regional oncology centres and the school health services in the surrounding health districts. Only in this way can schools and individual teachers be given appropriate support, which includes adequate confidentiality safeguards.

References

M Rogers Community Child Health, Macclesfield District General Hospital, Victoria Road, Macclesfield, Cheshire SK10 3BL

Glycosylated haemoglobin and cystic fibrosis

Sir,

Flückiger recently reported interference in glycosylated haemoglobin A1 (HbA1) estimation by penicilloylated haemoglobin. He concludes that measurements of HbA1 can be misleading in patients with cystic fibrosis receiving treatment with penicillins because of covalent binding of the penicilloylo moiety to haemoglobin producing HbA1 mobility of the respective haemoglobins. Raised HbA1 concentrations were found in patients with cystic fibrosis treated with long term β lactam antibiotics who did not have diabetes. Lower HbA1 concentrations were obtained on converting thiobarbituric acid values to the HbA1 equivalent for comparison. Although it is stated that the patients with cystic fibrosis did not have diabetes, it is not reported whether glucose tolerance tests were completed to determine if there was impaired glucose tolerance in these patients.

We studied 64 patients with cystic fibrosis measuring HbA1 by ion exchange column chromatography and spectrophotometry (Biorad haemoglobin A1 by column test) expressed as a percentage of the total haemoglobin. Our laboratory reference range is 5–3–8–8% representing the SD limits of the mean in normal paediatric departments. Forty-three patients with cystic fibrosis had a mean (SD) HbA1 concentration of 7–8 (0–9)%, range 5–9–8–8. This was not statistically different from the mean HbA1 concentration of 7–4 (0–94)% of 21 normal children admitted for routine operations. All the patients with cystic fibrosis were on long term antibiotic prophylaxis mainly with β lactam antibiotics. HbA1 concentrations were obtained on each of the 43 patients with cystic fibrosis on one to four occasions. There was no statistical difference between the mean HbA1 concentration of 7–6 (0–9)% (n=43) of the patients treated with flucloxacillin for at least a two to three month period before estimation and the mean HbA1 concentration of 7–7 (0–8)% (n=24) for patients on both flucloxacillin and ampicillin. These did not differ from the HbA1 concentrations of the control population. We found evidence of impaired β cell function in patients with raised HbA1 concentrations by measurement of C peptide concentrations during oral glucose tolerance test. Thus there was no evidence from our study that β lactam antibodies produce falsely raised HbA1 concentrations using the Biorad method. Although Flückiger reports a potential problem, the evidence that it should deter the measurement of HbA1 in patients with cystic fibrosis other than by specific techniques is lacking.

References

P R Stutchfield and D Isherwood Institute of Child Health, Alder Hey Children’s Hospital, Eaton Road, Liverpool L12 2AP
Glycosylated haemoglobin and cystic fibrosis.

P R Stutchfield and D Isherwood

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