cadaver arm to be achieved. Separate measurements of trabecular bone mass, cortical bone mass, and bone mineral concentration have also been achieved in patients. The results of this study have been presented in March 1975 at the International Symposium on Computer Tomography in Bermuda and will be further advanced at the European Society of Radiology in Edinburgh in June. It is important to stress that these results were obtained by modification of the E.M.I. brain scanner where the existence of a surrounding water reference makes accurate and absolute results possible.—We are, etc.,

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Prevention of Overdoses

SIR,—The inclusion of emetics in hypnotics proposed by Dr. P. M. Vicary (31 May, p. 503) to prevent the fatal overdose has been suggested before. There are barbiturates available, collectively known as Barbemets, which contain a small amount of ipecacuanha. I attempted to assess the relative safety of these preparations by comparing the death rate per million prescriptions of Barbemets with conventional barbiturate preparations. Over the period 1965-70 there were 93m. prescriptions for barbiturates and 12 354 deaths from barbiturate poisoning, a death rate of 133 per million prescriptions. Over the same period 109 000 prescriptions of Barbemets were issued and one death was recorded from a Barbemet preparation, a death rate of nine per million prescriptions, which would suggest that Barbemets are indeed less of a risk, though the fact of only one death being recorded is insufficient for statistical confidence. In the course of the same inquiry nitrazepam was also investigated and gave a result of 11 deaths per million prescriptions, suggesting it to be safer than barbiturates.

However, Dr. Vicary asks how the commonly used hypnotics can be made safer for those who take overdoses, and the simple device of packaging in blisters or putting the active preparation in a mass of inert substance both warrant more official consideration, since the taking of large numbers of tablets is made much more difficult. For example, 50 tablets of nitrazepam 5 mg weigh 27·5 g, four times greater than 50 tablets of Amytal 100 mg, which weigh 7·7 g, a factor contributing to the greater safety of nitrazepam.

A more radical proposal for reducing barbiturate deaths is to stop the prescription of barbiturates by regulation. Even though the prescribing of barbiturates is diminishing as a result of voluntary restraint, deaths from barbiturates are reducing rather less fast.—I am, etc.,

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Beta-blockers and Fibrinous Peritonitis

SIR,—We read with interest the description by Mr. J. F. Gurry and others (31 May, p. 498) of a further case of sclerosing peritonitis associated with treatment with practolol. We would urge caution, however, in accepting their suggestion that the more recent use of propranolol may have contributed to the development of the lesion in their patient.

We have had referred to us two patients who developed sclerosing peritonitis, one two months and the other six months (also with pleural effusion) after stopping practolol; neither patient had taken any other beta-blocking drug. One of the patients reported by Brown *et al.*[1] had a similar history and several others have been reported.[2-4] Since the abdominal effects of practolol can thus be delayed, it is difficult to implicate other beta-blocking drugs given at the same time or at a later date.—We are, etc.,

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1 Brown, P., *et al.*, *Lancet*, 1974, **2**, 1477.
2 Bendtzen, K., and Søborg, M., *Lancet*, 1975, **1**, 629.
3 Kristensen, K., Kristensen, J. S., and Thorborg, J. V., *Lancet*, 1975, **1**, 741.
4 Halley, W., and Goodman, J. D. S., *British Medical Journal*, 1975, **2**, 337.

*** The authors sent a copy of this letter to Mr. Gurry and his colleagues, whose reply is printed below.—ED., *B.M.J.*

SIR,—I have had the opportunity to read the letter from Dr. Thompson and Mr. Jackson, and in the absence of my two co-authors, who are overseas, I would like to comment on it.

It is most reassuring to learn that cases of sclerosing peritonitis have developed after cessation of practolol therapy without the subsequent use of propranolol. Though there have been previous reports of the delayed onset of peritonitis, the specific point of whether or not propranolol was used in the place of practolol has not been made until now.

In view of this report from Dr. Thompson and Mr. Jackson our cautionary note (31 May, p. 498) is no longer justified, nor is there any evidence to date to imply that propranolol therapy alone may cause this condition.—I am, etc.,

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Ocular Reactions to Beta-blockers

SIR,—I should like to draw attention to the following case of corneal perforation occurring during treatment with beta-blocking drugs.

A 55-year-old man presented at this hospital in May 1974 complaining of dry eyes. In July 1974 his left cornea perforated. He had suffered from angina for five years and had been started on propranolol in June 1970; this was continued for eight weeks, when practolol 500 mg daily was substituted. He remained on this dose of practolol until April 1973 (32 months). In April 1973 he complained to his general practitioner about dry eyes and oxprenolol 40 mg three times daily was substituted for the practolol. He remained on oxprenolol until July 1974, at which time the cornea perforated.

This case is not easy to assess in terms of causation. I accept that this man was taking practolol for about 2½ years before starting oxprenolol; nevertheless, he had been off practolol and taking oxprenolol for a full 15 months before the corneal perforation. Clearly oxprenolol is not above suspicion and I feel that this is sufficient reason for the case to be reported.—I am, etc.,

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Anaphylactoid Skin Reaction after Intradermal Secretin

SIR,—Drs. S. R. Bloom and A. S. Ward (18 January, p. 126) reported on the impairment of secretin release in patients with duodenal ulcer, thus substantiating the original idea of Demling *et al.*,[1] who discussed deficiency of endogenous secretin as a potentially relevant factor in the pathogenesis of that disease. The fact that the secretin deficit in Drs. Bloom and Ward's short-history patients was as great as in their long-history patients may argue in favour of it being a primary defect.

While conducting a trial of exogenous secretin as a possible therapeutic agent in duodenal ulcer we obtained data possibly associated with immunogenic properties of that hormone. About 15 minutes after intradermal injection of 40 IU of synthetic secretin[2] dissolved in 0·05 ml of 0·9% saline subjects developed a local reaction resembling cutaneous anaphylaxis (urticaria plus surrounding erythema) indistinguishable from that following intradermal administration of 0·05 ml of histamine dihydrochloride (0·1 mg/ml of 0·9% saline). Urticaria and erythema vanished within 60-120 minutes. To study that phenomenon in more detail different groups of subjects who had not previously received secretin were tested in the same way. Eight out of eight duodenal-ulcer patients showed anaphylactoid skin reactions to intradermal secretin, two out of three gastric ulcer patients, four out of six cases of ulcerative colitis or Crohn's disease, and 13 out of 23 subjects suffering from various gastrointestinal diseases other than those listed above (for example, chronic pancreatitis, gastric and colonic polyps, colon cancer). Interestingly enough, six out of seven healthy volunteers did not respond, while one healthy individual developed a questionable reaction. Systemic symptoms were noted in none of the subjects after intradermal secretin injection, nor was sensitization against secretin detectable in vitro when passive haemagglutination or blast-cell-transformation techniques were employed.

Viewing the present preliminary data it may be suggested that there is—especially in duodenal-ulcer patients—a factor conditioning anaphylactoid skin reaction due to intradermal secretin. An immunological mechanism based on the presence of secretin antibodies cannot be excluded—though there were positive reactions in subjects who had never before received secretin and in spite of negative in vitro tests. On the other hand, non-specific release of histamine or other vasoactive substances by secretin has not been reported so far. Whether or not there are relations between the impaired secretin release in duodenal ulcer patients and a special immunological disposition of those patients against secretin is an un-