Correspondence

Ventilator settings and active expiration

Sir,

We read with interest the paper by Field et al reporting an attempt to manipulate ventilator settings to prevent active expiration against positive pressure inflation.¹ We have previously shown how disastrous such an interaction can be, and as a consequence feel that any manoeuvre designed to try and lessen the incidence of this condition must be applauded.² For such techniques to be successful, however, an increased understanding of the aetiology of this condition is necessary. Unfortunately, the variety of ventilator settings of the eight infants actively expiring in the study by Field et al (peak pressures 12–28 cm H₂O, positive end expiratory pressure 0–3 cm H₂O, inspiratory time 0–1/10 second, ventilator rate 45–100 per minute) make it difficult to draw conclusions regarding their individual roles in provoking this reflex.

Our original description of active expiration was at ‘conventional’ frequencies of 30–40/min with an inspiratory to expiratory (I:E) ratio of 1:1 (in the presence of blood gases within the desired range), and we noted in that study that at higher frequencies infants tended to breath in synchrony with their ventilators or be apnoeic.³ In the study of Field et al higher frequencies also were successful in preventing active expiration in four of the eight infants included in their study.¹ We feel we should stress, however, that it is important not to draw conclusions that fast rates always result in reducing the incidence of active expiration. We have recently shown in a controlled study, including 17 infants, all actively expiring at conventional frequencies (30–40/min, I:E ratio 1:1), that in only approximately half were the infants’ rates of 60 and 120 breaths/min successful at suppressing active expiration. We also found in that study that at the higher rates (unlike our experience at conventional frequencies) infants change their pattern of interaction and in certain cases reverted back to active expiration after a period of synchronous breathing at the faster rates.⁴ We concluded that fast rates were therefore only successful in certain infants in prevention of active expiration, and even in those infants continuous or frequent recordings would be necessary to ensure that the disastrous reflex—that is, active expiration—and had not occurred, putting the infant again at risk of an air leak.

Field et al’s study indicated that I:E ratios may also be important in the aetiology of active expiration, a physiologically I:E ratio in their study being apparently less detrimental, but in two of their eight cases of active expiration neither a change of rate nor I:E ratio was successful in preventing this reflex.⁵ Artificial ventilation is made up of many features—positive end expiratory pressure, positive inspiratory pressure, I:E ratio, ventilator rate, flow, type of ventilator, etc. To give clearer guidance about a pattern of ventilation that may avoid active expiration, we would suggest that a systematic investigation, which we have undertaken and hope to report in the near future, may be more successful.

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Sir,

We read with interest Field et al’s paper concerning prevention of active expiration against positive pressure inflation by manipulation of ventilator settings.¹ They add a sixth category of interaction to those described² and give an example of this, showing a period of ventilation lasting 12 seconds. Four episodes occur during which spontaneous expiration happens during ventilator inflation. As five or more episodes in 30 seconds is one of their criteria for active expiration, it is not clear that this ‘mixed’ interaction group are separate and at lower risk of pneumothorax. The classification of baby-ventilator interaction requires further study, but we do not feel that the addition of this ‘mixed’ group is contributory.

This study, in a selected group of neonates, compares the effects of an incomplete series of changes between arbitrarily chosen ventilator time settings in a non-randomised sequence.

The paper seems to show that active expiration against the ventilator is less likely with faster ventilator rates, or low inspiratory to expiratory (I:E) ratios, than slower rates, or high I:E ratios. Common to both fast rates and low I:E ratios is a short inspiratory time, and this may be the reason for a reduction in active expiration in this study. In our studies babies with respiratory distress syndrome breathe with inspiratory times of 0.24–0.4 seconds. It is when ventilator inflations last longer than the babies’ own ‘intrinsic’ inspiratory time that active expiration against the ventilator may occur.

In Field et al’s study babies from 24 to 40 weeks’ gestation, from a few hours to 7 days of age, and with respiratory distress syndrome of variable severity were all grouped together. Spontaneous respiratory times vary with a number of factors—that is, gestation, postnatal age,⁶ and severity of respiratory distress syndrome. These times vary as respiratory time constants change with the course of the respiratory illness. To prevent active expiration against the ventilator, time settings may need to be adjusted individually and possibly changed with time.

The paper mentions the use of ‘physiological’ I:E ratios. We do not know what I:E ratios are ‘physiological’ in intubated premature babies with respiratory distress syndrome.
More work on the spontaneous respiratory activity of ventilated babies is needed.

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References


Drs Field, Milner, and Hopkin comment:

We thank Drs Greenough and Morley and Drs South and Morley for their comments. Both letters raise similar questions, and in response we would just like to make three points.

(1) Our aim was to investigate the role of ventilator manipulation in controlling active expiration against the ventilator. We feel that we have shown this to be possible in a proportion of ventilator dependent infants.

(2) To elucidate the situation further will require a prospective study of ventilator dependent infants in whom pressure and volume monitoring is continuous from the time of beginning ventilation. Currently, such a system is not available.

(3) A discussion and clarification of what constitutes ‘active expiration against the ventilator’ would be a useful step in aiding further research in this area.

A service for problem families

Sir,

Dr Polnay’s encouraging account of the Radford Family Centre gives welcome attention to an area central to child health today—namely, how best to help families handicapped by disadvantage. There are analogies with the management of similar problems in developing countries that are worth exploring, in this case with Nutrition Rehabilitation Centres.

Nutrition Rehabilitation Centres were set up in many countries in Africa and elsewhere in the 1960s and 70s with the aim of preventing malnutrition by teaching mothers how to feed their children better. The concept is now being reassessed as a result of evaluations that cast doubt over their effectiveness. Yet the aims of the centres were broadly similar to those formulated for the Nottingham families by Dr Polnay. What went wrong?

Firstly, the concept that malnutrition is due mainly to ignorance has been falsified: usually, adverse socioeconomic circumstances are the prevailing aetiological factor. Secondly, though the centres were successful in rehabilitating children with malnutrition in the short term, their impact on long term problems was limited: eight months after admission the children’s nutritional state and family’s dietary practice were little different from a control group. It was considered that the families had ‘practical difficulties in implementation, chiefly related to low income’. These findings were confirmed in Lukman’s study.

As a result of such evaluation nutrition policies in developing countries are changing to emphasise community based programmes that train community workers who will help the local people (not just the parents of malnourished children, who have low social status) to confront the root causes of malnutrition. Though the analogy with family centres should not be pursued too closely, the problems the families face are similar: poverty, lack of community support, and a feeling of ‘powerlessness’. As with malnutrition, the children admitted represent the tip of an iceberg—there are many more less severely affected outside.

In finding remedies, ultimately, the professionals must stand aside and assist in the development of community based health initiatives involving local people as activators, to combat the root causes of ‘problem families’.

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Dr Polnay comments:

I would accept that there are three problems, namely, poverty, ignorance, and neglect, rather than the two, ignorance and neglect. Whether these factors lead to problems in the children depends on the balance that exists between the individual vulnerability of the family, the quality of the professional services that are available, and the hostility that they meet from their environment. This is illustrated in the attached diagram. In the Family Centre we have worked very much in the areas of personal vulnerability. However, other social, medical, and educational initiatives in the area have also addressed themselves to the other two issues, namely the stresses that the environment produces and the services available outside to support the families.

Long term follow up studies of early intervention programmes are few and far between. However, Guteilus et al and Schweinhart et al have produced promising long term results.

Viewed in its wider context, our record for dealing with disadvantaged communities may not be quite as impressive. References to the Medical Officer of Health for the Report...