PROPRANOLOL IN ACUTE MYOCARDIAL INFARCTION

SIR,—I have read the observations of Dr. Balcon and his colleagues of King's College Hospital,[1] and also the report of a multicentre trial.[2] There is no doubt that propranolol is a most interesting drug. I have several patients with severe ventricular arrhythmias who failed to respond to any of the other anti-arrhythmic agents, including phenytoin and antazoline, either singly or in combination, but who have had a dramatic response to oral propranolol. I am not entirely convinced by the numerous papers in the September issue of the *American Journal of Cardiology*, and I am very dubious about the safety of the drugs when given *intravenously* to a patient with a severe acute myocardial infarction. During a recent world trip I heard of a number of patients, in Copenhagen, U.S.A., and elsewhere, who died immediately after, or during, an intravenous injection of 5–7·5 mg. of propranolol. The proponents of the intravenous use of this drug should remember that it may be administered rapidly by a sleepy resident at 3 A.M., and this may well prove fatal to the patient with acute myocardial infarction, whether or not atropine is given, and whether or not congestive failure or other established contraindications are present. In addition, the individual response to any drug is an unpredictable quantity, and a good result in some patients is not a sufficiently good reason to claim that the intravenous use of this drug is an important contribution to drug treatment in acute coronary-care units or elsewhere.

I would certainly agree with Dr. Balcon and his colleagues that Dr. Ikram and Dr. Nixon,[3] and many others, might do well to reconsider the use of routine intravenous propranolol in myocardial infarction. Much of the available evidence on the "safety" of intravenous propranolol is based on patients who did not have myocardial infarction, and this fact seems to have been partly overlooked.

Sir Charles Gairdner Hospital,
Shenton Park,
Western Australia. W. G. SMITH.

ABDOMINAL ARTERIAL MURMURS IN LIVER DISEASE

SIR,—I had great pleasure in reading the article by Dr. Clain and his colleagues.[4] I have already described this systolic murmur.[5] A systolic murmur was audible in 20 cases of malignant liver—7 cases with primary malignant hepatomas, and 13 with secondary malignant disease of the liver. I did not hear the murmur over livers other than malignant ones.

I suggest that the detection of a systolic bruit over the liver is to be regarded as diagnostic of malignant disease of the liver, whether primary or secondary.

Ein Shams University,
44 Ahram Street,
Heliopolis, Cairo. M. H. MAAMOUN.

FIBRINOLYSIS IN CITRATED AND OXALATED BLOOD

SIR,—Dr. Menon's observations and conclusions last week (p. 116) on the fibrinolytic potential of euglobulin fraction prepared from oxalated and citrated blood are not new. In an excellent, but largely neglected, series of methodological studies on euglobulin fractionation techniques Blix [6] demonstrated conclusively the inhibitory effect on fibrinolysis, as measured by fibrin plates and euglobulin-lysis times, of the anticoagulant sodium oxalate compared with sodium citrate.

South-East Scotland Regional Blood Transfusion
Research Laboratories,
Royal Infirmary,
Edinburgh 3. P. C. DAS.

UREA DETERMINATION AND DEXTRAN-40

SIR,—Dr. Lambooy and Mrs. van Amson [1] report that they were unable to determine the urea content of blood-serum in patients who had received 10% dextran-40 (low-molecular-weight dextran, 'Rheomacrodex') infusions, owing to cloudy precipitation making photocolorimetric determination impossible. Their urea-determining method "consists in adding 3 ml. reagent (8g. *f*-dimethylaminobenzaldehyde in 500 ml. 96% alcohol and 50 ml. 36% hydrochloric acid) to 3 ml. deproteinised serum, dilution 1/10, and doing a photocolorimetric determination ten minutes later."

The explanation for their observation most probably is that dextran is precipitated by ethyl alcohol. The optical density of the turbidity is a measure of the amount of dextran present in the specimen. The quantity of dextran precipitated by ethyl alcohol has been used as a simple method for the determination of dextran concentrations in blood and urine by Jacobsson and Hansen.[2]

Department of Medicine,
Karolinska Sjukhuset,
Stockholm, Sweden. C. G. NILSSON.

ISOLATION OF LISTERIA MONOCYTOGENES

SIR,—The isolation of *Listeria monocytogenes* from contaminated materials such as fæces and vaginal secretions may have been unsuccessful so far because of the lack of good selective media. The cold-enrichment technique followed by plating on blood-agar or potassium-tellurite-agar shows positive results only after a long period, which means in some cases that listeria can be isolated only after 1–1½ years. Gram-negative bacteria and *Proteus* spp. especially interfere with the isolation of listeria by overgrowing blood-agar; potassium-tellurite-agar often inhibits the development of listeria colonies. Beerens and Tahon-Castel [3] have described a new medium containing nalidixic acid, which suppresses the growth of gram-negative bacteria, and which has given surprising results in our laboratory during recent months.

A year ago a worker in an egg-product factory died from listeria meningitis. Fæces of 85 workers in this factory were obtained and investigated for *L. monocytogenes*. Only 1 of the samples proved to be positive after 4 months with the old method. Using nalidixic-acid/blood-agar for the first time, listeria was isolated (about 10–20 *L. monocytogenes* colonies per plate) from another 4 fæcal samples, which remained negative on blood-agar. 3 other samples, which were negative on blood-agar after storage at 4–6°C for 6–8 months, appeared to be positive after inoculation on to the new medium.

The medium described by Beerens and Tahon-Castel is prepared from the following ingredients: meat extract 3 g.; tryptose (Difco) 10 g.; sodium chloride 5 g.; nalidixic acid 0·04 g.; agar 15 g.; and distilled water to 1000 ml. A solution made by heating at pH 7·2, is then autoclaved for 20 minutes at 120°C, and cooled to 50°C. 7·5 ml. of horse-blood is added to 100 ml. of the medium, which is poured into Petri dishes. Nalidixic acid may be sterilised, but it can also be added together with the blood after it has been dissolved in distilled water.

Laboratory for Zoonoses,
National Institute of
Public Health,
Utrecht, The Netherlands. E. H. KAMPELMACHER.

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2 A Multicentre Trial. *ibid.* p. 1435.
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6 Blix, S. *Scand. J. clin. Lab. Invest.* 1961, suppl. 58.

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