the treatment of gastric ulceration in Chinese subjects.

The purpose of my letter, however, was not to discuss the merits (or otherwise) of carbamoxozone but to share with readers some recent developments in the area of drugs in the treatment of gastric ulceration.

—I am, etc.,
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Wartime Penicillin

SIR,—The Personal View by Dr. Terry Hamblin (10 August, p. 407) states that he is reminded “of the wartime doctor forced to watch children dying of pneumonia, while the penicillin to save them went to cure the troops’ gonorrhea.” Such a reminiscence is wholly imaginary (in any case the author was rather young at the time) and should not be allowed to pass unchallenged. As a wartime consulting surgeon to the Central Mediterranean Forces I know that in the Italian campaign at least the use of both calcium and sodium penicillin was carefully controlled, as both, but particularly the latter, were in short supply. They were used exclusively for the wounded.—I am, etc.,

HAROLD EDWARDS
Baron, Cambridge

Coroners and the Public Interest

SIR,—It does indeed seem hardly right that in the recent dental fatality you refer to (10 August, p. 374) the B.M.J.’s request for the depositions should have been turned down by the Westminster coroner. As a private individual I have seldom had such requests ignored or refused and have usually been sent a complete photocopy of the proceedings free of charge. But I have always made it clear that my purpose in requesting the information was to elucidate the causes of collapse in these cases with a view to preventing future tragedies.

It should, however, be realized that inquest proceedings in these dental fatalities are of little diagnostic value unless supplemented through direct personal communication with those in the surgery when the collapse occurred, as has been previously pointed out both by myself (3 February 1973, p. 293) and by Dr. M. P. Coplans and Mr. I. Curson (13 January 1973, p. 109). Too much credence also tends to be placed on the views of the expert witness. Whatever value it may have for the coroner a single unchallenged medical opinion, and one that in a coroner’s court is not allowed to be cross-examined with medical commonsense, should not be accepted outside that court.—I am, etc.,

J. G. BOURNE
Salisbury, Wiltshire

Amitriptyline and Imipramine Poisoning in Children

SIR,—Dr. H. J. S. Matthew (29 June, p. 726) gives us the impression that he reads only part of our letter (18 May, p. 386). We indeed gave the result of in-vitro experiments to substantiate our statement that the amount of imipramine recovered in the dialysate is not so small as has been supposed. In fact, we had been stimulated to do the experimental work by the reports of others1 and by our own experience2 of successfully treating by haemodialysis cases of massive overdose of imipramine—despite poor recovery of the drug in the dialysate. A most striking finding was that imipramine apart from being “cleared” by the dialysis membrane is rapidly taken up by the plastic material (polyvinylchloride) of the extracorporeal blood-line system. We suppose that this uptake, which after three hours of dialysis amounts to 60-70% of the drug’s initial value, explains the poor recovery of imipramine in the dialysate in clinical dialysis. It will possibly offer a new therapeutic approach to the management of imipramine poisoning.

By refusing, on the one hand, to accept haemodialysis in the treatment of imipramine poisoning until its efficacy can be proved, and by ignoring, on the other hand, experimental evidence of the value of dialysis Dr. Matthew unfortunately has contributed to the confusion in the field of acute poisoning—a field in which he himself is obviously engaged.3 We would be grateful if this letter of ours should be considered only to enrich the folklore of poisoning.—We are, etc.,

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3 Bandtzh, W., and Bartelheimer, H. R., Archiv für Toxikologie, 1970, 26, 133.

Methoxyflurane Nephrotoxicity

SIR,—May we make a couple of remarks further to your timely leading article on methoxyflurane nephrotoxicity (4 May, p. 239)?: Firstly, we believe an important aspect of our recent study in man1 relates to the differences in methoxyl fluran metabolism and renal sensitivity to inorganic fluoride. These individual differences have been supported in an animal model.2 Furthermore, enzyme induction by phenobarbital causes a doubling of metabolism and thus increased nephrotoxicity.3 The widespread potential of drugs to cause enzyme induction makes it difficult to predict which patients may be predisposed. Thus, our recommendation for dosage restriction is purposes well below the toxic level to allow for the unforeseen addition of factors such as nephrotoxic antibiotics, enzyme induction, and individual variations in metabolism and renal sensitivity.

There is now much evidence that inorganic fluoride is the primary cause of the acute renal lesion. In an animal model doses of fluoride in the same molaric acid approximating that which results from methoxyfluran anaesthesia were injected. At this dosage only inorganic fluoride produced renal functional and pathological abnormalities characteristic of methoxyfluran nephrotoxicity.4 The mechanism of fluoride nephrotoxicity is suggested by recent observations of graded reductions in renal medulla sodium concentration related to increased medullary inorganic fluoride concentration. These changes were associated with increased urine flow.1 Abolition of the hyperosmotic interstitium in the medulla would explain both the inability to concentrate and dilute urine observed in man.1 However, it seems likely that fluoride may have quite widespread effects on different portions of the nephron.—We are, etc.,

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Therapeutic Starvation

SIR,—Dr. J. A. Innes and others reported (18 May, p. 356) results of long-term follow-up of therapeutic starvation which were well worth the toxic level to allow for the unforeseen addition of factors such as nephrotoxic antibiotics, enzyme induction, and individual variations in metabolism and renal sensitivity. In contrast with the evaluation of Innes and co-workers we had a control group. Our study compared the long-term survival of 50 obese south-western American Indians who underwent an initial fasting programme and subsequent conventional therapy with another group of the same ethnic composition matched for sex, age, initial weight, and diabetic status but receiving only low-calorie conventional management. For each of the seven years of follow-up an increasingly greater percentage of the fasted patients than the control subjects weighed less than 80% of their original weight. The ultimate change in mean weight was statistically significant (P<0.05)

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