Renal masses and ultrasound

SIR,—While I agree with Dr Thomas Sherwood (20 December, p 682) on the value and accuracy of ultrasound in the "diagnostic pathway for patients with a renal mass," several observations should be made.

(1) Fluoroscopy is an unnecessary further investigation before the puncture of cystic lesions already identified by ultrasound.[1] Diagnostic puncture may be made at the time of the ultrasound study under sonar control. Since January 1974, 55 wholly transonic lesions of the kidney have been punctured in Brighton by this technique. No false-positive or false-negative punctures were made but in two cases greatly dilated hydronephrotic moieties of duplex kidneys were entered. These had been falsely interpreted as simple cysts at both urography and ultrasound study. The remaining lesions were shown to be simple cysts. Of 19 echogenic space-occupying lesions of the kidney, all showed pathological circulation at arteriography and all were interpreted as carcinomata by both techniques. At operation 18 were found to be renal carcinomata and one to be a hamartoma. No tumour was punctured. Before cyst puncture it has been our practice to perform a hydatid complement fixation test whenever possible. As a theoretical consideration it could be argued that hydatid complement fixation testing should be mandatory before the elective puncture of all renal cysts.

(2) It is essential to demonstrate that the whole of a space-occupying lesion identified at urography can be accounted for at the time of sonar or fluoroscopic puncture. Where there is doubt it is essential to proceed to selective renal angiography.

(3) Ultrasound provides adequate renal identification for the purpose of elective biopsy.[2] As such it affords a higher return of successful "first attempt" biopsies than "blind" biopsy. It obviates repeat urography and fluoroscopic biopsy where this might have been the earlier practice.

(4) Antegrade pyelography may be effected under sonar control, the method of examination being as for renal cyst puncture.

(5) The overall accuracy of renal tumour identification has been shown to be greater than that reported by Dr Sherwood. Barnett and Morley[3] identified 11 of 12 renal neoplasms, and in an objective "blind" review of sonar scans one radiologist correctly identified four out of five tumours.[2]

As Dr Sherwood has illustrated so ably, the ability to distinguish between solid and cystic space-occupying lesions of the kidney allows further investigation to be elective rather than blind.

RICHARD BURWOOD

X-ray Department,
Royal Sussex County Hospital,
Brighton

[1] Mountford, R A, *et al*, *British Journal of Radiology*, 1971, **44**, 860.
[2] Berlyne, G M, *Lancet*, 1961, **2**, 750.
[3] Barnett, E, and Morley, P, *British Journal of Radiology*, 1971, **44**, 733.

Prevalence of gall stones in Dundee, 1974-5

SIR,—Following our observation (22 November, p 427) that there were short-term fluctuations in the prevalence rate of gall stones in Dundee from 1953 to 1973 it was predicted that in the subsequent period a decline in prevalence rate would occur after an apparent zenith in 1971. To verify this hypothesis the post-mortem records of the Dundee centralised pathology department at Ninewells Hospital were studied from its inception in March 1974 to the end of September 1975. By chance in this period identical numbers of necropsies were performed on men and women (390 each).

Yearly prevalence of gall-bladder disease in Dundee during 1963-75 expressed as ASSMR. Overall prevalence for 1953-73 = 1·00

	1963	64	65	66	67	68	69	70	71	72	73	74-5
Men	0·76	0·73	0·85	0·88	0·98	1·06	1·32	1·13	1·89	1·71	1·55	1·25
Women	0·66	0·98	0·74	0·94	0·94	1·08	1·26	1·13	1·59	1·42	1·50	1·02

The figures were too small to derive age-specific prevalence rates, but the overall prevalence of gall stones + cholecystectomies in this period were 58 (15·8%) for men and 102 (26·1%) for women. The predicted figures from the standardised rates for 1953-73 were 46·21 and 99·68 respectively, giving age-sex specific morbidity ratios (ASSMRs) of 1·25 and 1·02 respectively. This represented a fall in prevalence rate since 1971 and reinforced the conclusion that there has been no relentless increase in the amount of gall-bladder disease in the twentieth century.

M C BATESON

University Department of Medicine,
Ninewells Hospital,
Dundee

Failure to confirm anticonvulsant hypomagnesaemia

SIR,—Recently Christiansen and his colleagues[1] reported that in a study of 226 epileptic patients chronically treated with various combinations of phenytoin, phenobarbitone, and primidone there was a statistically significant lowering of serum magnesium concentrations. We have attempted a similar study in a group of 100 seizure patients receiving various combinations of dilantin and phenobarbitone who were studied as a part of a larger study.[2] In our investigation we used a matched paired design which involved seizure-treated and institutionalised patients from Norristown State Hospital, Norristown, Pennsylvania, who were matched and paired according to age, sex, race, building residence, and duration of hospital stay. Serum magnesium was measured with an atomic absorption spectrophotometer (Perkin-Elmer model 403) with an 0·75% EDTA solution as diluent.[3] The observed serum magnesium range for this laboratory is 0·74-0·90 mmol/l (1·8-2·2 mg 100/ml) in normal subjects and the coefficient of variation is 0·6% on duplicate measures of serum magnesium. Analysis of the data consisted of a comparison of the paired means using Student's *t* test and of the variances using the F ratio.

As can be seen in the table, we did not find significant hypomagnesaemia in these patients. However, we did detect a statistically significant increase ($P<0·01$) in the variance within the seizure population receiving anticonvulsant therapy. We conclude that, while there is some important effect of this therapy on magnesium metabolism, we were unable to demonstrate under the rigorous conditions of a matched paired design any significant decrease in magnesium concentration. It is possible that the differences between our findings and those of Christiansen *et al* are related in some combination to (1) their use of an unmatched normal outpatient control population and/or (2) the considerably less likely possibility of significant differences in the effects on magnesium metabolism of primidone, which was used for seizure treatment in only a few of the patients in the population we studied.

These results stand in contrast to a pronounced effect of this anticonvulsant therapy on serum calcium levels in the same patient population. Moreover, we concur with the findings of Christiansen *et al* that there were no significant correlations between serum calcium and magnesium levels in either the seizure-treated or the control population. Nevertheless, the findings do raise important questions about variations in magnesium metabolism in institutionalised patients receiving this form of therapy. More specifically, the findings of Anast *et al*[4] that magnesium is involved in parathyroid function suggests that the well-described abnormalities of calcium and vitamin D metabolism in seizure patients undergoing anticonvulsant therapy may also involve magnesium metabolism in some as yet unknown manner.

SOLOMON H KATZ
IRA GERSTMAN
HARRY W LAUTENBACHER
MARY L HEDIGER

Division of Psychoendocrinology,
Eastern Pennsylvania Psychiatric Institute,
W M Krogman Center, and
Department of Anthropology,
University of Pennsylvania,
Philadelphia, Pennsylvania

[1] Christiansen, C, Nielsen, S P, and Rodbro, P, *British Medical Journal*, 1974, **1**, 198.
[2] Katz, S H, *et al*, unpublished observations.
[3] Willis, J B, *Nature*, 1960, **186**, 249.
[4] Anast, C S, *et al*, *Science*, 1972, **177**, 606.

	No	Serum magnesium (mmol/l)		Variance	*t*	F ratio
		Mean	SD			
Seizure patients	100	0·82	0·09	0·0203	0·24	1·66*
Matched controls	100	0·82	0·07	0·0122		

Conversion: SI to traditional units—magnesium: 1 mmol/l ≈ 2·4 mg/100 ml.
*Significant at $P<0·01$.

A place to be born

SIR,—In an age when we are constantly being reminded to separate fact from dogma, to test the significance or adequacy of the observed