Vitamin D and Myocardial Infarction

SIR,—Professor V. Lindén (14 September 1974, p. 647) reports a study of vitamin D consumption in different Norwegian population groups, in particular persons who had qualified for a disability pension because of myocardial infarction, angina pectoris, or degenerative joint disease. From his results Professor Lindén concludes that a high intake of vitamin D is a risk factor in the occurrence of myocardial infarction and that 30 μg a day is the critical level. He found that the chief source of the vitamin D consumed was fish liver, which is an important item in the diet of the population providing the subjects for the study. The logic underlying the conclusions reached by Professor Lindén on the basis of his data is fundamentally unsound, for his findings permit of other possible explanations.

It is true that Professor Lindén has convincingly shown the stated vitamin D consumption to be greater in the myocardial infarction group than in the controls, but he has not shown that this higher intake is responsible for the higher incidence of the disease. On the contrary, it is conceivable that the higher incidence and the higher intake of vitamin D had a common cause—for example, dietary habits—possibly arising from a difference in the living standard of the groups.

As mentioned above, the excessive intake of vitamin D was chiefly due to the consumption of large amounts of fish liver. The observed relationship may thus very well be not between vitamin D and the disease but between fish liver and the disease, since fish liver contains more than just vitamin D—for example, heavy metals.

The completion of a questionnaire on intake of medicines and dietary habits may presumably yield different results—even though the intake may be the same—according to, for example, a difference in morbidity between the groups. This retrospective study thus has serious shortcomings. This is the more unfortunate because the results might be taken by the uninitiated as support for propaganda against vitamins.—We are, etc.,

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Xanthinuria as a Cause of Hypouricaemia in Liver Disease

SIR,—In 1967 we treated a patient with a xanthine stone in the bladder.[1] In a biochemical study of 30 members of the patient's family we found three more cases of xanthinuria. None presented clinical symptoms.[2]

In August 1974 we re-examined a brother of the proband. He had a history of alcoholism but his previous biochemical study had shown no xanthinuria. At that time his serum urate level was 0·26 mmol/l (4·4 mg/100 ml), serum oxypurines 1·8 mg/l, and urinary urate 2·3 mmol/24 h (392 mg/24 h). No urinary oxypurines were found on paper chromatography. After an episode of jaundice in 1971 he had progressively developed ascites, oedema, epistaxis, and hepatic encephalopathy. The clinical and biochemical picture was that of liver cirrhosis without haemochromatosis. He refused a liver biopsy. He also had persistent hypouricaemia (serum urate 0·049 mmol/l (0·84 mg/100 ml)) and hypouricosuria (urinary urate 0·3 mmol/24 h (50·5 mg/24 h)). His serum oxypurines (xanthine plus hypoxanthine) were 5·6 mg/l. Xanthine oxidase was not found in the blood cells. The urinary excretion of purines was xanthine 368·7 mg/24 h and hypoxanthine 67·7 mg/24 h.

Hypouricaemia in liver diseases has been attributed to an increase in renal uric acid clearance.[3,4] In our case the hypouricaemia was associated with xanthinuria, suggesting that liver insufficiency may unmask a partial defect of xanthine oxidase in people genetically conditioned even though they have not previously shown any overt biochemical abnormality. Therefore in cases of hypouricaemia associated with liver disease xanthinuria should be looked for both in the patient and among members of the patient's family.—We are, etc.,

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A Health District Courier Service

SIR,—Mr. I. J. Y. Cook (10 May, p. 335) raises a subject that has wider applications. With rising postal charges the expenditure on postage by family practitioner committees and the 10 000 general practice units that communicate with one another each week will certainly be between £500 000 and £1m this year.

One way of cutting down these costs drastically is to use local hospitals as reception stations for packets to and from local general practitioners and the F.P.C.s. Each area health authority already has its own courier services between its various units, and the F.P.C. is now part of the authority.

We have used a local hospital in this way for some time and the speed of deliveries has been improved.—I am, etc.,

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Tolamolol in Treatment of Angina Pectoris

SIR,—In their account of a double-blind comparative trial of tolamolol, propranolol, practolol, and placebo in the treatment of angina pectoris Dr. Graham Jackson and his colleagues (29 March, p. 708), report that four patients reacted adversely when treated with tolamolol, one developing a severe bradycardia, two sustaining a cardiac infarct, and one developing crescendo angina. Three of these patients subsequently died.

The authors quote the expected yearly mortality rate to be about 4% and argue that as the trial "lasted 10 months with each patient" they believe that these deaths represent the natural history of angina pectoris. However, they later state that "the total duration of the trial was 10 months" and it would appear from the protocol that each patient was followed for only six months and that during that time he was on tolamolol for only eight weeks. Furthermore, only 47 patients entered the trial. This would give an annual incidence of severe adverse reaction on tolamolol of 55·3% and a related annual mortality of 41·5%, which is markedly in excess of the expected yearly mortality rate for angina pectoris.

Surely, in a trial of a new drug, patients developing possible adverse reactions should not be excluded from the analysis, particularly if the possible adverse reaction subsequently leads to death. From the information contained in the article it is my opinion that the conclusion reached, for reasons already given, is very misleading.—I am, etc.,

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Radioactive Bromide Partition Test and Mumps Meningoencephalitis

SIR,—The radioactive bromide partition test is a valuable additional aid in the differential diagnosis of lymphocytic meningitis, particularly in the early diagnosis of tuberculous meningitis when the bacillus is not demonstrable in the cerebrospinal fluid and sugar levels are equivocal. The critical serum:C.S.F. ratio is 1·6. In tuberculous meningitis the ratio falls below this level and is a strong indication for starting specific treatment at once. In non-tuberculous lymphocytic meningitis higher values between 2 and 3 are found. Mandal *et al.*[1] found the test 100% accurate in four tuberculous and 15 non-tuberculous patients.

Early this year a boy aged 7 years was admitted to hospital with signs of meningitis. Relevant features in the history were a recent attack of chickenpox, a recent addition to the household of a puppy currently sick and under treatment by a veterinary surgeon, and the occurrence of mumps in the boy's school. High fever continued for seven days. There was a continuing pleocytosis (90% lymphocytes) in the C.S.F for 15 days. Though C.S.F. protein and sugar were within normal limits our experience is that a normal sugar content does not exclude tuberculous meningitis.[2] *X*-ray examination of the chest and the Mantoux test were negative. Laboratory tests excluded leptospirosis, toxoplasmosis, and infectious mononucleosis. Eight days after admission the bromide partition test showed a ratio of 1·4—that is, within the range of tuberculous meningitis. Electroencephalography showed focal disturbances in the left mid-temporal zone reported as not incompatible with a meningitic process but unlikely to be associated with a space-occupying lesion. Tubercle bacilli were not seen in the C.S.F., there was no evidence of tuberculosis elsewhere, nor was there a family history of tuberculosis. Furthermore, there was the history of contact with mumps. Mumps meningitis may be distinguished from other forms of viral meningitis by prolonged fever (in this case 12 days) and higher cell counts in the C.S.F. For these reasons antituberculosis drugs were not prescribed in spite of the results of the bromide partition test. Subsequent results of paired sera showed a rise in antibody titre (5/512) to mumps S virus, indicating a recent infection. No tubercle bacilli were grown on culture nor were any viruses isolated in rhesus monkey kidney tissue. A final E.E.G. showed a normal record. The patient made an uneventful recovery.

One case of mumps meningoencephalitis does not invalidate the bromide partition test. It is a reminder that the results of laboratory aids must not be considered in isolation from clinical and pathological observations when a decision has to be taken

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