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Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are now being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

Acute Systolic Hypertension during Myocardial Infarction

SIR,—The formal study by Dr. K. M. Fox and others (19 July, p. 128), delineating and re-emphasizing the relatively poor immediate prognosis of patients who have systolic hypertension during the development of myocardial infarction, is valuable and of practical importance. The finding within the first few hours after the onset of symptoms of a raised systolic blood pressure in patients not suspected of having pre-existing hypertension should alert physicians to the increased risk of serious arrhythmias, and indicates that prophylactic antiarrhythmic therapy should be considered at this early stage.

The wisdom of introducing hypotensive treatment is more in question, since the hypertensive response is transient, seldom extreme, and probably supportive in terms of perfusion of the brain and kidneys during a period of low cardiac output. And it does not follow that the incidence of cardiac failure would be reduced by such treatment. This acute hypertensive response is one of the systemic signs of sympathetic activity, excessive in such patients, to myocardial ischaemia. In support of this view, we have observed, when documenting some of the metabolic and hormonal changes which occur during myocardial infarction,^{1,2} a positive correlation between raised systolic blood pressures and plasma total catecholamines in patients examined within five hours of the onset of symptoms. There was also a positive correlation between the blood glucose/serum insulin ratio with systolic blood pressure, and this is relevant to the increased incidence of glycosuria observed by Dr. Fox and colleagues in these patients with systolic hypertension.

Serum free fatty acids are raised for the

first 12 hours or so after the onset of symptoms of myocardial infarction, and this is another manifestation of the sympathetic response. Recent analysis of earlier reported data³ has shown a positive relationship ($r = 0.69$) at 4–8 hours after the onset of symptoms between serum free fatty acid concentrations and systolic blood pressure. Raised serum free fatty acid levels are associated with a high incidence of arrhythmias in patients with myocardial ischaemia,⁴ and this may be one explanation of the increased incidence of arrhythmias in patients with transient systolic hypertension.

Systolic hypertension should be carefully looked for as a clinical sign of an excessive sympathetic response to myocardial ischaemia and infarction, particularly since prophylactic antiarrhythmic treatment may be indicated and since metabolic intervention is now becoming a reality.⁵ But, in my view, the raised blood pressure should be left untreated.—I am, etc.,

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- 1 Vetter, N. J., et al., *Lancet*, 1974, 1, 284.
- 2 Strange, R. C., et al., *European Journal of Clinical Investigation*, 1974, 4, 115.
- 3 Oliver, M. F., Kurien, V. A., and Greenwood, T. W., *Lancet*, 1968, 1, 710.
- 4 *Lancet*, 1975, 1, 313.
- 5 Rowe, M. J., Neilson, J. M. M., and Oliver, M. F., *Lancet*, 1975, 1, 295.

Candida Endocarditis

SIR,—May I comment on your leading article (2 August, p. 264) summarizing the slightly improved but still gloomy situation in regard to the management of candida endocarditis?

Possible toxicity aside, only one of the three patients reported by Record *et al.*¹ who were given 5-fluorocytosine survived. This is a small series but hardly perhaps a real improvement upon the survival from established treatment with amphotericin B. You say that what is usually required to improve survival is early recourse to further surgery in addition to antifungal therapy. This is almost certainly more important than attempts of treatment with alternative antimycotic agents, which alone are no more likely to succeed than is amphotericin. There are a number of points which make 5-fluorocytosine an unhappy alternative recommendation.

It is known that minority populations of yeast-like fungi exist that are highly resistant to 5-fluorocytosine. These have been selected during a single course of treatment with this drug in both urinary candidiasis following renal transplant² and cryptococcal meningitis.³ The same situation has now been recorded in relation to treatment of a case of endocarditis caused by *Candida parapsilosis* relapsing on the 37th day of treatment with 5-fluorocytosine.⁴

In my opinion initial therapy should be with amphotericin. 5-Fluorocytosine should not be used alone to cover the replacement of the affected valve, nor as postoperative treatment. It may be given in addition to amphotericin, particularly in the post-operative phase. The emergence of yeasts resistant to 5-fluorocytosine will be prevented and amphotericin may increase the rate of incorporation of 5-fluorouracil into yeast RNA,⁵ possibly by facilitating penetration into the cell by its own action on the cell membrane.

It should be remembered that 5-fluorocytosine is active only against yeast-like fungi. It has no activity against filamentous fungi. These are less common causative agents, but in the series you cite⁶ 25 of the 116 cases reported were not due to candida and 15 of these were caused by *Aspergillus spp.*