descriptions of vaccinia, and in the present case vaccination both in infancy and in adulthood was followed by acute diarrhoea. (According to the hospital notes relating to the episode in infancy no intestinal pathogen was isolated.) The symptom may have been a nonspecific intestinal reaction to a constitutional phase of the illness, but Gurvich *et al*[4] isolated vaccinia virus from the pharynx of 18 out of 80 children three to 20 days after vaccination, and it seems possible that the virus itself may sometimes reach the gastrointestinal tract. Accidental vaccinia may follow the transfer of infected material from the patient himself, or from another who has been recently vaccinated. In the present case transmission was probably by the fingers to a perianal region excoriated by diarrhoea. In the treatment of accidental vaccinia in infants, especially when there is evidence of a toxic reaction the administration of vaccinia immune globulin (VIG) has been recommended.[5]

I thank Dr Helen Mair for confirming the diagnosis, and Dr T M Pollock for helpful comments.

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Diet and diuretics in pregnancy and subsequent growth of offspring

Campbell and MacGillivray[1] studied the effect of weight reduction in the last ten weeks of pregnancy on the incidence of pre-eclampsia in primigravidae gaining more than 570 g a week between 20 and 30 weeks of gestation. One group received two tablets of Navidrex K (cyclopenthiazide 250 μg, potassium 600 mg) daily, and another a 5-MJ (1200 k-cal) diet. A third, untreated group acted as controls. The patients were matched for social class, cigarette smoking, age, height, and weight for height at 20 weeks of gestation. When pregnancies complicated by pre-eclampsia were excluded the birth weight of the children of the untreated mothers was found to be greater than that of the children of mothers in either of the treated groups.

The subsequent growth of 61 of the children is reported here.

Patients, methods, and results

The 61 mothers had been normotensive throughout pregnancy; 17 were in the diuretic group, 22 in the dieted group, and 22 in the untreated group. Because of the wide age range of the children (3·8-5·7 years) all measurements were plotted on the appropriate centile charts[2 3] (HCB 18, HCG 19; Creaseys Ltd) before the three groups were compared. All measurements were made by me. Children were weighed on a beam balance in their underclothes. Head circumference was measured with a tape measure, and height with the Harpenden Stadiometer. Each mother was sent a tape measure and instructions on how to record her husband's height. The height of each child was related to mean parental height on a centile chart[2] designed for this purpose.

Distribution of centiles among the 61 children

	Centile	Diuretic group (n = 17)	Dieted group (n = 22)	Untreated group (n = 22)
Weight	0–50	3	9·5	4
	>50	14	12·5*	18
Height	0–50	4	10	4
	>50	13	12	18
Head circumference	0–50	4·5	11·5	12
	>50	12·5*	10·5†	10

*One value on 50th centile.
†Three values on 50th centile.

The table gives the results. The children of mothers in the dieted group weighed less than those of mothers in either the untreated or diuretic group ($P < 0.05$). Similarly when height was adjusted for mean parental height the children of mothers in the dieted group were shorter than those of mothers in the other two groups ($P < 0.05$). Head circumferences of children in the diuretic group were greater than those in the dieted and untreated groups ($P < 0.05$). The distribution of weight and height of the children in the dieted group was similar to the standards,[2 3] while the children in the untreated and diuretic groups were significantly heavier than the standard ($P < 0.01$) and both groups were significantly taller than the standard ($P < 0.01$ and $P < 0.05$ respectively).

Comment

It is well known that a persisting size deficit occurs in prenatally undergrown humans and animals.[4] This study suggests that dietary restriction of women with excessive weight gain in pregnancy not only impairs fetal growth but results in a persistent size deficit in the height and weight of the offspring. Whether this effect is harmful or beneficial is uncertain, but it should be borne in mind when dietary restriction in pregnancy is recommended.

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Immunoreactive beta-melanocyte-stimulating hormone and melanin pigmentation in systemic sclerosis

Skin pigmentation is a common feature of systemic sclerosis and may resemble that seen in Addison's disease. Previously we showed that the increased plasma β-melanocyte-stimulating hormone concentrations in chronic renal failure (β-MSH) are due to impaired renal metabolism of the hormone,[1] and we wondered whether the decreased renal plasma flow that occurs in systemic sclerosis[2 3] might similarly impair renal metabolism of β-MSH and thereby explain the pigmentation in the disease.

Patients, methods, and results

Sixteen patients with systemic sclerosis were studied, of whom six showed moderate to severe pigmentation, and plasma β-MSH was measured with a modification of the method of Thody and Plummer.[4] Renal function had been studied several years earlier in 11 of these patients[3] and some impairment of renal blood flow, but not of glomerular filtration, had been found. No recent studies of renal function had been made but the blood urea concentration was normal in all.

The mean plasma β-MSH level in the patients with systemic sclerosis was 14·4 ± SE 2·9 ng/l, compared with 16·2 ± 1·2 ng/l in 55 normal controls.