Assessment of a new device for delivering aerosol drugs to asthmatic children

C M DACOMBE, R G DALTON, D J GOLDIE, AND J P OSBORNE

Department of Clinical Chemistry and Department of Paediatrics, Southmead Hospital, Bristol

SUMMARY The effect of changes in packed cell volume was studied in two commonly used reagent strip methods (Dextrostix and Reflotest) of measuring blood glucose and in a filter paper blood spot method. It was found that the results with both the reagent strip methods were greatly haematocrit-dependent. Attention is drawn to the possibility of false diagnosis of hypoglycaemia in haemoconcentrated patients and of normoglycaemia in anaemic patients.

The use of reagent strips and reflectance meters for the rapid measurement of blood glucose is now common, particularly for the diagnosis of hypoglycaemia in the neonatal period and for the management of diabetics. However, in both the sick neonate and the uncontrolled diabetic wide fluctuations in packed cell volume (PCV) occur. In particular, in the neonate the PCV may increase to 70% or more. It is therefore important that any method of estimating blood glucose concentration should not greatly depend on PCV.

Previous workers1 2 have suggested that the effect of PCV on the glucose results obtained with one reagent strip/reflectance meter method (Dextrostix/Eyetone) is negligible within the range of 30–50% which they studied. However, this is a fairly narrow range and it is not possible from their results to estimate the effect of more pronounced changes in PCV.

We therefore decided to examine the effect of changes in PCV over a range from 20 to 80% on blood glucose estimations by two widely used reagent strip and reflectance meter methods. We also investigated the effect of PCV on whole blood glucose concentrations measured in filter paper blood spots.3

Material and methods

Dextrostix test strips and an Eyetone meter were provided by the Ames Company and Reflotest strips and a Reflotmat meter by the Boehringer Corporation London Ltd. Each system was used strictly in accordance with the manufacturer's instructions, including frequent checks of quality control.

Filter paper blood spot measurements were made using the glucose oxidase method of Wakelin et al.4

Sixteen samples of 25–40 ml venous blood from

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Correspondence to Professor A D Milner, Department of Child Health, E Floor, East Block, University Hospital, Queens Medical Centre, Clifton Boulevard, Nottingham NG7 2UH.

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healthy volunteers and diabetic patients were collected in heparinised tubes. Each sample was divided into 7 or 8 aliquots. One aliquot was used for the assay of plasma glucose by an autoanalyser method (either neocuproine or hexokinase).

The packed cell volumes of the remaining aliquots were adjusted to 20%, 30%, 40%, 50%, 60%, 70%, and, if sufficient aliquots were available, to 80%. This adjustment was achieved by measuring the initial PCV of the sample and then by redistributing calculated volumes of plasma from the aliquots after centrifugation. The adjusted PCV of each sample was confirmed by direct measurement.

The two reflectance meter methods and the filter paper blood spot method were each used to determine the blood glucose concentration on 8 sets of samples. Each determination was performed in triplicate and the mean value taken. When the assay was by a reflectance meter method it was carried out within 2 hours of collecting the blood and when by means of the blood spot method within 24 hours. No preservative was used.

Results

The plasma glucose concentrations as measured by the routine automated methods ranged from 3 to 21 mmol/l (7 samples <5 mmol/l, 5 samples 5–10 mmol/l, 4 samples >10 mmol/l).

<table>
<thead>
<tr>
<th>Case</th>
<th>Serum glucose by automated hexokinase method (mmol/l)</th>
<th>Blood glucose by Dextrostix/Eyetone at various PCVs (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.1</td>
<td>4.0 1.5 3.4 3.0 2.9 1.8 1.5</td>
</tr>
<tr>
<td>2</td>
<td>16.1</td>
<td>21.0 18.9 17.0 13.9 10.8 8.6 6.5</td>
</tr>
</tbody>
</table>

For ease of comparison the recorded blood glucose concentration at differing PCVs for each method was expressed as a percentage of the value obtained in the aliquot with a PCV of 40%. The Figure shows the pooled results of these values plotted as mean ± the standard error of the mean versus PCV. There was a progressive reduction in recorded blood glucose concentration with increasing PCV with both reagent strip systems but there was no change with the filter paper blood spot method. The changes were more pronounced with Dextrostix than with Reflotest. The magnitude of the effect was found to be independent of the initial blood glucose concentration (Table).

There was good agreement between the automated plasma glucose results and the whole blood filter paper results at all levels of PCV, but in the case of the reagent strips a good agreement with the automated plasma glucose result was observed only in samples with a PCV of 40%.

Discussion

The fact that the whole blood glucose when measured by the filter paper blood spot method did not alter with changing PCV showed that adjusting the PCV did not in itself cause an appreciable change in whole blood glucose concentration. Furthermore the good agreement between the whole blood glucose concentration and the plasma glucose measured by the routine automated methods indicated that there was no appreciable difference between whole blood and plasma glucose.

Previous work suggested that the effect of PCV on the Dextrostix/Eyetone system was negligible over a narrow range of PCV. The present work, over an extended range of PCV values, points to the importance of PCV on the results obtained with the two reagent strip methods. The reason for the changes in observed glucose with changing PCV is not clear, but such changes cannot reflect differences between plasma and whole blood glucose and the fact that there were marked differences between the two systems also makes such an explanation unlikely. It seems probable that the differences reflect...
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the ease with which glucose can diffuse into the pads of the test strips.

These observations show that if the PCV is greatly abnormal, the Destrostix/Eyetone system (and to a lesser extent the Reflotest/Reflomat system) will give misleading results. This is mainly of relevance in polycythaemic patients, particularly in small-for-dates neonates who are routinely screened for hypoglycaemia and in whom a false diagnosis may be made if glucose concentrations are assessed only by a reagent strip method. Neonates with rhesus isoimmunisation are also screened for hypoglycaemia and since they can be severely anaemic a false diagnosis of normoglycaemia may be made.

References


Correspondence to Dr D J Goldie, Department of Clinical Chemistry, Southmead Hospital, Westbury-on-Trym, Bristol BS10 5NB.

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Tracheal compression by an anomalous innominate artery
A report of 2 cases in a family

JACQUELINE Y Q MOK AND HAMISH SIMPSON

Royal Hospital for Sick Children, Edinburgh

SUMMARY The case histories of 2 brothers who presented with stridor soon after birth are described. In each, tracheal compression resulted from an anomalous innominate artery. We know of no previous familial reports of this condition.

The clinical entity of an anomalous innominate artery compressing the trachea and causing respiratory symptoms was first recognised by Gross and Neuhauer in 1948.1 However, scepticism exists as to the clinical significance of this anomaly, as it can be an incidental finding in asymptomatic subjects. The 2 brothers presented here had had severe stridor since birth caused by compression of the trachea by an anomalous innominate artery, requiring surgery for its relief.

Case reports

Case 1. The patient, a boy, was born after a normal pregnancy to healthy and unrelated parents. Delivery was by assisted breech, and he weighed 3.35 kg at birth. He was asphyxiated with an Apgar score of 1 at one minute. Endo-tracheal intubation was carried out without any difficulty, and he was given intermittent positive pressure ventilation for 5 minutes. Thereafter he breathed normally and was transferred to the nursery.

At 6 hours, he developed inspiratory stridor with cyanosis and substernal recession. Laryngoscopy showed that the uvula, epiglottis, and vocal cords were red and slightly oedematous. This was attributed to previous intubation and he was allowed to settle in oxygen. Within hours, his condition deteriorated and endo-tracheal intubation was repeated to relieve respiratory distress. During the next 10 days, several trials of extubation resulted in recurrence of the symptoms. He was then transferred to the Royal Hospital for Sick Children in Edinburgh for further investigations. Clinical examination showed a small, scrawny infant with an endo-tracheal tube in situ. No other abnormalities were noted. Serum sodium, potassium, calcium and urea, blood gas tensions and pH, and a full blood count were normal. Bacteriological cultures from throat, umbilicus, and blood were negative. Routine chest x-ray film was normal, but lateral views of the neck during a barium swallow showed anterior indentation of the air-filled trachea at the level of T3 without associated compression of the oesophagus. Angiographic studies subsequently confirmed an anomalous origin of the innominate
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C M Dacombe, R G Dalton, D J Goldie and J P Osborne

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