tioned on the grounds that the yield of active disease is low and the value of chemoprophylaxis unestablished. Probably the greatest value is from BCG vaccination of children. The incidence of tuberculosis in children of parents from the Indian subcontinent compared with that of white children is 50 times greater if born abroad and 20 times greater if born in Britain.[18] BCG vaccination should therefore be given at birth for those born in Britain and as soon as possible after arrival here for those born abroad.[19]

Screening by chest radiography before arrival in Britain is desirable, but as most disease becomes evident only after arrival in Britain, screening of high risk individuals would need to continue.

BCG vaccination

The DHSS currently recommends that BCG vaccination be offered routinely in schools to children aged 10-14 years throughout Britain, and this programme has been shown to be highly effective in preventing tuberculosis.[20] It is necessary for this policy to be maintained everywhere, including those regions that currently have a low prevalence of tuberculosis, as children living in these areas may subsequently go to work in regions where there is a much higher prevalence of tuberculosis, such as in London and the industrial cities of the Midlands, where the risk of infection is much greater than in the place of their schooling. Because of the mobility of the population BCG vaccination should not be abandoned until the risk of infection is low everywhere in Britain.

Before receiving BCG vaccination all children should receive tuberculin testing using the multiple puncture Heaf technique. Those with positive reactions (grades 2, 3 and 4) should be referred to the local chest clinic for examination and chest *x* ray. Those with grade 3 and 4 reactions merit follow up by chest *x* ray examination, and chemoprophylaxis may be given to Asian children. BCG vaccination is given to children who give negative or grade 1 reactions. The technique of BCG vaccination is described in a DHSS circular.[21] The technique of administering a truly intradermal injection by needle and syringe is one that needs training and practice, and new staff should be trained by experienced operators to avoid injections that are subdermal instead of intradermal, which may cause local abscess formation.

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Clinical curio: addiction to sherbet fountains

Although modern confectionery is said to damage both the teeth and the digestion, reports of metabolic disturbances have been confined to liquorice abuse. We describe here a case of severe metabolic alkalosis after eating large quantities of sodium bicarbonate and liquorice in the form of sherbet fountains or lemon kali.

A 25 year old woman presented with a week's history of giddiness, slurring of speech, headache, and profound muscular weakness. She admitted to the ingestion of no drugs other than oral contraceptives. On initial examination she was slow mentally with slurred speech but no confusion or disorientation. Her blood pressure was 97/75 mm Hg. She had mild right sided cerebellar ataxia, profound muscle weakness, but no other abnormalities. Viral encephalitis or demyelination was tentatively diagnosed, but on admission considerable biochemical abnormalities were found: serum sodium concentration was 125 mmol(mEq)/l, potassium 1·5 mmol(mEq)/l, chloride 58 mmol(mEq)/l, urea 3·8 mmol/l (29 mg/100 ml), calcium 2·46 mmol/l (9·8 mg/100 ml), albumin 43 g/l, and glucose 4·8 mmol/l (86·5 mg/100 ml). Serum osmolality was 255 mosmol/kg and urine osmolality was 392 mosmol/kg. The urinary pH was alkaline at 9, and a midstream urine specimen was normal. Arterial blood gas estimation showed pH 7·6 (hydrogen ion concentration of 24·8), a normal arterial oxygen value of 13·3 kPa (100 mm Hg), a carbon dioxide value of 7·03 kPa (52·7 mm Hg), and a bicarbonate concentration of 82·8 mmol(mEq)/l. Urinary electrolytes showed a sodium of 60 mmol/l, potassium of 47 mmol/l and a urea of 9 mmol/l (0·05 g/100 ml). A Synacthen test was normal. This pronounced metabolic alkalosis, for which initially no cause was apparent, reverted to normal after treatment with intravenous physiological saline for 24 hours. After direct questioning she admitted to being passionately fond of lemon sherbet, often eating up to eight Barrett's sherbet fountains a day as well as buying lemon kali powder. Indeed, for the first 48 hours of her hospital admission her father had been bringing her in half pound bags of lemon kali powder. After we explained the hazards all her biochemical measurements reverted to normal, and she returned to good health.

Metabolic alkalosis induced by amounts of sodium bicarbonate is unusual, and there was no evidence of excessive gastric loss of chloride due to vomiting,[1] deficient chloride intake,[1] or excess of renal retention of bicarbonate as shown by a urinary pH of 9.[2 3] There was no evidence of intrinsic renal disease, and she had not been taking diuretics.

Biochemical analysis of a random sample of Barrett's sherbet fountain showed a hollow stick of liquorice weighing 6 g and a yellow sweet effervescent powder weighing 22 g. We presume that it is a mixture of sucrose, sodium bicarbonate, and either citric or tartaric acid. A 1% solution of the powder was of pH 5·2 and contained 9 mmol/l of bicarbonate, 7 mmol/l of sodium, but no potassium. Its osmolality was 38 mosmol/kg. A variable but considerable bicarbonate load[2] under the influence of the aldosterone like effect of excess liquorice ingestion produced a kaliuresis and a consequent complex metabolic alkalosis with hypokalaemia probably explained her cerebellar dysfunction and muscle weakness.—G KAYE, senior house officer, and E R WILLIAMS, consultant physician, Coventry.

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