Response

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'There are more ways of killing a cat than choking her with cream'[1]

We are delighted that our article has prompted such lively discussion and glad to have the opportunity to respond to some of the comments made by Professor Milner and Drs Morley and South. We certainly do not discount fast rate ventilation, as stated by the latter authors; rather, we wish to present arguments about the relation between ventilator settings and lung mechanics that bear heavily on the selection of appropriate settings in different illnesses.

We welcome support from both commentaries for our own reservations about the present day use of techniques derived from studies performed many years ago,[2 3] and we certainly agree that these studies are open to criticism. We do not, however, understand Drs Morley and South's strictures about randomisation and changes with time. The questions that were addressed were to do with exploring the alterations in blood gases and other variables that took place after changes in ventilator settings in babies selected for study because of severe hyaline membrane disease (HMD). There would be no purpose in randomising the babies: the alterations in settings *were*, in fact, randomised, within the hypotheses being tested, and observations were made after a standard time. It is true, of course, that the use of historical controls in another study[4] was not ideal, as argued by Professor Milner. The reasons for adopting this approach were given at the time and also debated at length subsequently.[5] The data were, however, collected prospectively and the pathological studies performed 'blind'.

We think Professor Milner would concede that historical controls are better than no controls at all—a feature of some of the papers that Drs Morley and South believe we unfairly dismissed. In three of these papers,[6–8] Drs Morley and South draw special attention to the enrolment of large numbers of babies and to randomisation, but they seem to discount our concerns about the importance of diagnosis. We strongly challenge this stance, as argued in our article.

We also do not accept Professor Milner's assertion that the results of the recent trials are the reverse of those predicted from a consideration of time constants. These trials used inappropriately long inspiratory times in infants who did not necessarily have bad HMD—a measure that can be expected to have caused severe hyperinflation. We reiterate that lumping all conditions together may cause any beneficial effects in one illness to be completely submerged by adverse effects in another. The results of trials of this sort can tell us only that if we were to ignore the diagnosis and ventilate all infants in the same way then fast rates may cause less pneumothoraces than slow rates. Where the diagnosis is not known (a problem that Drs Morley and South consider to be perhaps more common than we do) this information could be of value, but it is not really what we want to know when deciding how best to ventilate an infant who clearly has, say, severe HMD, meconium aspiration, or apnoea with normal lungs.

We agree with Drs Morley and South that gas trapping is unlikely at fast rates in severe HMD and also with Professor Milner's view that inadvertent positive end expiratory pressure (PEEP) amounting to 13% of peak airway pressure is unlikely to be harmful, but we think there is little evidence to support his statement that fast rates are being recommended only for infants with severe HMD. And even these infants, ventilated according to Professor Milner's protocol at a rate of 120/minute, which would yield an expiratory time of 0·27 seconds, would be vulnerable to excessive inadvertent PEEP if recovery began or secretions accumulated in the endotracheal tube.[9] Only one τ_{RS} might then be available for expiration and 30% or more of the peak pressure would remain as inadvertent PEEP.

We agree with both commentaries that the interaction between spontaneous and ventilator breaths is an important issue. We are puzzled, though, by Drs Morley and South's suggestion that this effectively shortens time constants. Synchronous respiratory efforts may hasten the rate of change of lung volume, but efforts that are out of phase will have the opposite effect. Undoubtedly, if synchrony can be achieved by fast rates then this is an important benefit. We have discussed the paucity of data on this point, and we note that Drs Morley and South refer to their own recent paper[10] on this matter (published when our article was in press): this paper provides data only on a single infant, though a total of 20 are said to have shown similar results.

We of course heartily welcome Drs Morley and South's closing remarks. We do not think that the available information strongly favours any particular regimen. Our own regimen[11] does not, incidentally, involve only the use of slow rates and long inspirations, as inferred by Drs Morley and South. We

come back to our view that techniques of ventilation and clinical trials should be sensitive to what is known about lung pathophysiology and lung mechanics and should not be undertaken as though the lung in normal infants and in infants with all sorts of different illnesses may be expected to behave in the same way.

References

[1] Kingsley C. *Westward ho!*

[2] Reynolds EOR. Effect of alterations in mechanical ventilator settings on pulmonary gas exchange in hyaline membrane disease. *Arch Dis Child* 1971;**46**:152–9.

[3] Herman S, Reynolds EOR. Methods for improving oxygenation in infants mechanically ventilated for severe hyaline membrane disease. *Arch Dis Child* 1973;**48**:612–7.

[4] Reynolds EOR, Taghizadeh A. Improved prognosis of infants mechanically ventilated for hyaline membrane disease. *Arch Dis Child* 1974;**49**:505–15.

[5] Reynolds EOR. General discussion. In: *Major mental handicap: methods and costs of prevention*. Amsterdam: Elsevier/Excerpta Medica/North-Holland, 1978: 135–7. (Ciba Foundation symposium, No 59.)

[6] Bland RD, Kim MH, Light MJ, Woodson JL. High frequency mechanical ventilation in severe hyaline membrane disease. *Crit Care Med* 1980;**5**:275–80.

[7] Spahr RC, Klein AM, Brown DR, MacDonald MH, Holzman IR. Hyaline membrane disease: a controlled study of inspiratory to expiratory ratio in its management by ventilator. *Am J Dis Child* 1980;**134**:373–6.

[8] 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**:957–61.

[9] Prendiville A, Thomson A, Silverman M. Effect of tracheobronchial suction on respiratory resistance in intubated preterm babies. *Arch Dis Child* 1986;**61**:1178–83.

[10] South M, Morley CJ. Synchronous mechanical ventilation of the neonate. *Arch Dis Child* 1986;**61**:1190–5.

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