

Association of diabetes and coeliac disease

Sir,

I read with interest the report of Dr. Chambers (1975) in the *Archives*, describing yet another child with coexistent coeliac disease and diabetes mellitus and also for the first time coexistent hyperthyroidism. The association between diabetes mellitus and coeliac disease is now well documented (Walker Smith, Vines, and Grigor, 1969; Visakorpi, 1969; Thain, Hamilton, and Ehrlich, 1974). One partial explanation for this association may be a common genetic predisposition. Approximately 80% of children with coeliac disease have the histocompatibility antigen HL-A 8 (McNeish, Nelson, and MacKintosh, 1973). Cudworth and Woodrow (1974) have also found that 54% of patients with juvenile onset diabetes have the HL-A 8 antigen. It has been suggested that one or more immune response genes predisposing to coeliac disease are in linkage disequilibrium with HL-A 8 (Strober, 1974). This may be the basis for predisposition to both coeliac disease and diabetes mellitus that possession of these HL-A 8 antigens appears to endow.

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Fatal pneumococcal septicaemia in an 8-year-old child after splenectomy for trauma

Sir,

It is usual to defer elective splenectomy in young children until after the age of 4 on the grounds that their defences against infection may as a result be impaired

up to school age. Even after this age some physicians prescribe long-term antibiotic prophylaxis because of the risk of infection, particularly pneumococcal, to which they are markedly susceptible. Eraklis *et al.* (1967) reviewed 467 splenectomized children mostly above this age and found no mortality from overwhelming infection among those children whose spleens had been removed after accidental injury. It may be thought from this experience that when the indication for splenectomy was trauma, antibiotic prophylaxis was not necessary. A recent case in our care suggests differently.

An 8-year-old boy who 10 months previously had splenectomy after an accident was admitted with a 4-hour history of pyrexia but otherwise was not manifestly ill. He was put on antibiotic pending results of blood culture. During the next few hours his condition deteriorated dramatically and he died 6 hours after admission. All 3 blood culture bottles yielded a growth of pneumococci. The post-mortem findings were essentially negative. No splenic tissue was found.

Having had this experience we have advised our surgical colleagues to keep such children on antibiotic prophylaxis. We do not know when this should be discontinued but arbitrarily suggest age 10 years.

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Monoparesis following CPAP

Sir,

May we report another example of monoparesis as a complication of CPAP with a head box. A male infant, birthweight 2190 g at 35 weeks' gestation, developed respiratory distress syndrome soon after birth. At age 34 hours he was placed in a Vickers head box with a maintained pressure of 10 cm. He remained in the box, gradually improving, until the 6th day. After his transfer to an incubator he became pyrexial within 24 hours and then gravely ill with staphylococcal septicaemia, including localized collections of pus in the skin over the skull, sternum, and sacrum. With antibiotics he slowly improved and as he was becoming more vigorous on the 12th day, abnormality of the left arm was noticed. There was complete paralysis of deltoid, a flicker of movement in biceps, and grasp reflex was normal. Before discharge on the 36th day some shoulder movement was noticed, and at follow-up the left arm was considered normal by 3 months of age.

At the time we considered that this Erb's palsy was probably due to pressure from the collar of the head box, but did consider the alternative suggestion that a localized collection of pus in relation to the plexus had been present; there was no clinical evidence of such a collection and x-rays of neck and shoulder were normal.

In view of the report of Turner, Evans, and Brown (1975), we now believe our case to be a third example.

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Calorie requirements for successful breast feeding

Sir,

Daily calorie intakes were assessed by 24-hour recall in 13 healthy women while breast feeding between 4 and 38 weeks post partum and in the subsequent non-lactating state. 9 mothers were lactating adequately, that is, no complementary feeds were given. 4 mothers had an inadequate milk supply, as complementary bottles had to be given at 2 or more feeds a day. All the babies were healthy. Their weights at the time of assessment were as follows: between the 3rd and 10th centiles 1, 10th-25th 4, 25th-75th 7, and 75th-90th 1. The younger babies, under 4 months, were receiving little or no solid food, and the major part of the older babies' fluid intake was breast milk.

The results are shown in the Table. Calorie intakes of the 9 successfully breast feeding mothers was considerably higher than that of the 4 mothers unsuccessfully breast feeding, and this was particularly striking in the mothers who were not losing weight. The intake of the unsuccessful mothers was only little more than in the nonlactating state, whereas the successful mothers were eating up to 50% more than normal. One unsuccessful mother, consuming 1950 kcal, increased her intake to 3910 kcal day. Her milk supply quickly increased and complementary feeds were no longer necessary. Furthermore, 3 successful mothers who tried to 'diet' to lose weight more rapidly, found

an immediate reduction in milk supply with subsequent irritability and failure to gain weight of their babies. On reverting to eating at will, the babies became satisfied and resumed their normal weight gain patterns.

Although both the Department of Health and Social Security (1969) and the United Nations Food and Agriculture Organization (1957) recommend extra calorie intakes of 500 and 1000 kcal respectively for lactating women, none of the mothers were aware of any extra requirements. Indeed many of the successful mothers reported 'feeling guilty' at the amount they were eating to satisfy their appetites. The unsuccessful mothers were either anxious about their babies and admitted to having lost their appetites, or felt they ought to diet.

The present findings are in agreement with those of Thomson, Hytten, and Billewicz (1970) who found that successfully lactating women, most of whom were losing weight, had an average daily intake of 690 kcal/day more than bottle feeding mothers.

Insufficient milk is a commonly reported cause for failure of breast feeding (Newson and Newson, 1965). The usual clinic advice to mothers is to drink more fluids to increase their supply, though this has been shown by Illingworth and Kilpatrick (1953) to be ineffective. Since the present preliminary results indicate that the calorie content of a mother's diet may be critical in her ability to produce an adequate milk supply, mothers should be informed of their extra calorie requirements, and advised to eat more if in doubt about their milk supply.

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TABLE

	Daily calorie intake		Increase above normal	
	Lactating (mean \pm SD)	Nonlactating (mean \pm SD)	Mean kcal	Paired 't' test
Successful mothers (no. = 9)	2886 \pm 419	2041 \pm 315	845	P < 0.001
Weight steady (no. = 5)	3030 \pm 525	2052 \pm 441	978	P < 0.001
Losing weight (no. = 4)	2705 \pm 150	2028 \pm 61	677	P < 0.005
Unsuccessful mothers (no. = 4)	1920 \pm 75	1783 \pm 458	137	NS