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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


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Glycosylated haemoglobin and cystic fibrosis

Sir.

Flückiger recently reported interference in glycosylated haemoglobin A\textsubscript{1} (HbA\textsubscript{1}) estimation by penicilloylated haemoglobin.\textsuperscript{1} He concludes that measurements of HbA\textsubscript{1} can be misleading in patients with cystic fibrosis receiving treatment with penicillins because of covalent binding of the penicilloyl moiety to haemoglobin producing HbA\textsubscript{1} mobility of the respective haemoglobins. Raised HbA\textsubscript{1} concentrations were found in patients with cystic fibrosis treated with long term \beta lactam antibiotics who did not have diabetes. Lower HbA\textsubscript{1} concentrations were obtained on converting thiobarbituric acid values to the HbA\textsubscript{1} 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 HbA\textsubscript{1} by ion exchange column chromatography and spectrophotometry (Biorad haemoglobin A\textsubscript{1} by column test) expressed as a percentage of the total haemoglobin. Our laboratory reference range is 5.3–8.8% representing 2 SD limits of the mean in normal paediatric departments. Forty three patients with cystic fibrosis had a mean (SD) HbA\textsubscript{1} concentration of 7.8 (0.9)%, range 5.9–8.8%. This was not statistically different from the mean HbA\textsubscript{1} 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 \beta lactam antibiotics. HbA\textsubscript{1} 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 HbA\textsubscript{1} concentration of 7.6 (0.9)% (n=43) of the patients treated with flucloxacinil for at least a two to three month period before estimation and the mean HbA\textsubscript{1} concentration of 7.7 (0.8)% (n=24) for patients on both flucloxacinil and ampicillin. These did not differ from the HbA\textsubscript{1} concentrations of the control population. We found evidence of impaired \beta cell function in patients with raised HbA\textsubscript{1} concentrations by measurement of C peptide concentrations during oral glucose tolerance test.\textsuperscript{2}

Thus there was no evidence from our study that \beta lactam antibodies produce falsely raised HbA\textsubscript{1} concentrations using the Biorad method. Although Flückiger reports a potential problem, the evidence that it should deter the measurement of HbA\textsubscript{1} in patients with cystic fibrosis other than by specific techniques is lacking.

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Glycosylated haemoglobin and cystic fibrosis.

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Arch Dis Child 1988 63: 1000
doi: 10.1136/adc.63.8.1000

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