Dr Conway comments:
I am pleased to read of Dr Haque’s interest in our paper and apologise for not referring to his work, of which I am aware. None the less, I think it is difficult to extrapolate from his population to ours, for reasons I have already outlined.

Dr Haque, in reference to our work, questions the ‘justification of giving weekly painful and costly injections’. At the time of the conception and early stages of our study, intravenous immunoglobulin preparations had not received full product licencing in this country. May I remind Dr Haque that our study was approved by the hospital ethical committee and that we would not have given these infants intramuscular injections except as a logical extension of our previous work and in reasonable expectation of their being of benefit to the child. The cost of prophylactic immunoglobulin treatment, if successful, is not more than offset by the saving in expensive antibiotic use.

I absolutely refute Dr Haque’s suggestion of possible nursing bias. The substantiation of a doctor’s academic hypothesis weighs little with our intensive care nurses compared with the continued well being of their patients. Moreover, in a busy regional unit with much active research in progress, every nurse was not necessarily always aware of which baby was, or was not, receiving immunoglobulin treatment.

The preparation used contained 250 mg in 1.7 ml and volumes injected were therefore small. As stated, injections were at weekly intervals until discharge to home. We were also not surprised that the concentrations of serum IgG were below those of term infants, for reasons similar to those described by Dr Haque. We did not measure IgM concentrations.

The insertion of an endotracheal tube it is reasonable also to pass a nasogastric tube and if large volumes of green liquid are obtained from the stomach this suggests that the problem is intestinal obstruction and not in utero passage of meconium.

References

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Facts for teachers of children with cancer
Sir,

The valuable paper by Dr Stevens and colleagues raises important issues.

1. Teachers should be more informed in a general sense; in as much detail as they themselves would find helpful, about conditions such as malignant disease, cystic fibrosis, and diabetes (the most obvious examples) which can have a pervasive effect on the well being of children in school.

2. As an important general rule (to which there may be exceptions in individual circumstances), teachers should not be given medical details about individual children. The confidentiality aspects of passing on such information cannot be controlled in mainstream or even special schools in the way which is presumably practicable in the context of teachers’ work in the health services dominated environment of a hospital.

Doctors who do not work regularly in schools may be unaware of the extent to which educational records are increasingly ‘open’, with corresponding availability to staff of any medical information in writing which goes to the school, and the encouragement of informal exchanges of information. Medical matters tend to fascinate large numbers of the non-medical public, including professionals, and we can neither expect nor monitor an adequate level of respect for the confidentiality of certain information in non-medical environments such as schools. A 13 year old girl with advanced malignant disease was quoted recently on television as saying that ‘everybody’ at her school knew about her condition and its implications, and that this single circumstance caused her more misery than anything else.

3. Dr Stevens et al quote the British Paediatric Association Report on the school health services but do not directly mention the school health service. In my view a relevant school health service could and should make it its business to provide appropriate support and advice to schools and individual teachers as necessary about individual children, which implies close links between
Correspondence

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 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 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 A1c 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. 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.

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