melanotrophin, and also has a close neural relationship with adrenal medullary tissue. There also seems to be a subtle balance between the hypothalamic/pituitary system and the pineal gland. If one of these (opposed) endocrine systems suffers a deficiency or lesion, the other will show hyperfunction. This could explain the increased HVA and VMA values in the child. We saw a similar case, but in a young man, showing increased VMA values and also operated on for a suspected pheochromocytoma, which was negative (Visser and Axt, 1975). Through morphometric techniques we proved a new clinicopathological entity, 'adrenal medullary hyperplasia', causing overproduction of catecholamines. The increased HVA values in the infant discussed were always higher than the VMA values, suggesting a combined hyperfunction of both pineal gland and adrenal medulla caused by hypopituitarism.

In conclusion, we expect that in the infant under discussion there existed (a) a lesion of the adenohypophysis, contracted in the seventh month of pregnancy by pneumonia and possible hypoxia of the mother; (b) subsequent hyperpinealism responsible for the production of HVA and VMA combined with hypertension (probably intermittent) during life; (c) (relatively) increased weight of cerebrum and heart; and (d) disturbed cortico/medullary volume ratios in the adrenals.

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Dr. Kleinberg comments:

We are intrigued by Dr. Visser's suggestion that our child is in fact a 'typical SIDS child.' Such infants are usually considered to be normal, to be growing and developing normally, and are not believed to have life-threatening illness before their unexpected death. Our child presented a marked failure to thrive and his death was neither unexpected nor sudden. We recognize, of course, that many SIDS infants have disturbances of autonomic cardiovascular systems and/or sleep states, but these abnormalities have been generally uncovered in chance observations upon clinically normal infants who later succumb to SIDS. Lastly, the pathologist commented that adrenals and pineal were unremarkable. Though the weight of the pituitary was unavailable, the brain weight was 460 g.

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Treatment of severe Asian rickets with vitamin D-fortified chupatti flour

Sir,

Vitamin D deficiency, leading to severe rickets and osteomalacia, is common among Asians in Britain (Dunnigan *et al.*, 1962; Ford *et al.*, 1972, 1976). The fortification of chupatti flour with vitamin D has been shown to raise levels of serum 25-hydroxy-vitamin D and to reduce the incidence of biochemical rickets in those consuming it (Pietrek *et al.*, 1976). We report here the successful use of the flour in a case of severe, untreated Asian rickets.

A 16-year-old Asian youth presented with a history of gradual bending of both legs which had become increasingly painful over the previous 6 months. There was moderate bowing of both tibiae. Serum Ca was 7·6 mg/100 ml (1·9 mmol/l), phosphate 3·3 mg/100 ml (1·06 mmol/l), and alkaline phosphatase 65 King Armstrong units/100 ml. Serum 25-hydroxy-vitamin D was low at 3·5 ng/ml (Belsey *et al.*, 1974). Plasma proteins, urea, and electrolytes, D-xylose absorption, and faecal fats were normal. *X*-rays of wrists and knees showed the typical epiphyseal and metaphyseal changes of rickets. A jejunal biopsy was normal and an iliac crest bone biopsy showed bone trabeculae with thickened osteoid seams on their surfaces consistent with the diagnosis of osteomalacia (Dr. T. Anderson). He was considered to be a typical case of adolescent Asian rickets without evidence of renal disease or malabsorption.

The patient's family was supplied for 16 months with flour fortified with 6000 units vitamin D_2 per kilogram, prepared as described previously (Pietrek *et al.*, 1976). The patient consumed 4 chupatties (about 160 g flour) daily containing approximately 1000 units vitamin D_2; about half the vitamin appears to be destroyed in cooking (Pietrek *et al.*, 1976), providing him with an effective intake of about 500 units daily.

The biochemical response to the consumption of the flour is shown in the Fig. Serum Ca and alkaline phosphatase levels returned to normal after 7 months and serum 25-hydroxy-vitamin D levels rose to a peak of 22 ng/ml after 10 months and levelled out at about 18 ng/ml. Radiological healing was complete at the end of the 16-month period and a second bone biopsy at this time showed complete histological healing.

This case report confirms that vitamin D-fortified chupatti flour is effective in the treatment of Asian rickets. Biochemical, radiological, and histological healing showed that the vitamin D preparation used retains its biological