valuable insight into the effects of low blood glucose on neuronal function and again underlines the risk of overscrupulous attention to glycaemic control in insulin dependent diabetes.

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Rare cancers and specialist centres

SIR,—Who treats cancer in Britain has become a never ending saga. At a time when resources are scarce we have to take a hard look at value for money from different configurations of cancer service. The paper by Dr M B McIllmurray and others (8 March, p 1986) may be plausible to those outside the specialty but is clearly misdirected if used as a model on which to base cancer services throughout the United Kingdom. Do the authors really think that by treating three patients with germ cell tumours over as many years they can gain the necessary experience to manage reliably this highly curable but complex malignancy? Do they feel able to work without constant contact with specialist colleagues in radiotherapy, surgery, and pathology in managing difficult clinical problems? While the general care of a cancer patient should be carried out locally by appropriate health care teams, including the patient's own general practitioner, specialist care must be firmly in the hands of those concerned full time in cancer management. To bring the latest advances to the bedside of the cancer patient is a full time endeavour whether in the general hospital or special centre.

The saddest feature of the paper is the absence of the visiting radiotherapist from the authorship. It is only by close cooperation that cancer treatment can move into the twenty first century.

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SIR,—There is much of value in both the leading article by Dr R T D Oliver (8 March, p 641) and also the article by Dr M B McIllmurray and colleagues (p 669)

Surely all that is needed to link the two totally is a commitment from a district medical oncology service not to treat the rare diseases—that is, paediatric malignancies, lymphomas, leukaemias, germ cell tumours—which need the experience of the regional centre, but to treat the common solid tumours. There is increasing evidence that cytotoxic chemotherapy after surgery in patients with breast cancer improves survival,[1] and this may also be true of squamous cell cancer of the head and neck,[2] of pancreatic cancer,[3,4] and of stomach cancer. At present most patients with these diseases are not offered any cytotoxic chemotherapy because of the lack of facilities of the regional centres.

Surely the medical oncology services based on district general hospitals should concentrate on these diseases rather than on lymphomas and acute leukaemia, which made up 31% of the cases seen in the Lancaster medical oncology service but only 4% of the total registrations of malignant disease in the North Western region (Office of Population Censuses and Surveys, 1981). This would allow a district service to develop its own skill in treating common tumours; there are enough patients with these tumours to do worthwhile research; and such a policy would allow patients with rare disease the benefit of the specialist skills of the regional centres.

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Risk of AIDS to health care workers

SIR,—In discussing the risk to laboratory staff from human T cell lymphotropic virus type III (HTLV-III) Professor Alasdair M Geddes has focused mainly on patient samples (15 March, p 711). Little attention has been paid to the standard and quality control materials which are handled by laboratory staff throughout the working day (and night).

It is easy to lose sight of the fact that these materials are frequently prepared from pooling sera from many individuals, who are often paid donors. In view of this 32 different reagents from 11 suppliers (based in the United Kingdom and North America) used in our chemical pathology department were tested for HTLV-III antibody: 17 were positive by the more sensitive Wellcome assay, six were also positive by the Organon assay, and one was positive by an indirect immunofluorescence assay. The manufacturing process required to produce these reagents and the dilution of the individual sera contributing to these pools complicates the interpretation of these HTLV-III antibody results. It is therefore important that, rather than screen the final product, manufacturers should exclude samples from high risk groups and use only HTLV-III negative sera. The containers of these reagents should also be improved to prevent the common laboratory accident of fingers being cut on the sharp edges of glass ampoules or the metal closures that hold container stoppers in place. Reduction of the risk of infection by such measures as heating may be possible for some analytes but not all—for example, thermolabile compounds or those in which protein binding affects the analyte of interest.

The recent report that no evidence of spread of HTLV-III virus from patient to laboratory staff could be found in spite of regular contact with specimens from infected patients provided hepatitis B type precautions were observed is reassuring.[1] However, a clear difference exists between the hazard presented by the infrequent and recognised "high risk" patient sample that may enter the laboratory and the risk from the day to day use of contaminated standards and quality control materials that are already in laboratories. Apart from drawing the attention of other laboratories and the manufacturers to this problem, these findings should reinforce the need to implement and maintain the safety codes of practice when working with both patient samples and serum based laboratory reagents.

We thank the virology laboratory, John Radcliffe Hospital, Oxford, for performing the HTLV-III antibody tests.

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Profound hypophosphataemia in patients collapsing after a "fun run"

SIR,—Dr Dale's article (15 February, p 447) is interesting though not entirely surprising. Exercise inevitably means high levels of catecholamines, and large compartmental shifts of phosphate (down to 0·25 mmol/l (0·77 mg/100 ml)) and potassium (to 1·9 mmol/l (5·9 mg/100 ml)) have been observed following overdoses of sympathomimetics.[1]

Hypokalaemia is a common finding in clinical situations where circulatory adrenaline is raised—for example, after myocardial infarction.[2,3] In these acute clinical situations phosphate concentrations are rarely measured.

The hypokalaemia caused by adrenaline is similar in its mechanism to that caused by salbutamol—that is, a β_2 receptor mediated potassium flux.[4,5]

Similar substantial falls in phosphate and potassium, seemingly in parallel with each other, have been shown in my recent trial (unpublished) of giving 10 mg nebulised salbutamol to healthy subjects at rest. The fall in phosphate was quick, occurring within 20 minutes, and values had almost completely returned to normal at two hours (table).

Phosphate concentrations (mmol/l) in 12 healthy subjects after salbutamol

Case	Time				
	0 min	10 min	20 min	40 min	120 min
1	0·86	0·42	0·46	0·58	0·88
2	1·28	0·94	0·96	0·96	1·10
3	1·02	0·76	0·74	0·80	1·06
4	0·78	0·72	0·80	0·82	1·06
5	0·94	0·84	0·84	0·78	0·90
6	0·90	0·74	0·70	0·74	0·76
7	1·32	1·06	0·98	1·00	1·08
8	1·28	1·10	1·02	0·94	0·96
9	1·02	0·72	0·66	0·74	0·86
10	1·30	1·10	1·02	1·02	1·16
11	1·10	0·92	0·88	0·92	1·04
12	0·92	0·80	0·82	0·82	0·96

Conversion: SI traditional values—Phosphate: 1 mmol/l≈3 mg/100ml.

Although intravenous salbutamol raises plasma, insulin, and glucose values,[6] intravenous adrenaline does not raise insulin concentrations,[4] and insulin has been shown not to have any effect in β_2 receptor mediated hypokalaemia.[7]

Therefore I believe that the fall in phosphate, seen in both my study and Dr Dale's, may represent an intracompartmental shift of phosphate mediated via β_2 cell membrane receptors and may be seen often in conditions when circulatory catecholamines are raised, such as severe exercise.