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

- ¹ Stevens MCG, Kay JJ, Kenwood CF, Mann JR. Facts for teachers of children with cancer. *Arch Dis Child* 1988;**63**:456-8.
- ² British Paediatric Association. *Report on the school health services*. London: British Paediatric Association, 1987.

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Glycosylated haemoglobin and cystic fibrosis

Sir,

Flückiger recently reported interference in glycosylated haemoglobin A₁ (HbA₁) estimation by penicilloylated haemoglobin.¹ He concludes that measurements of HbA₁ can be misleading in patients with cystic fibrosis receiving treatment with penicillins because of covalent binding of the penicilloyl 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 2 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.²

Thus there was no evidence from our study that β lactam antibiotics 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

- ¹ Flückiger R. Glycosylated haemoglobin in cystic fibrosis. *Arch Dis Child* 1988;**63**:223-4.
- ² Stutchfield PR, O'Halloran S, Teale JD, Isherwood D, Smith CS, Heaf D. Glycosylated haemoglobin and glucose intolerance in cystic fibrosis. *Arch Dis Child* 1987;**62**:805-10.

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