	Patients		Controls (n = 22)
	Toxic (n = 9)	Non-toxic (n = 71)	
Plasma digoxin (nmol/l)	2.91 ± 0.90	1.42 ± 0.82	—
Plasma magnesium (mmol/l)	0.885 ± 0.11	0.83 ± 0.11	0.855 ± 0.06
Erythrocyte magnesium (mmol/l)	2.215 ± 0.345	2.32 ± 0.365	2.085 ± 0.225

Conversion: SI to Traditional Units—Plasma digoxin: 1 nmol/l ≈ 0.78 ng/ml.
Plasma and erythrocyte magnesium: 1 mmol/l ≈ 2 mEq/l.

were higher in both patient groups than in the controls but were significantly higher only in the non-toxic group ($P < 0.01$). There was no significant difference in mean erythrocyte magnesium levels between the two patient groups.

Diuretic therapy was documented in 33 of the patients; the mean plasma and erythrocyte magnesium levels in this group were 0.8 ± 0.07 and 2.275 ± 0.27 mmol/l (1.6 ± 0.14 and 4.55 ± 0.54 mEq/l) respectively. Both measurements were significantly different from the control values, the plasma level being lower and the red cell level higher ($P < 0.005$).

Our results suggest that though diuretic therapy does cause a lowering of mean plasma magnesium levels (by 6.4%) the reduction is only slight. This does not necessarily indicate an insignificant depletion of tissue stores, it merely reflects the inadequacy of sampling the plasma compartment. Erythrocyte measurements of magnesium, despite an approximately 3:1 concentration ratio compared with plasma, are not appropriate for the detection of magnesium depletion since digoxin therapy appeared to produce an increase in mean concentration of the ion, possibly by alterations in Na^+/K^+ ATPase activity in red cell membranes.

The absence of any correlation between plasma magnesium concentration and the incidence of digoxin toxicity suggest that the routine measurement of this ion is of little assistance in the detection of digoxin toxicity, the plasma digoxin assay being of much greater value. However, plasma magnesium should be one of the measurements made in cases of equivocal digoxin toxicity complicated by an apparently normal plasma digoxin level. It has even been demonstrated recently² that digitalis-induced ventricular arrhythmias can be abolished by magnesium chloride treatment in the presence of normal plasma magnesium levels.—We are, etc.,

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- 1 Archer, W. H., Emerson, R. L., and Reusch, C. S., *Clinical Biochemistry*, 1972, 5, 159.
- 2 Ghani, M. F., and Smith, J. R., *American Heart Journal*, 1974, 88, 621.

Dementia Associated with Clonidine Therapy

SIR,—We have noted recently an interesting side effect of clonidine in one of our patients.

A 74-year-old man being treated satisfactorily for hypertension with debrisoquine 80 mg a day, bendrofluthiazide 2.5 mg daily, and clonidine 0.1 mg three times a day, with a past history of depression on methyldopa, developed dementia approximately five months after starting clonidine. This was manifest by loss of memory, unsteadiness

of gait, drowsiness, aggressive behaviour with a change in personality, and urinary incontinence.

He was admitted to hospital for investigation. Routine investigations and an E.E.G. were normal. Approximately one week after clonidine was withdrawn he showed marked improvement. His blood pressure was then controlled on a small dose of debrisoquine, methyldopa, and a diuretic. He was seen two months later in the outpatient department and had made a complete recovery and was back to his usual self.

So far as we know, transient dementia has not before been reported with clonidine.—We are, etc.

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Teaching and the Patient

SIR,—The practical training of medical students inevitably involves some invasion of patients' privacy. The Patients Association is anxious that patients should suffer as little mental distress and physical discomfort as possible and we think that improvements in medical teaching methods could and should be made with this objective in mind.

We believe that the direct involvement of patients should be reduced to the minimum compatible with good teaching by the use of all possible aids and devices, and that when it is necessary for students to see patients ideally this should be on a one-to-one basis. We should be grateful to receive comments from any of your readers who have views on how these objectives could best be achieved.

Following discussions with the Department of Health and Social Security we think it might be possible to obtain a grant for research into this subject and we should be glad to hear from anyone who would be interested in carrying out or co-operating in such a project. The aim would be to investigate and demonstrate the extent to which the ideal can be achieved in practice.—I am, etc.,

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Aetiology of Chronic Renal Failure and Renal Osteodystrophy

SIR,—Dr. J. F. Moorehead and his colleagues (7 December, p. 557) suggested that patients with polycystic disease were less likely to develop erosions of bone than other patients with chronic renal failure during treatment with intermittent haemodialysis. This conclusion is at variance with a previous report¹ and with the findings from our own renal unit,² which suggested that poly-

cystic renal disease was not associated with a lower incidence of renal osteodystrophy.

It is not clear why the patients reported by Dr. Moorehead and his colleagues had such a low overall incidence of erosions when compared to that found in other centres. It seems probable, though details are not supplied, that the overall incidence of erosions of bone was half that found in our own population of dialysis patients in the Oxford area, which is nevertheless lower than that found in many other centres.³ Our own findings² suggested that patients with interstitial nephropathy may have a greater incidence of osteitis fibrosa. While their report suggested that the incidence of erosions was low in the polycystic group of patients, they did not compare these figures with those from the remaining patients when subdivided according to the aetiology of their renal disease. The manner in which this factor biased their control group of patients is therefore unclear.

Their conclusion that erosive bone disease was not progressive in these patients may also be unjustified on the evidence presented. A cohort of 53 patients were studied to demonstrate that the natural history of renal osteodystrophy on dialysis was not biased by a diminishing population of patients with time. However, this tactic was apparently not carried out on the polycystic patients. A further source of bias could be that their patients with polycystic disease were studied for a shorter period of time than their control patients. In the control population a marked increase of erosions occurred principally after the sixth year of dialysis. None of the patients with polycystic disease was studied for a comparable length of time. Furthermore, the finding of differing incidences of erosions with time could also result from changes in their dialysate calcium.⁴ Thus more control patients (treated for longer periods) would have been exposed longer to lower concentrations of calcium in the dialysate fluid.

We therefore feel unable to support the view that polycystic disease protects against the development of erosions.—We are, etc.,

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- 1 Pendras, J. P., *Archives of Internal Medicine*, 1969, 124, 312.
- 2 Henderson, R. G., Ledingham, J. G. G., and Woods, C. G., *Kidney International*, 1974, 6, 62.
- 3 Henderson, R. G., et al., *Proceedings of the Royal Society of Medicine*, 1974, 67, 38.
- 4 Tarter, G. L. V., et al., *British Medical Journal*, 1973, 4, 315.

Abortion Service

SIR,—Mr. D. B. Paintin and Professor R. W. Beard in their review of the remarkable tape recordings in *Babies for Burning* (8 February, p. 340) use the phrase "In concentrating on the 'sanctity' of the life of the fetus they [the authors] disregard the well-documented evidence that the unwanted baby and her mother may be condemned to a way of life that we regard as unacceptable these days." Now, dear reviewers, give us the evidence for this thesis.—I am, etc.,

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