Normal full-term infants require about 100 IU of vitamin D daily to prevent rickets and premature infants about 200 IU. The adult requirements may be so low as to be met by the amount in unfortified foods and exposure to sunlight.² A survey of American children (newborn to 17 years) showed that half ingest 400-800 IU daily; almost 10% consistently consume over 1000 IU daily.³

In contrast, in the past half-century magnesium intakes have fallen, whereas dietary contents of protein, fat, sugar, and calcium have risen.⁴ High intakes of these nutrients, and of vitamin D, increase magnesium requirements and increase susceptibility to magnesium deficit.^{5,6} Metabolic balance studies indicate that Occidental magnesium intakes are suboptimal.⁷ The cardiovascular and renal lesions of experimental magnesium deficiency and experimental hypervitaminosis D are similar, as are those of infantile hypercalcaemia, an outbreak of which in England was related to excessive vitamin D—2000-4000 IU daily.^{6,8} Increased magnesium intakes protect against cardiomyopathy produced by many agents, including excessive vitamin D.^{5,9}

It may be the combination of excesses of most nutrients, with the exception of magnesium, that contributes to the high incidence of ischaemic heart disease in the industrialized countries. Perhaps the amount of magnesium provided by hard water¹⁰ may be sufficient to correct a marginal deficit, thereby contributing to the lower death rates from ischaemic heart disease in hard-water than in soft-water areas.—I am, etc.,

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- 1 Clark, I., and Bassett, C. A., *Journal of Experimental Medicine*, 1962, 115, 147.
- 2 *Recommended Dietary Allowances*. Publication 1694, Food and Nutrition Board. National Academy of Sciences, Washington, D.C., 7th edn., 1968, p. 24.
- 3 Dale, A. E., and Lowenberg, M. E., *Journal of Pediatrics*, 1967, 70, 952.
- 4 Friend, B., *American Journal of Clinical Nutrition*, 1967, 20, 907.
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- 6 Seelig, M. S., and Bunce, G. E., *Magnesium in the Environment*, Proceedings of Symposium. Division of Agriculture, University of Georgia, Fort Valley, Georgia, 1972.
- 7 Seelig, M. S., *American Journal of Clinical Nutrition*, 1964, 14, 342.
- 8 Seelig, M. S., *Annals of the New York Academy of Sciences*, 1969, 147, 537.
- 9 Seelig, M. S., and Heggveit, H. A., *American Journal of Clinical Nutrition*, 1974, 27, 59.
- 10 Hankin, J. H., Margen, S., and Goldsmith, N. F., *Journal of the American Dietetic Association*, 1970, 56, 212.

Wandering Gall Bladders

SIR,—I noted with interest your leading article (26 July, p. 193) and would like to report a further variation of the "left-sided gall bladder."

A 34-year-old woman presented at the outpatient clinic with a three-month history of epigastric pain, nausea, and vomiting. On examination she was found to have a large, mobile, non-tender mass in the right loin. Intravenous pyelography was performed but the preliminary plain abdominal film (see figure) provided the basis for the diagnosis. The pyelogram suggested that the three opacities seen on the right side of the abdomen in the plain x-ray were renal calculi lying in a large non-functioning right kidney. The oval opacity lying to the left of the third lumbar vertebra

and containing multiple filling defects was shown on the pyelogram to lie anterior to a normally functioning left kidney. An oral cholecystogram showed concentration of the dye in the oval opacity and confirmed it to be a gall bladder containing limey bile and multiple radiotranslucent gall stones. An erect film of the abdomen showed the stomach bubble to lie on the left side of the abdomen and thus discounted the possibility of complete situs inversus.

At laparotomy a huge hydronephrotic right kidney was found to have rotated the liver through 180° around the axis of the falciform ligament, bringing the gall bladder into a left-sided position. Right nephrectomy and cholecystectomy were performed, the kidney containing three calculi and the gall bladder multiple calculi of the mixed type.

Cholelithiasis occurs in 7-15% of the population of the United Kingdom,¹ some 10% of these stones being radio-opaque. Limey bile is a rare finding during biliary tract radiology with an incidence quoted as low as only one case in 6700 gall bladder examinations.² Urinary tract calculi develop in 2-3% of the population of the Western world, of which 90% will be radio-opaque.³ The finding of a false left-sided gall bladder is a rare occurrence in itself, but for the diagnostic features detailed above to be present on a single plain abdominal radiograph seems extraordinary. Incidentally, no abnormality in the patient's calcium metabolism could be demonstrated.—I am, etc.,

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- 1 Heaton, K. W., *Clinics in Gastroenterology*, 1973, 2, 67.
- 2 Nolan, B., Ross, J. A., and Samuel, E., *British Journal of Surgery*, 1960, 48, 201.
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Wastage from Training in Radiology

SIR,—A survey has recently been completed into the numbers of doctors leaving radiology either during or immediately after training. The survey was carried out retrospectively by asking the consultants in charge of training departments in the United Kingdom and Eire for the numbers of doctors entering training in the years 1968-73 inclusive and the numbers known to have left radiology. The overall wastage rate was 11% with some regional variations (see table).

At the end of 1973 there were 653 consultants in radiology in England¹ and 39 in Wales,² the total of 692 being 6% of all consultants. At the same time there were 97 consultant radiologists in Scotland.³ In England and Wales the number of consultant

	No. Entering Training	No. Leaving Radiology
Scotland	97	12 (12%)
Wales	31	1 (3%)
Eire and N. Ireland	33	9 (27%)
England (excluding London)	303	27 (9%)
London	178	22 (12%)
Total	642	71 (11%)
Mean/year	107	12 (11%)

radiologists increased over the six years 1968-73 from 595⁴ to 692, this annual increase of 2.7% being a little less than the 3.4% annual increase for all consultants over this period. The number of senior registrars in England and Wales increased from 79 to 150 and those in other training grades from 162 to 195 in the same period,⁵ annual increases of 15% and 3.4% respectively. In the seven years up to 1974 an average of 18 consultant or senior registrar radiologists emigrated each year and this increased to 35 in 1974.⁶

Training programmes have to take into account the expected vacancies occurring from deaths, retirements, emigration, and expansion as well as wastage from training. There appears to be no previous estimate of this last factor.

I am most grateful to all those radiologists who completed questionnaires and to Mr. P. Powesland of the University Department of Psychology and Professor J. H. Middlemiss for their help.

—I am, etc.,

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- 1 *Department of Health and Social Security Annual Report*, 1973, p. 106. London, H.M.S.O., 1974.
- 2 *Health and Personal Social Service Statistics for Wales*, pp. 24-25. Cardiff, H.M.S.O., 1974.
- 3 *Health Bulletin*, 1974, 32, 198.
- 4 *Department of Health and Social Security Annual Report*, 1968, p. 174. London, H.M.S.O., 1969.
- 5 *Health and Personal Social Service Statistics for England*, 1973, p. 37. London, H.M.S.O., 1973.
- 6 Faculty (Royal College) of Radiologists Survey.

Treatment of Shoulder Subluxation in the Hemiplegic

SIR,—Your leading article on "Shoulder Pain from Subluxation in the Hemiplegic" (14 June, p. 581) rightly emphasized this common problem in hemiplegics. Until the present, treatment of such chronic painful shoulders has been inadequate. For patients who regain the motivation and ability to undertake household activities, indefinite application of a sling is annoying and may predispose to shoulder-hand syndrome.

Previously surgical treatment has been limited to shoulder fusion. In many cases such immobilization would further impair the limited function of a hemiplegic's upper extremity. A few models of total glenohumeral joint prostheses have been designed for resurfacing the degenerative articular surfaces in arthritic patients in whom the capsule of the joint is intact. These models have been contraindicated in the presence of a degenerative capsule or paralytic rotator cuff. Recently Fenlin¹ has described an operation for use in patients with degeneration of the rotator cuff which should be of great benefit to hemiplegics with chronic, painful, subluxing shoulders. It is a glenohumeral joint replacement with methyl methacrylate fixation wherein the "ball" is captive in the socket to provide mechanical

stability for the articulation. The unique design of the Fenlin prosthesis appears to satisfy all of the complex requirements of a rotator cuff replacement.

It will be recalled that the anatomical musculocutaneous cuff functions not only to stabilize the glenohumeral joint, while allowing it great freedom of motion, but also to fix the fulcrum against which the deltoid can contract and elevate the humerus with power.² The hemiplegic's paralysis of the rotator cuff provokes not only painful subluxation of the joint but also loss of the ability to fix the glenohumeral fulcrum. The deltoid, then, is unable to elevate the humerus and merely shrugs the shoulder. In the hemiplegic who gradually regains muscle strength in the upper extremity the elongated rotator cuff does not decrease to its normal size. Hence shoulder pain continues and is usually accompanied by weakness in elevation of the humerus.

In the Fenlin design a large ball is attached to the scapula, while the socket is secured to the humerus. The geometry of the large ball diminishes the magnitude of mechanical force necessary for the deltoid to initiate elevation. Also, the implant is designed with an inherently stable fulcrum so that the deltoid can elevate the humerus without assistance from the rotator cuff. The positions of the ball and socket are reversed from their normal anatomical location to diminish the potential for dislocation. The other novel feature of this implant is a robust scapular anchor that greatly reduces the likelihood of loosening of the component. Early clinical experience with the Fenlin prosthesis is encouraging. Further trials in hemiplegics appear to be indicated.—I am, etc.,

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¹ Fenlin, J. M. jun., *Orthopedic Clinics of North America*, 1975, **6**, 565.

² Inman, V. T., Saunders, J. B., and Abbott, L. C., *Journal of Bone and Joint Surgery*, 1944, **26**, 1.

Corticosteroids in Treatment of Anaphylaxis

SIR,—The excellent paper by Dr. P. Davies and others (24 May, p. 434) on the value and complications of intravenous pyelography raises an important point in suggesting that patients who are thought to be at risk for anaphylaxis due to possible iodine sensitivity should be given steroid cover. In the absence of a controlled clinical trial of the effect of steroids in acute anaphylaxis it would seem that the commonly held belief that they are the mainstay of the treatment of anaphylaxis is ill-founded. Prophylactic steroid treatment is unlikely to offer a significant degree of protection against acute anaphylaxis mediated by IgE in man. If there is any effect it is likely to be delayed, as in the rat, necessitating pretreatment with a single large dose 12-24 h ahead of the risky procedure. If the patient has type III hypersensitivity steroids could be expected to give complete protection, but this mechanism by itself is unlikely to produce the sudden dramatic emergency with which we are concerned. In patients with dual sensitivity the initial reaction will be no less dangerous, but subsequent management will be easier if steroids have been given.

Though glucocorticoids are unlikely to modify the anaphylactic reaction itself, a very high dose directly reduces capillary permeability and may help to sustain the blood pressure. Here one is seeking the effect which, for instance, is also useful in the treatment of cerebral oedema. For this role a water-soluble phosphate should be activated in the tissues more readily than the hemisuccinate of hydrocortisone, and, since they penetrate tissues much faster than other preparations, dexamethasone or betamethasone phosphates are the best choices. A reasonable dose of either would be 80 mg intravenously. An equivalent dose of hydrocortisone hemisuccinate would be 2.5 g.

Adrenaline, with its blend of α and β sympathetic effects, will help to antagonize the initial catastrophic loss of blood pressure while at the same time antagonizing the intracellular consequences of anaphylaxis. Whatever the risk to the heart, when faced with profound anaphylactic collapse most doctors give adrenaline intravenously, using 0.5 ml of 1/1000 solution, diluted if possible, given slowly. Their choice is almost certainly correct, since in profound shock a peripheral injection would never reach the rest of the body. Perhaps in the future intravenous salbutamol will be judged superior. For the second injection it is logical to give intravenous aminophylline, which potentiates the intracellular β sympathetic effect. Only the third injection should be steroid.—I am, etc.,

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Perforation of Small Intestine and Slow-K

SIR,—While not discounting the possibility that Slow-K may cause small-intestinal ulceration and perforation, I feel that this preparation should not be incriminated on the evidence presented by Dr. M. A. Farquharson-Roberts and his colleagues (26 July, p. 206) and that other aetiological factors should be considered—namely, cardiovascular disease.

Of the large number of causes of small-intestinal ulceration, cardiovascular disease (arteriosclerosis and hypertension) appears to be the commonest, probably producing the damage by localized gut ischaemia.^{1,4} Thus the long history of hypertension and multiple strokes in the 57-year-old patient reported may have been of greater significance than the administration of Slow-K.

I know of no specific histological features associated with iatrogenic ulceration, and histological differentiation of ulceration due to potassium chloride medication from ulceration due to other causes has been found impossible.³ Therefore further details of the "almost certainly iatrogenic" histological picture would be required before the conclusions reached could be accepted.—I am, etc.,

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¹ Teicher, I., et al., *Surgery, Gynecology and Obstetrics*, 1963, **116**, 196.

² Alexander, H. C., and Schwartz, G. F., *Gastroenterology*, 1966, **50**, 224.

³ Wayne, D. M., and Helwig, E. B., *American Journal of Clinical Pathology*, 1968, **49**, 26.

⁴ Carlson, H. C., *Radiologic Clinics of North America*, 1969, **7**, 97.

Pyridoxine and Oestrogen-induced Glucose Intolerance

SIR,—It has been suggested that women using contraceptives should take a pyridoxine supplement to prevent oestrogen-induced changes in tryptophan metabolism.¹ These changes include the production of increased amounts of an intermediate, xanthurenic acid, after a tryptophan load. This compound has been claimed to produce diabetes due to the formation of an inactive complex with insulin.²

The possibility that xanthurenic acid is responsible for oral contraceptive-induced glucose intolerance and gestational diabetes has been examined clinically. The reduction by pyridoxine of excessive xanthurenic acid excretion after a tryptophan load has been associated with an improvement in glucose tolerance.³⁻⁵ However, we do not believe that the two are causally related, as has been suggested, but rather that the beneficial effects of pyridoxine are possibly due to normalization of other tryptophan metabolites. Some women suffer from oestrogen-induced glucose intolerance independent of any abnormalities in xanthurenic acid excretion. Furthermore, we have found that the administration of xanthurenic acid (Sigma, Grade A, 99% pure) 120 mg/kg intraperitoneally in ammoniated saline failed to alter blood glucose levels in male Sprague Dawley or Wistar rats. We repeated the experiments using the special high fat diet which had been used in the original studies on the diabetogenic effects of xanthurenic acid.⁶ Once again xanthurenic acid was found to be inactive (see fig.) In further experiments it was found that xanthurenic acid did not impair glucose tolerance when given one hour before an oral glucose load (3 g/kg given as a 50% w/v solution). In a single experiment in a male albino rabbit xanthurenic acid 100 mg/kg given intravenously did not alter blood glucose concentrations over a period of three hours.

Effect of intraperitoneal injection of xanthurenic acid on blood glucose concentrations in male Wistar rats. Blood samples (0.1 ml) were obtained by heart puncture under pentobarbitone anaesthesia. Results reported by Kotake⁶ are also indicated.

Our failure to demonstrate any diabetogenic effect of xanthurenic acid could not be due to contamination with the 8-methyl ether of xanthurenic acid, which has been reported to be antidiabetogenic,⁷ since the sample of xanthurenic acid used was found