LETTERS TO THE EDITOR

Changes in deep body temperature before a cot death

EDITOR,—Infection has been implicated in sudden infant death, although its precise role remains uncertain as only minimal necropsy changes are seen in many cases. The infant described here had many cot death risk factors and showed no outward signs of illness and no specific necropsy changes.

Case report

A boy born at 35 weeks' gestation to an 18 year old gravida 2 mother, weighed 2475 g with Apgar scores of 5 and 8 at 1 and 5 minutes. After resuscitation at birth, the baby required two days of ventilation for respiratory distress during which time he was treated with antibiotics, given a bolus of albumin, and 10% dextrose for an episode of hypoglycaemia. Phototherapy was used for one day. Bottle feeding commenced at 4 days of age, he showed good weight gain, and was discharged home weighing 2740 g on day 18.

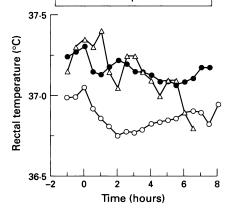
He lived with his mother and her partner, both smokers, and a brother aged 18 months. He had no previous illness, was receiving multivitamin drops daily, and was feeding well.

At 8 weeks of age this infant was recruited to a research study aimed at assessing the effects early infection have on the deep body temperature pattern. This study involved the use of three thermister probes, a soft anal probe inserted into the rectum to 5 cm from the anal margin, a skin probe attached to the right shin, and a third probe to measure the ambient room temperature, placed in the cot about one foot from the sleeping infant. All three probes were attached to a Grant data Squirrel logger, set to record the temperatures at minute intervals, throughout the night from one hour before bedtime until the next morning.

On the night of monitoring the baby was well, but the baby's brother had a cough and cold with a runny nose. The baby weighed 4450 g, went to sleep at 23.30 hours, woke for a feed at 01.30, and was put to bed at 02.40 hours. He was placed supine in a crib, wearing clothing and covers totalling 9.7 tog (Shirley Institute, Manchester). All members of the family slept in the same room. The mean (SD) overnight ambient room temperature was 28.47 (3.64)°C. Monitoring was commenced at 17.30 hours and continued throughout the night until 10.00 hours the next morning when the baby was found dead in his crib, in the semiprone position.

At postmortem examination no specific abnormality was discovered. Vitreous humour electrolyte concentrations were normal. No pathogens were isolated from blood, cerebrospinal fluid, lungs, or faeces. There was no marked cellular infiltrate in the lungs to suggest any infection and virology studies on lung tissue showed no influenza A and B, adenovirus, or respiratory syncytial virus. Histology revealed a very fatty liver but there were no lipid deposits in the renal tubules or other organs so that a primary metabolic problem was unlikely. The liver appearances suggested secondary metabolic

- △ Cot death
- Babies in prodromal phase
- o Normal temperature curve



Overnight rectal temperature patterns of babies showing normal temperature patterns, babies showing raised temperature patterns during the prodromal phase of illness, and the rectal temperature pattern of the baby who died. Time 0 indicates bedtime; each point shows the mean (SEM).

upset, possibly due to an infective process. Cause of death was recorded as cot death.

The figure shows the deep body temperature pattern of the dead baby for eight hours before death. The temperature was consistently raised above 37°C from the time monitoring commenced until five hours after being put to bed when death (presumably) occurred and the temperature fell. The fall in temperature to below 36·8°C,² which normally occurs with sleep in babies of this age, was absent, and produced an overall pattern similar to that of other babies monitored during the prodromal phase of infections.

The disturbance of this baby's deep body temperature mimics those changes seen in prodromal illness elsewhere³ with a period of maximal weight loss and heart rate elevation. Should death occur during this period, no clinical signs are observed and necropsy findings are normal, while body weight is reduced.⁴

J A JACKSON S A PETERSEN P A MCKEEVER M P WAILOO University of Leicester, School of Medicine, Clinical Sciences Building, Leicester Royal Infirmary, PO Box 65, Leicester LE2 7LX

- 1 Wailoo MP, Petersen SA, Whittaker H, Goodenough P. Sleeping body temperatures in 3-4 month old infants. Arch Dis Child 1989; 64: 596-9
- 2 Lodemore M, Petersen SA, Wailoo MP. Development of night time rhythms over the first six months of life. Arch Dis Child 1991; 66: 521-4.
- Jackson JA, Petersen SA, Wailoo MP. Body temperature changes before minor illness in infants. Arch Dis Child 1994; 71: 80-3.
 Petersen DR, Benson EA, Fisher LD, Chinn NM,
- 4 Petersen DR, Benson EA, Fisher LD, Chinn NM, Beckwith HB. Postnatal growth and the sudden infant death syndrome. A prospective study. Am J Epidemiol 1974; 99: 389-94.

Patterns of obesity in boys and girls after treatment for acute lymphoblastic leukaemia

EDITOR,—We read with interest the paper by Odame *et al* demonstrating that obesity is a late effect in children who have received cranial irradiation as part of treatment for acute lymphoblastic leukaemia (ALL).¹

We have also retrospectively assessed body mass indices in two groups of children, at least two years after treatment for malignancy in Cardiff. Group 1 (12 boys and 26 girls) were long term survivors of ALL treated on standard MRC protocols, including cranial irradiation (18 Gy (n=32) or 24 Gy (n=6)). Group 2 (16 boys and 21 girls) were long term survivors of other childhood malignancies, (eight acute myeloblastic leukaemia, four non-Hodgkin's lymphoma, two Hodgkin's lymphoma, nine Wilms' tumour, seven neuroblastoma, three rhabdomyosarcoma, three yolk sac tumours, and one Ewing's sarcoma), who received cytotoxic chemotherapy but no radiotherapy. Statistically there was no difference in age at diagnosis between the two groups (median 3.4, range 0.9-9.9 years for group 1 and median 3.0, range 0.4-13.4 years for group 2). Body mass indices were expressed as SD scores (BMI SD score) derived from the Tayside Growth Study² and are shown in the table below.

Whereas in group 1 both sexes had significant increases in BMI SD score in the years after diagnosis (girls more so than boys), in group 2 this was only transiently the case for girls and did not persist beyond four years after diagnosis. Interpretation of data beyond six years from diagnosis is limited by the small numbers of cases who have survived for this length of time in remission.

Our data, therefore, provide further evidence that obesity is a persistent late effect of treatment for ALL, particularly in girls. When compared with children treated for other malignancies, the differences in body composition tends to support the hypothesis that cranial irradiation may be a major aetiological factor. As obesity during adolescence may have future health and socioeconomic consequences,³ research is currently underway in our unit to investigate and elucidate the precise mechanisms for the onset of obesity in these patients.

J T WARNER J W GREGORY Department of Child Health, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN D K H WEBB

DR HWEBB
Department of Child Health,
Llandough NHS Trust Hospital,
Penarth,
South Glamorgan CF64 2XX

- Odame I, Reilly JJ, Gibson BES, Donaldson MDC. Patterns of obesity in boys and girls after treatment for acute lymphoblastic leukaemia. Arch Dis Child 1994; 71: 147-9.
 White EM, Wilson AC, Greene SA, et al. Body
- 2 White EM, Wilson AC, Greene SA, et al. Body mass index centile charts to assess fatness of British children. Arch Dis Child 1995; 72: ???-??.
- 3 Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 1993; 329: 1008-12.

Mean (SE) BMI SD scores at two yearly intervals from diagnosis of malignancy

	Diagnosis	+2 Years	+4 Years	+6 Years	+8 Years
Group 1	-0·27 (0·18)	0·55 (0·15)*	0·67 (0·18)*	0·76 (0·26)†	1·12 (0·28)†
No	32	38	38	21	15
Group 2	-0·16 (0·22)	0·39 (0·18)*	0·29 (0·21)†	0·16 (0·30)	-0·71 (0·29)
No	25	35	24	14	

^{*}p<0.001; †p<0.01 compared with BMI SD score at diagnosis.