‘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 measure-
ments in young infants by this technique are so unreliable3
that their use even for timing the babies’ respiratory efforts
are highly questionable.

References
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Endotracheal resuscitation of preterm infants at birth. Arch Dis
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bronchial suction on respiratory resistance in intubated preterm
3 Beardmore CS, Wong YC, Stocks J, Silverman M. Assessment
of the catheter tip pressure transducer for use in infant lung

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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 endo-
tracheal 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 pres-
sure 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 carbohy-
drate 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 confounding 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
1 Whincup G, Milner RDG. Prediction and management of
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Updated information and services can be found at:
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