The target he sets is 35% of total energy without prejudicing growth or producing an unpleasant diet. Your readers might be interested in some preliminary findings of dietary intake at 18 months in a sample of 170 Hong Kong Chinese infants who are participating in a longitudinal study of growth and nutritional inter-relationships from birth to 5 years.

Fat contributed about 30% of total daily energy consumption at 18 months. Most infants were then eating a diet of rice, fish, meat, and vegetables. Butter and high fat desserts were rarely given and ice cream only occasionally consumed. Energy intake was 0.414 MJ/kg/day, very similar to energy intake in recent studies from Canadian² (0.401 MJ) and Australian³ children (0.418 MJ) of the same age. Our infants enjoyed excellent health and were growing and developing normally.

In Hong Kong even with its obvious western influences the traditional Chinese diet in early childhood is low in total fat and cholesterol and saturated fats. As children grow older, however, the situation is likely to change as ubiquitous 'fast foods' increase their stranglehold on children's diets. It remains to be seen whether the very low fat intake of the early years in any way influences health in the long term especially in relation to cardiovascular diseases.

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Educating medical students about death and dying

Sir,

I read with interest the article by Black *et al*, which points out the need to address the stressful feelings medical students and physicians have about dying and death.¹ The authors propose a course with objectives varying from identifying the dying patients' and family needs and their dealing with grief, to clearing away barriers set up by physicians surrounding death. A course of this type would no doubt improve a physician's ability to deal with death and dying, and would be helpful to their dying patients and their families. However, I feel the course proposed lacks

one more important objective: obtaining permission for a necropsy and feedback to the family about the necropsy findings.

The information gained from a necropsy, and properly presented to the family, may relieve the guilt that is a consequence of most deaths, provides assurance that the patient received proper medical care, and facilitates the grieving process.²⁻⁴ As Hill and Anderson point out, the necropsy not only gives the physician the benefit of learning from his or her own experience, but also enables the medical student to accept the fact of death as an inescapable part of caring for the sick.⁴

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Rise in urea concentration after arginine hydrochloride infusion

Sir,

In order to test the renal ability of children to acidify their urine, an adequate degree of metabolic acidosis is usually obtained either by the oral administration of ammonium chloride or by the infusion of arginine hydrochloride.¹ The intravenous infusion of arginine hydrochloride—which is used also to study growth hormone, glucagon, and insulin release^{2,3}—has been described as safe and free of immediate or delayed untoward effects. Transient renal dysfunction, hypophosphataemia, and hyperkalaemia have, however, been described after arginine administration to normal subjects.⁴

When reviewing arginine acidification tests performed in our clinic, we observed a significant progressive increase in plasma urea concentrations in children with normal glomerular filtration rate undergoing the test. In seven children aged 5 months to 6 years, the intravenous administration of arginine hydrochloride (100-150 mmol/m² over 120-150 minutes) increased mean (SD) plasma urea from 5.1 (0.5) to 7.3 (0.6) mmol/l at 60 minutes ($p < 0.001$)

and to 9.5 (0.8) mmol/l at 120 minutes ($p < 0.001$) after starting the infusion of arginine hydrochloride.

The observed increase in plasma urea is likely to be due to the cleavage of L-arginine to urea and ornithine catalysed in the liver by the enzyme L-arginase. This enzyme is present in the liver of all ureotelic organisms and, like most mammalian enzymes, only acts on the L-isomer. To test this hypothesis, we measured the changes in plasma urea concentrations occurring in eight anaesthetised and mechanically ventilated rabbits infused with either L-arginine or D-arginine at doses comparable with those used during an acidification test. After the infusion of L-arginine (0.05 mmol/kg/minute) in three animals, mean (SD) plasma urea rose from 6.4 (2.1) to 7.6 (2.1) mmol/l at 60 minutes ($p < 0.01$) and to 8.9 (2.2) mmol/l at 120 minutes ($p < 0.01$). By contrast, the infusion of the same dose of the D-isomer in three other rabbits did not significantly modify plasma urea (5.9 (0.2) before the infusion, 6.0 (0.1) at 60 minutes and 6.5 (0.1) mmol/l at 120 minutes). Plasma urea also remained stable in the two saline infused control rabbits.

Our clinical and experimental data thus suggest that cleavage of L-arginine to urea and ornithine by the liver L-arginase is probably responsible for the increase in plasma urea concentrations observed in children infused with

arginine hydrochloride. The lack of endogenous D-arginase explains the failure of urea to increase after the administration of D-arginine. While this rise of plasma urea does not have any deleterious effects in children with normal renal function, it may likely lead to misinterpretation of urea data collected during an arginine infusion test.

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