Commentaries

C MORLEY AND M SOUTH

Department of Paediatrics, University of Cambridge

Ramsden and Reynolds highlight the dilemma faced by all those who ventilate ill babies. They suggest from some of the publications, theoretical calculations, and their experience that long inspiratory times and slow rates (30–40/minute) are the best way to ventilate babies with severe respiratory distress syndrome (RDS). Unfortunately, they discount fast rate ventilation (60–100/minute) without producing satisfactory evidence to show it is harmful.

The studies of Reynolds in the early '70s, when ventilation was relatively new, involved babies who were apnoeic, were unresponsive to hand ventilation, and required high oxygen concentrations. Small numbers were studied; six in one paper¹ and nine in another.² They were not randomised and no account was taken of changes with time. Since then, ventilators have improved, the babies cared for are smaller, and the indications for ventilation less extreme.

The authors cite and dismiss nine papers that reported improvements with fast ventilator rates. Some of these had large numbers (Spahr 69, Pohlandt 96, and Heicher 102), and were randomised. They are criticised by Ramsden and Reynolds because 'no account was taken of the diagnoses'. Despite varied causes for respiratory failure, these trials showed that fast rate ventilation resulted in less pneumothoraces, better oxygenation, and lower peak pressures.

The main thrust of Ramsden and Reynolds' argument against fast rates is that they may result in gas trapping, carbon dioxide retention, pneumothoraces, and compromised circulation. They calculate time constants for neonatal lungs based on passive lung mechanics. Unfortunately, this ignores the fact that most ventilated babies breathe spontaneously,³ often expiring actively.⁴ This effectively shortens the time constants and reduces the likelihood of excessive gas trapping. They present 'rough calculations', which indeed are 'sobering' if babies with healthy lungs were ventilated at 100 breaths per minute. If the compliance and resistance given for babies with RDS are used in these calculations, however, expiratory times could be as short as 0.15 seconds. Complete expiration would then occur at a rate of 100/minute without gas trapping.

The major abnormality in RDS is atelectasis and low lung volume. The more severe the lung disease, the faster the babies breathe and the shorter their

expiratory time.³ In this situation spontaneous fast respiratory rates predispose to gas trapping, thereby maintaining lung volume during expiration. There is no evidence that excess gas trapping occurs in spontaneously breathing babies with RDS.

Most babies breathe while being ventilated.⁵ At ventilator rates of 30–40 cycles per minute this is usually asynchronous with the ventilator and may increase the risk of pneumothorax or intraventricular haemorrhage. At faster rates babies spend more time breathing synchronously with the ventilator. Many do so continuously except when disturbed.⁶

Ramsden and Reynolds claim to base their ventilation technique on the time constants of their patients but describe no measurements of these. Measurements in ventilated premature babies are difficult and not practical for clinical use. They conclude that in general they follow the guidelines described in 1979 (slow rates with prolonged inspiratory:expiratory ratios).⁷ They state, however, that the use of slow rates and long inspiratory times in conditions other than severe RDS 'is certain to cause severe hyperinflation with disastrous results'. As babies with severe RDS are the minority presumably they would agree that this ventilation technique is unsuitable for most ventilated babies.

Unlike the authors, we are unable to make an early diagnosis of the cause of the respiratory failure. Neither can we detect complete expiration visually. Assessment of the publications and our studies of neonatal ventilation leads us to believe that most babies with RDS are best ventilated at fast rates. We now tailor the ventilation to the severity of the baby's illness by monitoring continuously the spontaneous respiratory timing, using this as a basis for the ventilator inspiratory and expiratory times. Thus as the lung disease improves spontaneous respiratory rate falls and the ventilator rate is reduced, eliminating excessive gas trapping. In our experience rapid rate ventilation often allows lower ventilatory pressures and does not cause overinflation or carbon dioxide retention and the incidence of pneumothorax is low. Babies breathing at the same rate as the ventilator are more settled.

Much more clinical research is required to elucidate the appropriate ventilator settings for use in neonatal respiratory failure.

References

- ¹ Reynolds EOR. Effect of alterations in mechanical ventilator settings on pulmonary gas exchange in hyaline membrane disease. *Arch Dis Child* 1971;**46**:152–9.
- ² Herman S, Reynolds EOR. Methods of improving oxygenation in infants mechanically ventilated for severe hyaline membrane disease. *Arch Dis Child* 1973;**48**:612–7.
- ³ South M, Morley CJ. Spontaneous respiratory timing in intubated neonates with RDS. *Early Hum Dev* 1986;**14**:147–8.

⁴ South M, Morley CJ, Hughes G. Expiratory muscle activity in premature babies. *Early Hum Dev* 1987. (In press.)

⁵ Greenough A, Morley CJ, Davis JA. Interaction of spontaneous respiration with artificial ventilation in preterm babies. *Pediatrics* 1983;**103**:769–73.

⁶ South M, Morley CJ. Spontaneous mechanical ventilation of the neonate. *Arch Dis Child* 1986;**61**:1190–5.

⁷ Reynolds EOR. Ventilator therapy. In: Thibeault DW, Gregory GA, eds. *Neonatal pulmonary care*. Menlo Park: Addison-Wesley, 1979:217–36.

A D MILNER

Department of Child Health, University Hospital, Nottingham

The review by Ramsden and Reynolds must be welcomed as it provides a thoughtful and, on the whole, balanced assessment of our current knowledge on how best to ventilate newborn infants. As our knowledge is incomplete, however, it is possible to examine the results of data available in a somewhat different light and to adopt a different, although I consider equally defensible, position.

The authors are rightly critical of the trials that have been carried out. This is inevitable as there are so many variables, so that it is extremely difficult to obtain data that are totally clear cut. They do not emphasise, however, that the two studies on which they base their present position are equally open to criticism. Their initial and now classic studies¹ purely explored the effects of different ventilator settings on blood gases and found that with the ventilators then available and the relatively mature babies in the neonatal units (all had birth weights over 1000 g) oxygenation improved at relatively low respiratory rates and long inspiratory:expiratory (IE) ratios. Recent studies, using similar experimental design but with better ventilators and more immature babies, have found that oxygenation and carbon dioxide excretion can be better at rates as high as 100 to 120/minute, although there is little evidence to suggest that rates above 60/minute are beneficial unless the baby is making respiratory efforts that are out of phase with the ventilator.^{2,3} The second study, from Professor Reynolds' unit, showing that slow ventilator rates led to improved mortality, used historical controls.⁴ I know that the authors would be the first to admit that this is far from ideal.

The authors place considerable emphasis on the need to allow sufficient time for expiration (and inspiration) to occur to avoid inevitable (alveolar) positive and expiratory pressure (PEEP) and allow maximal tidal exchange to occur for a given inflation pressure. Their claim that the time allowed for a volume change to occur must exceed three times the time constant is open to challenge. If, for example, only twice the time constant is allowed the calculated fall in tidal volume (13%) will be more than compensated for by the increased respiratory rate,

so that alveolar ventilation will rise rather than fall. It is also extremely unlikely that a PEEP of 13% of the inflation pressure will be 'fraught with danger'. The authors correctly point out that high rates may lead to problems if used in babies with normal lungs and, even more, in those with airways obstruction. High rate ventilation is not being recommended, however, for babies other than those with severe idiopathic respiratory distress syndrome (IRDS) who have short time constants, the same provisos that the authors make on the use of long inspiratory times. Although the studies published do suffer from design faults,^{5–7} it is interesting to note that all found a reduction in the incidence of pneumothorax with high rates and not the reverse as predicted by the time constant model of Ramsden and Reynolds.

In conclusion, we consider that the evidence available supports our current clinical practice. We use initial ventilator settings of 60/minute with an IE ratio of 1:1 and inflation pressures of 18–20/0–3 for babies with severe IRDS. If the baby fails to settle on these settings and continues to make respiratory effort against the ventilator we increase the rate to 100–120 with IE ratios of 1:1.2.⁸ If this manoeuvre does not lead to an improvement in oxygenation, a relatively rare event, we paralyse the baby with pancuronium and revert to rates of 60/minute. The rates are reduced as soon as the clinical condition of the baby permits.

References

- ¹ Reynolds EOR. Effect of alterations in mechanical ventilator settings on pulmonary gas exchange in hyaline membrane disease. *Arch Dis Child* 1971;**46**:152–9.
- ² Field D, Milner AD, Hopkin IE. High and conventional rates of positive pressure ventilation. *Arch Dis Child* 1984;**59**:1151–4.
- ³ Greenough A, Morley CJ, Pool J. Are fast rates an effective alternative to paralysis? *Pediatr Res* 1986;**19**:1077.
- ⁴ Reynolds EOR, Taghizadeh A. Improved prognosis of infants mechanically ventilated for hyaline membrane disease. *Arch Dis Child* 1974;**49**:505–15.
- ⁵ Bland RD, Kim MH, Light MJ, Woodson JL. High frequency mechanical ventilation in severe hyaline membrane disease. *Crit Care Med* 1980;**5**:275–80.
- ⁶ Heicher DA, Kasting DS, Harrod JR. Prospective clinical comparison of two methods for mechanical ventilation of neonates: rapid rate and short inspiratory times versus slow rate and long inspiratory time. *J Pediatr* 1981;**98**:956–61.
- ⁷ Pohlandt F, Bernsau V, Feilen K-D, et al. Reduction of barotrauma in ventilated neonates by increase in ventilation frequency—first results of a prospective collaborative and randomized trial of two different ventilatory techniques. *Pediatr Res* 1986;**19**:1077.
- ⁸ Greenough A, Milner AD. High frequency ventilation in the neonatal period. *Eur J Pediatr* 1987. (In press.)