amiloride, and hydrochlorothiazide plus amiloride plus timolol on blood pressure and on plasma electrolyte concentrations, including magnesium. Each treatment was separated by a four week placebo washout period, and each dose regimen was taken for four weeks before measurements were made.

During hydrochlorothiazide treatment of 50 mg/day plasma magnesium concentration decreased by 0.07±0.03 mmol/l (0.17±0.07 mg/100 ml) (mean ± SE, p<0.01 by Student’s t test) from 0.85 mmol/l (2.1 mg/100 ml) (placebo mean value), and on 100 mg/day it decreased by 0.10±0.02 mmol/l (0.24±0.05 mg/100 ml) (p<0.01). There were no symptoms associated with these falls. No significant changes in plasma magnesium concentrations occurred during treatment regimens which contained amiloride.

The lower concentrations of plasma magnesium described by Dr Sheehan and Dr White may have been partly due to the selection criteria used in their study but may also have resulted from the fact that all their patients were receiving treatment for heart failure and were therefore receiving diuretics against a background of secondary hyperaldosteronism, which would increase urinary magnesium loss and deplete total body magnesium.

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Spina bifida and vitamins

SIR,—Professor Rodney Harris and Dr A P Read (4 December, p 1651) believe that multivitamin supplementation to prevent neural tube defects should be offered to all women at risk unless they have enrolled in the controlled trial. I wish to sound a note of caution after a recent case of congenital malformation.

In February 1981 a 29 year old primigravida gave birth to a boy weighing 1380 g with osteogenesis imperfecta gravis. He died three minutes later. There was a family history of malformation in that the mother’s uncle had had a cleft palate. The parents received genetic counselling. It was noticed that the father had a large hairy patch over his lower back although radiographs of the spine were probably within normal limits. In addition to the risk of recurrence of the same type of osteogenesis we believed there was some risk of spina bifida, and vitamin supplementation was recommended for the next pregnancy. The mother took Pregnacare Forte F one tablet three times a day for two months before the last menstrual period of her second pregnancy and for 10 weeks thereafter. In June 1982 she gave birth to a girl weighing 3180 g with a cleft of the posterior palate.

This mother received a large dose of vitamin A above her dietary intake. Hypervitaminosis A is known to cause cleft palate experimentally1 and although Smithells et al2 found normal serum vitamin A concentrations in two women after 12 months of continuous supplementation, this cannot be taken to exclude any risk. The possibility that the amount of vitamin A received by this mother caused the isolated posterior cleft palate in her baby, who may have been genetically predisposed, cannot be ignored.

In the Fylde of Lancashire there was a significant drop in the incidence of anencephalus among conceptions after 1967,3 which was sudden and therefore unlikely to be due to dietary change. It seemed that vitamin deficiency was not a major cause of neural tube defect, at least in this area. Until the results of the controlled trial of multivitamin supplementation are known perhaps mothers who are not in the trial should receive dietary advice or folic acid treatment as suggested by Laurence et al.4

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References

Diuresis or urinary alkalinisation for salicylate poisoning?

SIR,—I would like to make two points about the management of salicylate poisoning (13 November, p 1383). Firstly, the authors maintain that alkali alone raised the urinary pH to a level higher than that obtained in the forced diuresis groups. Significant hypokalaemia occurred in the diuresis groups, however, but not in the alkali group. Hypokalaemia would lead to the production of more acid urine and tend to lower urinary pH. In view of this I cannot see how the urinary pTs are directly comparable unless hypokalaemia is corrected first.

Secondly, the authors maintain that the production of bicarbonate required to produce a strongly alkaline urine varies considerably and the urine may remain acid even in the presence of alkaliuria.” If the urine remains acid in spite of an alkali infusion, then, in this clinical setting, it is most likely that potassium depletion has not been fully corrected.

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*We sent a copy of this letter to the authors, who reply below.—Ed, BMJ.

Breast prostheses and seat belts

SIR,—As from 31 January 1983 it will be compulsory for drivers and passengers in the front seats of cars to wear seat belts (2 October, p 987). We have had to bring to our attention a very useful aid by a patient who has had mastectomy. This is a small chip, the Klunk-Klip, which attaches to the automatic inertia seat belt to relieve tightness and tension. It does not interfere with the working of the belt, and provides improved comfort for patients who have had breast or chest wall surgery. We feel that this clip could be of considerable help to a number of patients. The Klunk-Klip is manufactured by Tonken Auto Products Ltd, Woodford Halse, Daventry, Northants NN11 6FZ.

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Haemolytic uraemic syndrome: therapeutic effect of plasma infusion

SIR,—I would like to make two comments on the paper by Dr R Misiani and others (6 November, p 1304) on the therapeutic effect of plasma infusion in the haemolytic uraemic syndrome.

Four out of the seven adults with this syndrome had blood pressure recordings in the malignant hypertensive range. As implied in the title, haemolytic uraemic syndrome is a syndrome of multiple pathologies, of which malignant hypertension is one cause. The control of hypertension is just as likely to produce the results they found in the absence of plasma infusion. Indeed, Dr Misiani and others point out that “two of the adult patients had renal biopsies showing pronounced arterial and arteriolar lesions”—features consistent with malignant hypertension.