of venous and capillary sampling, but it would have been interesting to have had two groups, one in which capillary sampling preceded arterial sampling and one in which it was done afterwards. Our data did not show a variation in the white cell count, and I wonder what mechanism Dr Shohat proposes for his observed rise during and after lumbar puncture.

These points emphasise the difficulties in establishing a reference range of white blood count values for infants, particularly preterm infants, and in interpreting results by comparing them with published ranges. Application of the International Committee for Standardisation in Haematology guidelines for the standardisation of blood specimen collection procedures for reference values is obviously impractical, but it is clear that the method of sampling does affect the results obtained. Many of the published ranges are based almost entirely on capillary sampling so that arterial sampling, which is commonly done in these intensively monitored infants, may lead to a diagnosis of neutropenia. Conversely, as Dr Shohat points out, a painful procedure performed shortly before sampling might cause a neutrophilia equivalent to that found in older children and adults after exercise.

Reference


The metabolic load of stored blood.
Implications for major transfusions in infants

Sir,

In a recent article Ratcliffe et al described the changes in plasma osmolality, electrolyte balance, and metabolic substrates that can occur during storage of blood. These changes, an increase in osmolality, potassium, and lactate concentrations, and a decrease in sodium concentration, were ascribed to alterations in red cell permeability and continuing metabolism during storage. Infusion of large amounts of such stored blood may be harmful to the sick infant and may, for instance, result in severe hyperkalaemia.

Interestingly, storage of fresh frozen plasma (FFP) can also produce solute concentration gradients in the infusion bags. We describe (table) the osmolality and solute concentrations incidentally measured at the surface and at the bottom of a bag containing 'unshaken' thawed plasma. To confirm this finding, we stored FFP in five 20 ml glass tubes and subsequently thawed the samples at room temperature without shaking. The following mean (SD) plasma osmolality and sodium concentrations were recorded: 237 (5) mOsm/kg and 33 (5) mmol/l at the surface, and 425 (18) mOsm/kg and 199 mmol/l at the bottom. Thorough mixing of the plasma resulted in homogenous concentrations of 321 (5) mOsm/kg and 173 mmol/l.

The differences in osmolality and concentrations in a cell free solution probably occur during freezing, which starts at the surface, and the free water is frozen first. We conclude that the infusion of large amounts of unshaken thawed plasma could harm a sick neonate, and that this phenomenon could affect the accuracy of chemical analysis of samples of thawed plasma.

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Table. Osmolality and electrolyte concentrations from surface and bottom of unit of unmixed thawed plasma.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Surface</th>
<th>Bottom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osmolality (mOsm/kg)</td>
<td>237</td>
<td>679</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>127</td>
<td>277</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>2.6</td>
<td>5.2</td>
</tr>
<tr>
<td>Chloride (mmol/l)</td>
<td>57</td>
<td>92</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>21</td>
<td>40.7</td>
</tr>
<tr>
<td>Creatinine (µmol/l)</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Protein (g/l)</td>
<td>43</td>
<td>114</td>
</tr>
</tbody>
</table>

Endotracheal resuscitation of preterm infants at birth

Sir,

The meticulous studies into the mechanics of neonatal resuscitation carried out by Professor Milner and his colleagues over the last few years provide a unique and valuable body of information. I would, however, like to clarify certain points relating to the methodology and interpretation of their most recent work. In particular, how might the flow resistance of the resuscitation equipment affect the efficacy of resuscitation? The authors do not state the flow rate into their resuscitation circuit. The fact that the pneumotachograph was linear to 15 l/minute implies that an inspiratory flow rate up to this value could be provided for.

Careful analysis of figure 1 would suggest that the effective inspiratory flow resistance of the resuscitation circuit was very high. In the figure, the 'inspiration response' of the baby caused a peak inspiratory flow rate of 5 l/minute (0.08 l/second). This was achieved only by reducing the pressure at the endotracheal tube to ~8 cm H2O. Thus the apparatus resistance (resistance pressure/flow) was at least 100 cm H2O/l/second, compared with the intrinsic resistance of an intubated preterm baby of roughly 200 cm H2O/l/second. Could this be part of the explanation for the very small tidal volumes achieved?

Finally, figure 3 needs further explanation. In it a
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 unreliable that their use even for timing the babies’ respiratory efforts are highly questionable.

References

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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 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 value 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

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