Oesophageal pressure measurements in ventilated preterm babies

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SUMMARY Oesophageal pressure measured with an air-filled, thin latex balloon on a 6 French gauge catheter can accurately measure intraoesophageal pressures in ventilated preterm babies. Intraoesophageal pressures and intrapleural pressures are equivalent. Less than 10% of the applied positive ventilator pressure is transmitted to the oesophageal balloon and the intrapleural catheter (intercostal drain). The oesophageal traces show that ventilated babies breathe independently of the ventilator. The largest deflections recorded from the oesophageal trace are from spontaneous inspiratory activity. During paralysis with pancuronium spontaneous respiration is inhibited, peristaltic waves are still recorded, and there is little transmitted pressure from the ventilator to the oesophagus or intercostal drain.

Oesophageal pressure has been shown to reflect intrapleural pressure in spontaneously-breathing adults^{1,2} and babies.^{3,4} Because of its accessibility the oesophagus has become the conventional place in clinical studies from which to assess intrathoracic pressure changes for determining dynamic lung compliance and thoracic gas volume.⁵ During normal breathing with compliant lungs, the intraoesophageal pressure is slightly negative owing to the inherent tone in the diaphragm and intercostal muscles. With active inspiration large negative pressures are produced. Expiration is generally passive with the pressures returning to their baseline level; occasionally positive expiratory pressures can be produced by an active expiratory effort.

About 1½% of all newborn babies require ventilation in the first few days of life. There has been little scientific measurement of respiratory function in these babies because they are ill and very difficult to study or handle without adverse effects.⁶ In consequence the methods used for ventilation are based on skill, experience, and a knowledge of animal physiology, or on the study of older fitter babies. This, of course, may not be the same in ill preterm babies. Without an adequate knowledge of the pathophysiology of these babies the development of improved methods of treatment will lack a firm foundation.

This study concentrated on the oesophageal pressure measurements found in very preterm babies with respiratory distress syndrome who needed respiratory support with positive pressure ventilation.

In some cases the oesophageal pressures were compared with intrapleural pressures from intercostal catheters used for draining pneumothoraces.

Methods

Thirty preterm babies were studied during the first 2 weeks of life. Their gestational ages ranged from 24 to 34 weeks. All of them were being treated with positive pressure ventilation either for respiratory distress syndrome or its complications. The ventilation was delivered by a time-cycled pressure limited Bourne Ventilator No 201 with a gas flow of 5 litres a minute. Uncuffed 3.0 mm nasotracheal tubes were used in all the babies. The ventilating pressure was measured by a side arm on the nasotracheal tube close to the baby with a Mercury M10 transducer range ± 100 cm water.

Oesophageal pressures were measured using a thin latex (0.05–0.075 mm) air-filled balloon 30×6 mm which was firmly attached to the end of a 6 French gauge feeding tube. This was directly attached to a pressure transducer Mercury M10 range ± 100 mm of water. It was placed through the mouth into the stomach, open to air, and withdrawn to lie in the lower oesophagus at a position 1 cm beyond the spot where the baby's own inspiratory effort produced a negative pressure. This is similar to the technique of Milner *et al.*⁵ In use the balloon contained 0.1–0.2 mm air—that is, almost deflated, as in this condition the oesophageal balloon has been shown to reflect most closely local absolute pleural pressures.⁷ When

a diagnostic chest x-ray film was performed on the baby the balloon was left in place to confirm its position. Whenever possible the oesophageal pressure (Poe) was checked against the mouth/tracheal pressure (Pm) by briefly occluding the nasotracheal tube (airways) during spontaneous breathing.⁸ The Poe/Pm was generally between 95 and 102%. However on some occasions there was such a leak round the nasotracheal tube, shown by comparing inspiratory and expiratory volumes during artificial ventilation, that complete airways occlusion was not possible.

All measurements were performed with the baby supine in his own incubator in the intensive care area.

Gas flow in and out of the nasotracheal tube was measured with a Mercury F2L pneumotachograph inserted between the end of the nasotracheal tube and the ventilator connections. It has a volume of 1.5 ml, internal diameter of 3 mm, and a pressure drop of 0.1 mm of water/litre per second. Condensation was not a problem because the apparatus was used inside a warm incubator. If there was a leak from the nasotracheal tube the expiratory volume was used as an estimate of the volume of gas entering the chest. The flow signal was measured by a Mercury M11 differential pressure transducer and integrated to volume by a Gould integrator (model 13-4615-70). All the signals were recorded on a Gould rectilinear holograph.

Results

Fig. 1 shows recordings made on a baby of 29 weeks' gestation at age 24 hours, ventilated for the treatment of respiratory distress syndrome. It illustrates that during artificial ventilation the baby continues to breath spontaneously and the oesophageal pressure recordings show the normal negative pressures produced by spontaneous inspiration. It also shows

Fig. 2 Pleural pressure (upper trace), recorded from right to left, with negative pressures downwards. Below this is ventilator pressure, positive deflections upwards, their oesophageal pressure negative deflections downwards. The lower trace is the integrated volume signal. Points 'A' represent periods of oesophageal peristalsis. Points 'B' represent augmented inspirations as shown by increased negative deflections in the oesophageal and pleural pressure traces during ventilator inflation.

that despite positive pressure ventilation of up to 20 cmH₂O being applied to the trachea very little of

Fig. 1 Ventilator pressure (upper trace), recorded from right to left, oesophageal pressure (middle trace) with negative pressures downwards and inspiratory and expiratory volumes (lower trace). (The integrator resets to zero at zero flow.)

Fig. 3 Oesophageal pressure (upper trace) negative downwards, ventilator pressure (middle trace)—positive deflections downwards, pleural drain pressure lower trace—positive deflections downwards.

this pressure is transmitted to the oesophagus. It was always less than 10% of the applied pressure in all the babies studied. The pressure transmitted was related to the size of the applied pressure. In Fig. 1 the ventilating pressure decreases from right to the centre of the diagram and then increases again towards the left. There is a critical pressure at which the applied pressure is transmitted to the oesophagus and this is about 12.5 cmH₂O. This level is dependent on the lung compliance—that is the stiffer the lungs the less pressure is transmitted to the

oesophagus. This failure of ventilator pressure being transmitted to the oesophagus is not an artefact of the technique of oesophageal pressure measurement, because recordings from intrapleural drains taken simultaneously confirm the accuracy of the oesophageal pressure recording. This is shown in Fig. 2 which is recorded from a 1.9-kg baby made 90 hours after birth with the child on intermittent ventilation. A right-sided pneumothorax had been drained at 48 hours of age. The intrapleural pressures from both the oesophageal recordings and the intercostal drain are very similar. The predominant oesophageal and pleural deflections are negative owing to spontaneous inspirations. For the four ventilated breaths shown there was no evidence of ventilator pressure being transmitted to either the oesophageal or pleural recordings. The positive pressures shown at 'A' were owing to peristalsis. Ventilator inflation provokes an augmented inspiration by the infant shown at 'B'.

Figs 3 and 4 show the effects of paralysing the baby with pancuronium on the oesophageal pressure recording. In Fig. 3 the baby was 48 hours old. He had been paralysed because he was thought by the clinicians to be fighting the ventilator and had a pneumothorax. There was minimal transmission of ventilator pressure to the oesophagus or pleural drain. Fig. 4 is from a 2-hour-old baby who was paralysed for similar reasons. There was little transmission of the ventilator pressure even though the oesophageal balloon system was sensitive enough to record pressures from the cardiac impulse of about 1 mm/H₂O. In Fig. 5, taken from a baby of 1.765 kg at 36 hours, a similar effect can be seen but this shows that positive pressures from peristalsis in the oesophagus are preserved despite pancuronium, as this muscle relaxant drug does not affect the

Fig. 4 Integrated volume signal (upper trace), oesophageal pressure (middle trace), negative deflections upwards. In the centre of the trace the sensitivity is increased 10-fold (1 mm=0.5 mm/H₂O)—and this shows ventilator pressure is only minimally transmitted to the oesophageal balloon. When the baby is disconnected from the ventilator (extreme right of the trace) the oesophageal balloon system is sufficiently sensitive to record pressure changes caused by the cardiac impulse.

Fig. 5 Oesophageal pressure (lower trace) positive deflections upwards, ventilator pressure (middle trace), positive deflections upwards, pressure recording from intercostal drain, (upper trace) positive deflections upwards. This shows a component of oesophageal peristalsis persists despite the baby being paralysed by pancuronium, which is not affected by ventilator inflation. The change in pressure at 'A' is due to temporary disconnection of the transducer.

smooth muscle component of oesophageal contractions. They are, of course, not reflected in the pleural pressure recording.

Discussion

Oesophageal pressure was shown to be similar to pressure swings recorded from pleural drains by Hustead and Avery,⁴ on a single anencephalic infant and later by Dinwiddie and Russell.³ It was also shown to correlate well with mouth pressure changes at airways occlusion during the measurement of thoracic gas volume by Milner *et al.*⁵ who concluded that it was valid to use an oesophageal balloon system to measure changes in intrathoracic pressures. Milner did however find that changes in oesophageal pressure tended to be smaller than concomitant changes in airways opening pressure by an average of 20%. However in that study, a 5 French gauge catheter was used with an internal diameter of 1 mm and as pointed out by Beardsmore *et al.*⁸ the frequency response at such a catheter is inadequate. Beardsmore *et al.*⁸ used a 6 French gauge catheter (internal diameter 1.4 mm) and were able to validate the use of Poes as a measure of pleural pressure using the occlusion test.

More recently, Beardsmore *et al.*⁹ have shown that there is no significant difference in the pressure changes measured at the different positions in the oesophagus although measurements were more reproducible at the diaphragmatic end. However care was taken in this study to position the balloon in the lower third of the oesophagus. Mead *et al.*¹⁰ showed by comparing oesophageal pressure with the pleural pressure measured from catheters introduced in the intrapleural space that the best correspondence between the two was in the upright position rather than measurements taken lying down.

However in this study we were able to show good correlation between the pressure swings from the two sites even though our babies were studied in the supine position.

In the previous studies quoted the neonates were all breathing spontaneously. However in this study in 4 infants with intercostal drains *in situ* we were able to show that despite artificial ventilation the oesophageal balloon reflected accurately the intrapleural pressure as recorded from the intercostal drain. The correlation is perhaps surprising as one would expect that the higher magnitude pressure changes caused by the ventilator would interfere with the oesophageal balloon. However, the results show that very little of the ventilator pressures are transmitted to the oesophageal balloon either in the spontaneously breathing infant or in the paralysed state. This confirms earlier work by Stark *et al.*¹¹ who studied the effect of pancuronium on lung compliance. Stark explained this phenomenon as being due to the high compliance of the relaxed chest wall hence minimising the effect of lung volume changes on the oesophagus. Stark *et al.* went on to suggest that the chest wall compliance was greater in the less mature infants, such as those included in our study. However chest wall compliance is much higher than lung compliance in babies with respiratory distress syndrome and in both the paralysed and non-paralysed state must represent only minimal resistance to stretch by the ventilator compared with the lungs. This idea is supported by the fact that babies who are ventilated with normally compliant lungs require much lower pressures visibly to inflate the chest than infants with a similar gestational age with stiff lungs owing to respiratory distress syndrome and yet these infants have the same chest wall compliance as they are of the same degree of prematurity. Hence it would seem more probable

that lack of transmission of applied pressure is a function of the very low lung compliance rather than the high chest wall compliance.

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