

and recently Holmberg *et al* have reported developments which have improved the specificity of the tests.³ We tested our patient's serum by counterimmunoelectrophoresis using a pooled cytoplasmic extract from strains of *A israelii* kindly given to us by Dr Holmberg. Serological tests might be helpful for diagnosis as specimens are not easily collected for culture or histological examination in pelvic infections. The presence of a slow-growing anaerobe could easily be missed if there were a mixed growth of organisms on culture, and short courses of antibiotics might prevent cultural diagnosis. Serological testing of patients with long-standing pelvic inflammatory disease, particularly when associated with an IUCD, could be useful for detecting actinomycosis which might otherwise be missed. Circulating immune complexes were demonstrated in our case; we suggest that the disappearance of such complexes may be a useful marker for adequacy of treatment.

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- ¹ E Houang, personal communication.
² Communicable Disease Surveillance Centre of the Public Health Laboratory Service, *Communicable Disease Report*, 1978, No 5.
³ Holmberg, K, Nord, C E, and Wadstrom, T, *Infection and Immunity*, 1975, 12, 387 and 398.

Is pancreatic isotope scanning worth while?

SIR,—The short report by Dr Peter Cotton and his associates on isotope scanning of the pancreas (4 February, p 282) prompts us to record our recent results with this technique and explore some of the possible reasons why our experience differs.

We have previously published a study in 102 patients examined with a rectilinear scanner.¹ We now report the results in 200 patients examined by General Electric Radicamera with a Med II system. Scans were interpreted by three observers with experience of the technique but without knowledge of the precise clinical situation. The results are shown in the table below.

With experience and superior equipment the incidence of technically unsatisfactory scans is now negligible. However, compared with our previous study the false-positive rate is higher—36% compared with 30%—a trend which reflects an acknowledged bias towards reporting as abnormal a scan which is not unequivocally normal. Such a policy is of practical value when, as in this department, isotope scanning is used primarily as a screening test to select those patients who

require more detailed investigation for, in our experience and as others report, a normal scan virtually excludes chronic pancreatitis and cancer.¹⁻³

The high rate of "equivocal" reports, 20%, reported by Dr Cotton and his colleagues, may partly be conditioned by the apparatus used and influences the results in both normal and disease groups. Though we have no experience with the Elscint whole-body scanner, we abandoned the use of the Nuclear Enterprises gamma camera for pancreatic work after a few months' use in favour of the old rectilinear scanner, which provided more satisfactory imaging. We find it difficult to account for their low number of abnormal scans in patients with chronic pancreatitis and cancer (59%). Even if the equivocal reports are counted as abnormal the percentage rises only to 79. However, the results in relapsing pancreatitis are explicable since in relapsing acute pancreatitis the timing of the scan is all-important; total recovery of function can be anticipated within 6-8 weeks of the acute relapse. Dr Cotton and his colleagues do not distinguish between relapsing chronic and relapsing acute pancreatitis. The disadvantage of isotope scanning other than the high false-positive rate in subjects with a normal pancreas is that the scan cannot be repeated to follow functional progress in the individual patient within 4-6 months.

Finally, in our experience ultrasonography is not an ideal primary screening test since it depends even more than isotope scanning on the individual operator's technical expertise. In agreement with Doust and Pearce⁵ we have observed that in some 50% of patients with quiescent chronic pancreatitis the ultrasonic scan is normal. In these isotope scanning is abnormal in 96% of cases.

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- ¹ Braganza, J M, *et al*, *Gut*, 1973, 14, 383.
² Mitchell, C J, *et al*, *British Medical Journal*, 1976, 2, 1307.
³ Agnew, J E, Maze, M, and Mitchell, C J, *British Journal of Radiology*, 1976, 49, 979.
⁴ Russell, J G B, *et al*, *Gut*, 1977, 18, A418.
⁵ Doust, B, and Pearce, J, *Radiology*, 1976, 120, 653.

Coronary heart disease, age, and sex

SIR,—Drs R F Heller and H S Jacobs (25 February, p 472) suggest that testosterone may be responsible for the extra risk of coronary heart disease (CHD) that men before the age of 50 years encounter as compared with women of the same age; but "the mechanism of any risk it might produce is not clear."

More than 20 years ago I¹ discussed the possible relation of a relative deficiency of essential fatty acids (EFA) to atherosclerosis

and CHD and pointed out that as "the requirement of male animals for EFA is at least five times that of females," if we argued from lower animals to man "deficiency would be likely to be at least five times commoner in males than in females." The ratio of the male to female mortality rates for CHD (ICD 410-414) in England and Wales in 1973 was 6.1 at age 35-44 years and 5.5 at age 45 to 54 years. If the animal is castrated the requirement of the male is the same as that of the female.

Since prostaglandins are formed in the body only from EFA and since human males excrete roughly five times as much prostaglandin metabolites as do females² it might be supposed that the increased male requirement of EFA is caused by increased synthesis of prostaglandins. But this synthesis accounts for only a very small proportion of the daily requirement of EFA. For a reason that is obscure male animals become much more depleted than do females on a diet low in EFA.³

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- ¹ Sinclair, H M, *Lancet*, 1956, 1, 381.
² Samuelsson, B, *et al*, *Annals of the New York Academy of Sciences*, 1971, 180, 138.
³ Aftergood, L, and Alfin-Slater, R B, *Journal of Lipid Research*, 1965, 6, 287.

Diet and heart: the importance of metals

SIR,—In their very interesting prospective study on diet and the heart (19 November, p 1307) Professor J N Morris and his colleagues mention the possibility that their results might be accounted for by deficiencies in the diet of those with low energy intake. There may well be a reduced intake of mineral salts in those with low energy diets and there is now evidence that sudden death from ischaemic heart disease is associated with decreased concentrations of some metals in the heart muscle.

Decreased concentrations of magnesium in the hearts of those dying suddenly from ischaemic heart disease have been reported in several studies,¹⁻³ and Seelig⁴ has estimated that Western diets provide insufficient magnesium to maintain magnesium balance in men. There is a significant decrease in the concentration of magnesium in the heart muscle of normal men compared with normal women,⁵ which corresponds with the increased incidence of ischaemic heart disease in men.

In addition there are decreased concentrations of potassium and iron⁶ and an increased concentration of calcium^{3,6} in the heart muscle of subjects dying suddenly from ischaemic heart disease. The increase in calcium, as suggested by Anderson *et al*,³ is probably secondary to the decreased magnesium concentration. These results therefore suggest that those dying suddenly from ischaemic heart disease are relatively deficient in magnesium, potassium, and iron. Deficiencies of these same metals have been identified as contributing to the clinical problems in kwashiorkor and we have suggested that "empty calorie malnutrition"—diets adequate in calories but low in essential metals—may affect the heart both in kwashiorkor and in ischaemic heart disease.⁶

Published figures show⁷ that brown bread contains 30% more iron, twice as much potassium, and three times as much magnesium as white bread. The group

Scan reports on 200 patients

	No of patients	Scan report	
		Normal	Abnormal
Pancreas normal	137	88 (64%)	49 (36%)
Acute pancreatitis (and relapsing acute pancreatitis)	15	3	12
Chronic pancreatitis (and relapsing chronic pancreatitis)	25	1	24
Cancer of pancreas	23	1	22
	48	2 (4%)	46 (96%)