from the humidifier water heaters may often have been suppressed owing to negative feedback from the temperature probes, which were not protected from the radiant heat source.

The Fisher Paykel systems produced lower humidity as gas flow increased, while the Bennett system achieved greater humidity at increasing flow rates. Electronic hygrometry may prove a useful method for comparative assessment of humidifiers.

Inadequate humidification of the respiratory tract may reduce mucociliary clearance and predispose to chronic lung disease in intubated infants. In conditions in which ciliary motility is deficient, such as cystic fibrosis or Kartagener's syndrome, there is chronic retention of secretions with radiographic appearances like those seen in the chronic lung disease of mechanically ventilated infants. Obstruction of small airways by secretions might also increase the risk of peripheral gas trapping and air leak.

As there are no data to define optimum humidity during ventilatory support of infants prospective randomised studies are needed to investigate (i) the relation between acute and chronic respiratory complications and ventilator circuit humidity and (ii) whether intermittent instillation of saline into

the endotracheal tube reduces complications by increasing respiratory tract humidity.

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Intellect after malignancy

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SUMMARY A previous study has shown that significant intellectual deficits exist in children treated for leukaemia but not in those with solid tumours. Unexpectedly, the deficits had not increased in the two years since the original study, suggesting that the nadir had already been reached five years after diagnosis.

The improving survival rates for children with cancer have led to a greater awareness of the side effects of treatment and their long term consequences. A study of intellectual function after treatment for leukaemia or solid tumours using sibling controls showed deficits in both groups,¹ but these were consistently larger in the group with leukaemia. There was a suggestion that the deficits in children after treatment for leukaemia might be progressive, whereas for the other group they might

be improving. This report details a further investigation of the same group of children two years after the initial assessment.

Method

Nineteen of the original group of 23 children with leukaemia (12 boys and seven girls) and 12 of the original group of 19 with solid tumours (eight boys and four girls) agreed to be retested with their siblings. Further details of the two groups are given in Table 1. Eleven of the patients with leukaemia and six of the patients with solid tumours were younger than their sibling.

Each child received the necessary four British ability scales according to their age.² Thus all received the matrices test (abstract reasoning), the similarities test (verbal reasoning), and the recall of digits test (immediate memory). Those over 8 years had the speed of information processing test and

Table 1 Age (range, mean (SD)) in months at diagnosis and testing in the groups with leukaemia and solid tumours

Group	At diagnosis	At testing	
	Patients	Patients	Siblings
Leukaemia	16-106 52.6 (24.2)	99-202 139.2 (27.7)	88-201 133.4 (37.5)
Solid tumour	9-121 62.3 (31.4)	81-234 130.3 (43.7)	61-186 134.4 (38.1)

Table 2 Mean (SD) IQs of patients and siblings in the groups with leukaemia and tumours at initial assessment and follow up assessment

Group	Mean IQ			
	Initial Assessment		Follow up assessment	
	Patients	Siblings	Patients	Siblings
Leukaemia	99.2 (15.2)	107.9 (11.3)	98.3 (11.3)	109.1 (12.2)
Tumour	108.5 (10.6)	108.6 (12.9)	107.6 (14.0)	110.6 (11.3)

those from 5 to 8 years the naming vocabulary test (retrieval and application of knowledge).

The mean (SD) test-retest interval was 20.4 (1.6) months in the group with leukaemia and 18.8 (1.4) in the group with solid tumours.

Analyses were carried out using the total intelligence quotient (IQ) (made up of all tests) and the IQ profile (graphic representation of each test making up the IQ).

Results

Table 2 shows the mean IQs of patients and siblings in both the groups at initial and follow up assessment. Repeated measures analysis of variance showed no significant IQ changes over time from the first assessment in any of these groups. There was a trend that suggested that patients with leukaemia consistently scored below their siblings to a greater extent than the patients with solid tumours ($F=3.75$, $df\ 1,29$).

Comparisons of the IQ profile were made for all patients and siblings who were older than 8 years at initial assessment. These subjects had received exactly the same subtests on both occasions. There were 11 in the group with leukaemia and six in the group with solid tumour. Analysis showed patients' scores significantly lower than siblings' in the group with leukaemia ($F=7.49$, $df\ 1,70$, $p<0.001$), but there was no shape difference in the profiles—that is, the deficit encompassed all functions tested.

There were no such differences in the group with solid tumours.

Discussion

The present study confirms the previous finding that children who have been treated for acute lymphoblastic leukaemia have significantly lower IQs than their siblings and that there is no difference between patients and siblings in the group with solid tumours. The difference between the two groups of patients is probably due to the central nervous system (CNS) prophylaxis, using cranial irradiation and intrathecal methotrexate, received only by the group with leukaemia. The initial study was cross-sectional in that it studied children at different periods after treatment. In the first study it seemed that the deficits in the group with leukaemia were larger the longer after diagnosis that the assessment had been made, and the converse was true for the group with solid tumours. It was tentatively suggested, therefore, that deficits in the group with leukaemia might be progressive, but this has not been borne out by further study. This is in contrast to other studies by Meadows *et al*, who found delayed decrements occurring after three years,³ and Eiser, who with a longitudinal study found global rather than specific deficits occurring with time.⁴ It has been suggested that the effects of radiation may be delayed by an average of three years.⁵ In the present study patients had lived an average of five years from diagnosis at initial assessment and almost seven years at follow up. They may have therefore passed the time of maximum progression of the effects of radiation, and this might explain why their intellectual function is no longer declining. There is no evidence, however, to suggest any recovery of function. Whether or not this deficit is permanent will require prolonged follow up. It is hoped that by reducing the dose of cranial irradiation, as in the MRC UKALL VIII study, less intellectual impairment will be seen in the future, but any further reduction in the irradiation dose must await long term observation to ensure that there is not a recrudescence of the problem of CNS leukaemia.

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Intralipid microemboli

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SUMMARY A baby girl died after receiving intravenous Intralipid. At necropsy a pulmonary Intralipid microembolus, unrelated to the cause of death, was found. Serum taken immediately before infusion agglutinated Intralipid. C reactive protein concentration was raised. This supports the theory that C reactive protein may agglutinate Intralipid in vivo, causing embolisation.

There have been a number of published reports describing lipid emboli in the tissues of neonates receiving intravenous fat emulsions.¹⁻³ Intralipid tends to be agglutinated in vitro (creaming) by sera containing significantly raised concentrations of C reactive protein⁴ and is also agglutinated by purified

C reactive protein in the presence of calcium ions. It has been postulated that this phenomenon may occur in vivo, resulting in embolisation of Intralipid agglutinates.^{3,4} We describe a case that supports this hypothesis.

Case report

A baby girl was born by elective caesarean section at 34 weeks for breech presentation. She was found to have a tracheo-oesophageal fistula, which was surgically closed the following day. She remained critically ill and four days postoperatively was started on parenteral nutrition, including 10% Intralipid at an infusion rate of 0.08 g/hour. Immediately before infusion serum was obtained for estimation of C reactive protein concentration and a creaming test⁴

Figure (a) Intralipid microembolus (stained with Sudan black); (b) Normal appearance of Intralipid in serum; (c) Intralipid agglutinated (creamed) by serum.