂ O)	PEEP (cm H ₂ O)	Inspiration time (secs)	Expiration time (secs)	Ventilator (rate/min)	Inspired O ₂ concentration (%)	TcPo ₂ (mmHg)	TcPco ₂ (mmHg)
(a) *Original pattern—synchrony (4) mixed (1) Active expiration against the ventilator	5	17.6	2	0.4	0.44	80	72	57	53
		17.6	2	0.8	0.9	36	72	60	56
(b) Original pattern of interaction—synchrony (2) apnoea (1) Active expiration against the ventilator	3	16	2	0.6	0.6	52	61	56	53
		16	2	0.75	0.45	52	61	57	50

* (a) Studies in which active expiration against the ventilator was produced by a reduction of ventilator rate.

(b) Studies in which active expiration against the ventilator was produced by a reversed I:E ratio.

PEEP=positive end expiratory pressure.

Conversion: traditional units to SI—1 mmHg=0.076 kPa.

(time cycled, pressure limited) throughout. We have shown previously that over the range of ventilator rates used in the study (20 to 100 breaths per minute) that this machine is capable of working satisfactorily without appreciable alteration to the waveform or performance.^{4 5}

We feel that Greenough *et al* have identified an important marker for poor ventilator adjustment. The risks attached to this pattern of interaction have yet to be quantified. We also do not know whether the importance of the patterns is influenced by maturity, postnatal age, or the extent of lung pathology, but consider that the pattern can be modified by alteration in ventilator settings, possibly avoiding the need for paralysis.

References

- ¹ Greenough A, Morley C, Davis J. Interaction of spontaneous respiration with artificial ventilation in preterm babies. *J Pediatr* 1983;**103**:769-73.
- ² Greenough A, Morley CJ, Davis JA, Wood S. Pancuronium prevents pneumothoraces in ventilated premature babies who actively expire against positive pressure ventilation. *Lancet* 1984;**i**:1-3.
- ³ Heicher DA, Kirshching DS, Harrod JR. Prospective clinical comparison of two methods for mechanical ventilation of neonates: rapid rate and short inspiratory time versus slow rate and long inspiratory time. *J Pediatr* 1981;**98**:957-61.
- ⁴ Field DJ, Milner AD, Hopkin IE. The effect of inspiratory time on tidal volume when using intermittent positive pressure ventilation and high frequency positive pressure ventilation. *Arch Dis Child* 1985;**60**:259-61.
- ⁵ Field DJ, Milner AD, Hopkin IE. Calculation of mean airways pressure during neonatal intermittent positive pressure ventilation (IPPV) and high frequency positive pressure ventilation (HFPPV). *Pediatric Pulmonology* 1985;**1**:141-4.

Correspondence to Professor A D Milner, Department of Neonatal Medicine, City Hospital, Nottingham NG5 1PB.

Received 16 June 1985