LETTER TO THE EDITOR

PROPRANOLOL AND EXPERIMENTAL MYOCARDIAL NECROSIS

Sir.

Propranolol has been shown to have beneficial effect in occlusive coronary disease. Maroko *et al.* (1) have observed decrease in the size of infarcts by propranolol in coronary ligated dogs. However, myocardial infarction is not always a result of coronary occlusion. Infarction without the occlusion of coronary artery is well documented (3). The effect of propranolol in such type of myocardial infarction is not known and hence has been explored in the present study.

Myocardial infarction was produced in albino rats (English port strain) of either sex weighing 250±10 *g* by the injection of isoprenaline (85 *mg/kg s.c.*) once a day for two consecutive days as described by Rona *et al.* (2). Propranolol (5 *mg/kg*, orally) was administered to a group of 12 rats once a day for seven days and the animals were subjected to isoprenaline challenge on sixth and seventh days. A control group of 12 rats was treated with saline (1 *ml/kg*, orally) once a day for seven days. The animals were sacrificed 24 hr after the isoprenaline challenge and the degree of myocardial necrosis graded according to the method of Rona *et al.* (2). On macroscopic examination, the degree of necrosis in the control group was found to be 2.3±0.21. In the propranolol treated group the necrosis was reduced to 0.67 ±0.21 ($P < 0.001$). When analysed histologically the degree of necrosis in the control group was 2.7±0.23 and reduced to 1.2±0.12 ($P < 0.001$) in propranolol pretreated group. In none of the rats there was complete protection against isoprenaline-necrosis on microscopic examination (Fig. 1).

The protective action of propranolol against isoprenaline induced myocardial necrosis as observed in the present study is significant in the light of involvement of catecholamines in myocardial infarction resulting from acute stress. Catecholomines markedly enhance the work load of the heart resulting in relative ischeamia of the heart and its subsequent necrosis. The cardiac stimulation by catecholamines is through the activation of β-adrenoceptors. Propranolol being β-adrenoceptor antagonist is, therefore, expected to prevent the heart from the deleterious effects of catecholamines. Results obtained in the present study support this conjecture as propranolol pretreatment significantly reduced the degree of myocardial necrosis induced by isoprenaline.

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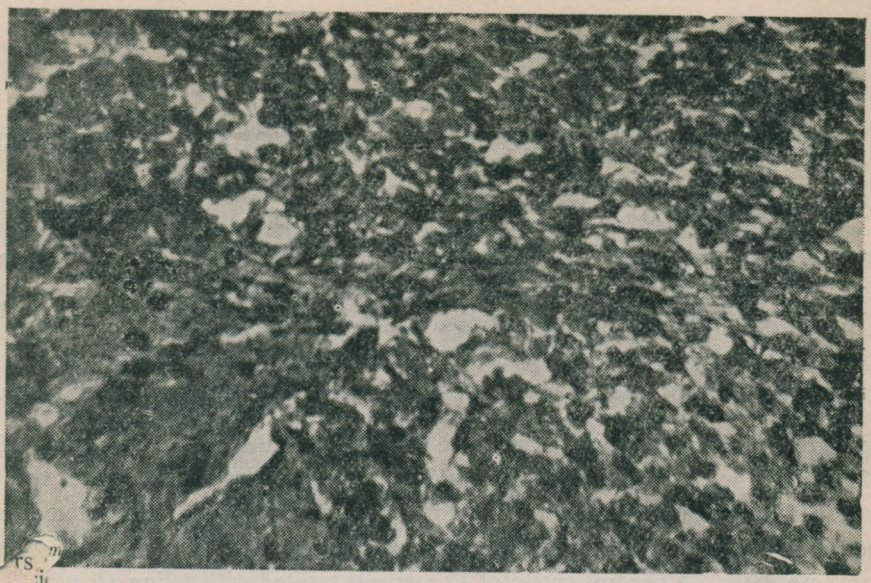

Fig. 1A: Photomicrograph of heart of rat pretreated with saline (1 *ml/kg*, orally) once a day for seven days and then subjected to isoprenaline challenge. There is massive infiltration of inflammatory cells, hyaline necrosis and vacuolation of cells. H.E. x 300.

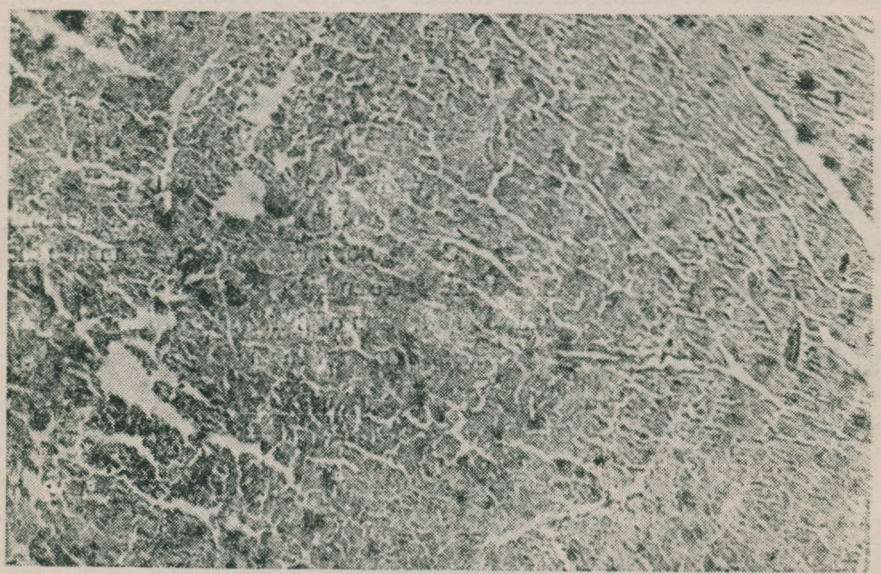

Fig. 1B: Photomicrograph of heart of rat pretreated with propranolol (5 *mg/kg*, orally) once a day for seven days and then subjected to is operanaline challenge. Note that only leucocytic infiltration and fragmentation of muscle fibres have been produced. No vacuolation of cells or hyaline degeneration is visible. H.E. x 300.