of 8 mmHg and 6 mmHg after intravenous and oral oxprenolol, respectively. There was little change in the left ventricular function curves relating stroke work to end-diastolic pressure either after intravenous or oral oxprenolol. There was a significant reduction in the left ventricular dp/dt (max) during exercise both after intravenous and oral oxprenolol, but this reduction could probably be largely accounted for by the decrease in exercising heart rate.

These results suggest that in most patients with uncomplicated angina pectoris, intravenous and oral oxprenolol will both result in a conspicuous relief of pain during exercise, and the long-term effects of the oral preparation may be predicted with reasonable certainty from the symptomatic effects of the acute intravenous administration of the drug. Oxprenolol results in a significant improvement in the exercise electrocardiogram whether given by intravenous injection or by mouth. The predominant haemodynamic change is a reduction in heart rate. The fact that left ventricular function is little changed by the drug either after its intravenous injection or after 6 months' oral treatment may possibly be explained by the positive benefit to myocardial oxygen consumption accruing from the direct restraint imposed on the tachycardia of exercise by the negative chronotropic effects of the drug outweighing the reduction in myocardial contractility occasioned by its negative inotropic actions due to adrenergic blockade.

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Potentiation of the cardiovascular effects of some catecholamines by a monoamine oxidase inhibitor

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The pressor effect of noradrenaline (NA) is not substantially potentiated by treatment with monoamine oxidase inhibitors (MAOI) (Horwitz, Goldberg & Sjoerdsma, 1960; Elis, Laurence, Mattie & Prichard, 1967; Pettinger & Oates 1968) and it has been generally assumed that the effects of MAOI on other catecholamines are insignificant.

Three healthy male subjects aged 27–41 years were studied. Small doses of NA (1·125–36 μg), adrenaline (1·125–36 μg) and isoprenaline (0·5–9 μg) were delivered intravenously at a constant rate over 30 s and dose related effects determined on heart rate and blood pressure by sphygmomanometry. The object was to simulate a possible accidental intravenous administration of catecholamines. Intravenous phenylpropanolamine (0·9–20 mg), was included as a comparison since it is known that the cardiovascular effects of oral phenylpropanolamine are markedly potentiated by a MAOI (Cuthbert, Greenberg & Morley, 1969). The observations were repeated immediately after each subject had received tranylcypromine (30 mg) daily for 8–14 days. The degree of potentiation was estimated from displacement of dose-response curves using a reciprocal transformation (Draper & Smith, 1966; Vere, 1971).

The results (Table 1) show that the pressor effect of intravenous phenylpropanolamine was potentiated approximately 4–5 times (systolic blood pressure) and 3–10

TABLE 1. *Degree of potentiation (ratios) of the effects of phenylpropanolamine, noradrenaline, adrenaline and isoprenaline on heart rate (HR), diastolic (DBP) and systolic blood pressure (SBP) in 3 healthy male subjects after a course of tranylcypromine (30 mg daily) for 8–14 days*

	Phenylpropanolamine potentiation of			Noradrenaline potentiation of		
Subject	Fall in HR	Rise in DBP	Rise in SBP	Fall in HR	Rise in DBP	Rise in SBP
D.W.V.	5·8	3·0	4·6	4·3	1·3	2·0
P.B.	6·3	10·4	4·6	1·6	†	†
M.F.C.	2·5	4·6	3·9	2·0	2·7	1·7

	Adrenaline potentiation of			Isoprenaline potentiation of		
Subject	Rise in HR	Fall in DBP	Rise in SBP	Rise in HR	Fall in DBP	Rise in SBP
D.W.V.	1·8	4·2	3·0	2·9	3·6	1·0
P.B.	4·5	2·0	1·8	2·3	2·5	−1·7
M.F.C.	2·6	‡	2·6	4·0	3·1	1·8

†In this subject noradrenaline caused bradycardia but satisfactory dose related effects on blood pressure were not obtained. There was no evidence of substantial potentiation.

‡In this subject low doses of adrenaline caused minimal falls in DBP, increasing the dose caused small rises in DBP which were potentiated to a similar degree to that of SBP.

times (diastolic blood pressure) while the reflex bradycardia was potentiated approximately 2·5–6 times. In contrast, the pressor effect of NA was only slightly potentiated although the reflex bradycardia was more marked. There was a moderate potentiation (approximately 2–4 fold) of the effect of adrenaline on the heart rate and diastolic pressure and a less marked potentiation of the rise in systolic pressure. Similar results were obtained with isoprenaline but the rise in systolic pressure was not potentiated.

The changes in blood pressure induced by intravenous catecholamines in subjects taking tranylcypromine appear to be unimportant. Since effects on β-adrenoceptors are potentiated, however, an increased risk of cardiac dysrhythmia may exist, though none was seen in our healthy subjects.

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Interactions between catecholamines and tricyclic and monoamine oxidase inhibitor antidepressive agents in man

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The pressor effect of indirectly acting sympathomimetic amines is potentiated in subjects receiving a monoamine oxidase inhibitor drug (Elis, Laurence, Mattie