1200 Correspondence

'rejection' pressure of 35 cm H₂O is achieved with an expiratory flow rate of 1.3 l/minute. Firstly, how can airway pressure rise to such a level at such a low flow rate unless the expiratory resistance of the circuit and blow-off volume are very high? Secondly, how did the baby manage to breathe out with an intrathoracic pressure of about +15 cm H₂O against an inflation pressure of +35 cm H₂O? The answer may be that the oesophageal pressure measurements in young infants by this technique are so unreliable³ that their use even for timing the babies' respiratory efforts are highly questionable.

References

- Hoskyns EW, Milner AD, Boon AW, Vyas H, Hopkin IE. Endotracheal resuscitation of preterm infants at birth. *Arch Dis Child* 1987;**62**:663-6.
- Prendiville A, Thomson A, Silverman M. Effects of tracheo-bronchial suction on respiratory resistance in intubated preterm babies. *Arch Dis Child* 1986;**61**:1178-83.
- Beardsmore CS, Wong YC, Stocks J, Silverman M. Assessment of the catheter tip pressure transducer for use in infant lung function tests. *Med Biol Eng Comput* 1982;**20**:657-60.

M SILVERMAN
Paediatric Department,
Royal Postgraduate Medical School,
London W12 0HS

Dr Hoskyns and Professor Milner comment:

We are grateful for Dr Silverman's interest in our paper. The flow resistance of the resuscitation equipment was 16 cm H₂O/l/second and 7 cm H₂O/l/second with endotracheal tubes of internal diameter 3.5 and 2.5 mm, respectively. These measurements were obtained using the inspiratory flow rates used for the studies — that is, three litres per minute — although we would now recommend that flows of at least six litres per minute are used in order to overcome the inflation pattern shown in figure 1. This has occurred because the baby has breathed in faster than the flow of air to the resuscitation circuit. Under these conditions the circuit resistance can be considered as infinite, as this is effectively a closed system.

We do not find it surprising that the airway opening pressure rises if a baby makes expiratory efforts during a period of inflation. In this situation the resistance is not just that of the resuscitation equipment but will include all the circuit back to the pressure relief valve of the resuscitator.

Finally we would entirely agree that oesophageal pressure measurements in immature babies are likely to be unreliable quantitatively but consider that this does not invalidate their use for timing events in the respiratory cycle.

Prediction and management of nocturnal hypoglycaemia in diabetes

Sir,

Whincup and Milner have shown that under standardised conditions in hospital nocturnal hypoglycaemia can be

predicted by a blood glucose concentration of less than 7 mmol/l at 10 pm, and that nocturnal hypoglycaemia can be largely prevented by a 10 g snack of carbohydrate for those with a blood glucose below 7 mmol/l.¹ Their advice therefore for parents managing children at home, or presumably doctors managing children at British Diabetic Association (BDA) Holiday Camps would be to test the blood glucose before bed and give a 10 g snack if the blood glucose was low. Anyone who has attended BDA Holiday Camps, however (including Whincup and Milner), will agree that life is never so simple. I have collected data from two holiday camps to determine if symptomatic nocturnal hypoglycaemia can be prevented. Under BDA rules no 'extra' blood tests or interventions that might upset camp life can be imposed on the children, so the data were collected from the record cards that the children normally complete at camp.

At one camp 16 children aged 12 to 16 years went to bed at 10 pm, two hours after their last snack. Tests for blood glucose were performed on 81 occasions and on 39 the result was ≤ 7 mmol/l. The children were given carbohydrate according to the following sliding scale: glucose concentration 2 mmol/l, 30 g carbohydrate; 4 mmol/l, 20 g; 7 mmol/l, 10 g. On 87 occasions no tests were performed. Overall the results were: blood glucose concentration at 10 pm ≤ 7 mmol/l, one episode of nocturnal hypoglycaemia; > 7 mmol/l, six episodes; and where the test was not done, one episode.

At the second camp there were 43 children aged 8 to 10 years. These children went to bed straight after a snack. Their blood tests were performed at 8 pm (before the snack) and carbohydrate given according to the same sliding scale. Tests were performed on 215 occasions and were ≤ 7 mmol/l on 120 occasions. Blood tests were not performed on 377 occasions. Overall the results were: blood glucose concentration at 8 pm ≤ 7 mmol/l, 12 episodes of nocturnal hypoglycaemia; > 7 mmol/l, two episodes; and where the test was not done, 10 episodes.

There were just as many hypoglycaemic episodes when those who had a glucose concentration ≤ 7 mmol/l were given carbohydrate as there were when tests were not done and nobody was given extra carbohydrate. The value of tests before bed at camp must therefore be questioned. Camp and home conditions, unlike hospital conditions, allow all sorts of compounding factors to creep in and tests at camp may not be that accurate. Moreover we did not test the younger children at 10 pm because that would have been too intrusive.

I suspect that Whincup and Milner's data would not look so convincing if they had carried out their trial under more 'free range' conditions.

Reference

- Whincup G, Milner RDG. Prediction and management of nocturnal hypoglycaemia in diabetes. *Arch Dis Child* 1987;**62**: 333-7.

R ROWLANDSON
Department of Child Health,
University of Wales College of Medicine,
Heath Park, Cardiff CF4 4XN