patients with functional psychiatric illness in the absence of clinical evidence of physical disease or alcoholism.

We are grateful to Dr P B Guyer and Dr K C Dewbury for help in reviewing the chest radiographs, and Mrs H Hills for helping to collect data.

The table giving details of the 21 patients with relevant radiographic abnormalities may be obtained from Dr Jennifer Hughes.

[1] Abramczuk JA, Rose NM. Pre-anaesthetic assessment and the prevention of post-ECT morbidity. *Br J Psychiatry* 1979;**134**:582-7.

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Adverse interaction between nifedipine and β-blockade

The calcium antagonist drug nifedipine is hypotensive and produces its systemic effect by reducing systemic vascular resistance.[1] It is also effective in treating angina pectoris, whether caused by coronary artery spasm or by effort.[2-4] β-Blockade is a well-established treatment for hypertension and for angina. In combination with propranolol, nifedipine reduces ST-segment deviations in monitored patients with exertional angina.[3] A combination of β-blockade and nifedipine should therefore be especially effective in treating co-existing hypertension and angina.[4] We studied 15 patients with stable effort angina and hypertension who were treated with nifedipine in addition to pre-existing treatment with β-blockade and a diuretic. One patient developed excessive hypotension.

Patients, methods, and results

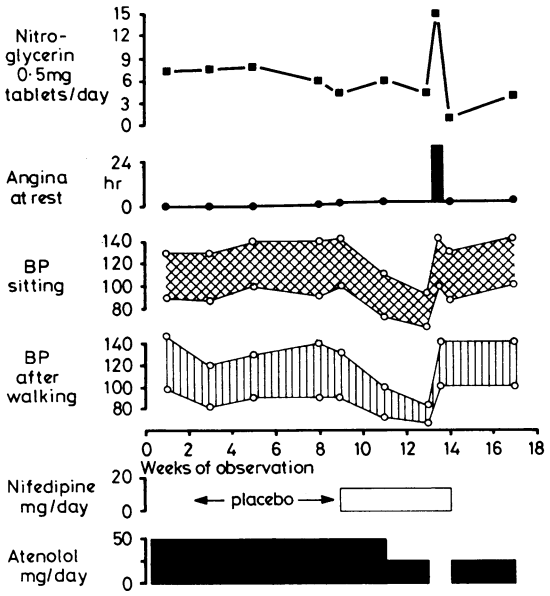
Fifteen patients with established hypertension and stable exertional angina were controlled by a standard dose of the cardioselective β-antagonist atenolol (50-100 mg/day) together with diuretic treatment and prazosin (average total dose 9 mg/day in eight patients) as well as intermittent sublingual nitroglycerin as required. The patients had been attending the hypertension clinic for several months or years. After an initial run-in period of six weeks control blood pressure readings were done in the sitting, standing, and lying positions; after walking exercise; and after hand-grip and cold-immersion tests. Nifedipine placebo (1 capsule twice daily) was then added for six weeks and the blood pressure readings were repeated at two-weekly intervals. Then active nifedipine (10 mg twice daily) was substituted for nifedipine placebo for a further six weeks. Finally, active nifedipine was replaced by nifedipine placebo and the readings were again repeated after one month.

Nifedipine was an active hypotensive agent, even in the presence of β-blockade, and was superior to placebo in reducing the sitting, standing, and post-exercise blood pressures as well as the cold-pressor response. Mean (±SE) sitting pressures (mm Hg) were as follows. Initial therapy, 162±7/108±4; same therapy+nifedipine placebo, 165±7/102±3; same therapy +active nifedipine, 148±6/94±3; same+placebo nifedipine, 164±7/105±4 ($p<0.005$ for all comparisons between active nifedipine and placebo). Side effects included headaches and dizziness as well as dilatation of varicose veins in two patients. The addition of nifedipine did not change the seveirty of angina, as judged by a subjective scoring system.

In one patient (figure) the blood pressure on active nifedipine and β-blockade progressively dropped down to sitting levels of only 90/60 with post-exercise levels of only 80/60. The patient felt extremely weak but his angina was no worse. The atenolol was stopped, his pulse rate and blood pressure rose, and a period of severe unstable angina with persistent chest pain at rest for 24 hours developed while he was taking nifedipine alone. There were no enzymatic changes of myocardial infarction. Stopping nifedipine and restarting β-blockade had little effect on the blood pressure but rapidly relieved the angina. Throughout the observations the patient received no other drugs except cyclopethiazide with potassium chloride one tablet daily and allopurinol 300 mg thrice daily.

Comment

Nifedipine was an active hypotensive agent even when added to β-blockade in these patients. It did little to improve angina, but effort tolerance rose. In one patient the combination of nifedipine and β-blockade progressively lowered the blood pressure over one month



Excessive hypotension produced in hypertensive patient by interaction between nifedipine and beta-blocker.

to such low levels that the patient became extremely weak and unable to exercise. His angina, however, was unchanged. When the atenolol was stopped unstable angina developed, which was relieved with the reintroduction of β-blockade and the cessation of nifedipine. Thus nifedipine was unable to treat the angina of β-blockade withdrawal. The possible advantages of nifedipine alone as an anti-anginal and anti-hypertensive agent in patients with hypertension and angina must still be assessed. When given to such patients who are already receiving β-blockade our results show that (*a*) it is the hypotensive rather than the anti-anginal effects of nifedipine that predominate; and (*b*) if excess hypotension develops it is the nifedipine and not the β-blockade that should be stopped.

[1] Olivari MT, Bartorelli C, Polese A, Fiorentini C, Moruzzi P, Guazzi MD. Treatment of hypertension with nifedipine, a calcium antagonistic agent. *Circulation* 1979;**59**:1056-62.
[2] Endo M, Kanda I, Hosada S, Hayashi H, Hirosawa K, Konno S. Prinzmetal's variant form of angina pectoris. Re-evaluation of mechanisms. *Circulation* 1975;**52**:33-7.
[3] Oakley GDG, Fox KM, Dargie HJ, Selwyn AP. Objective assessment of treatment in severe angina. *Br Med J* 1979;i:1540.
[4] Lynch P, Dargie H, Krikler S, Krikler D. Objective assessment of anti-anginal treatment: a double-blind comparison of propranolol, nifedipine, and their combination. *Br Med J* 1980;**280**:184-7.

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Corrections

Haemangioma of the cord: a further cause of raised maternal serum and liquor alpha-fetoprotein

An error occurred in this paper by Dr A J Barson and others (8 November, p 1252). Maternal serum alpha-fetoprotein concentrations should have been expressed as ng/ml not μg/ml. The fourth and fifth sentences of the case report should thus have read: "The maternal serum α-fetoprotein concentration at 16 weeks' gestation was raised at 65 ng/ml . . . At 19 weeks' gestation it was 165 ng/ml. . . ."

Comparison of nicotine chewing-gum and psychological treatments for dependent smokers

We regret that an error occurred in this paper by Mr Martin Raw and others (16 August, p 481). In table I the figures in the last two rows in the column headed "Nicotone gum" should have read "30(43)" and 35(51)."