Dr A T R Axon, Dr R Seidelin, and Mr E A Benson for permission to report this case; and to Dr D J Lintott for the radiological studies.

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General Infirmary, Leeds LS1 3EX
I HAMILTON, MB, MRCP, medical registrar, gastroenterology unit
L REIS, MD, surgical registrar

Wharfedale Hospital, Otley, Yorkshire LS21 2LY
SHERNAZ BILIMORIA, MD, medical registrar

Hammersmith Hospital, London W12 0HS
R G LONG, MD, MRCP, honorary senior medical registrar (now senior registrar, St Thomas's Hospital, London SE1 7EH)

Unpredictable response to nifedipine in severe cardiac failure

Nifedipine is a vasodilator with effects mainly on the arterioles and hence might have a place in the management of severe cardiac failure.[1 2] We set out to study the response to an oral dose of 30 mg but this was reduced to 20 mg after a patient developed severe hypotension. A similar reaction occurred in another patient on the smaller dose.

Patients, methods, and results

Six men with severe chronic cardiac failure were studied. Three had coronary artery disease and three had congestive cardiomyopathy. All were having treatment with diuretics and, when indicated, digoxin. Each gave his consent to the procedure, which was approved by the ethical committee. After diagnostic cardiac catheterisation a Swan-Ganz double lumen thermal dilution cardiac output catheter was positioned in the pulmonary artery and a fine polyethylene catheter in the brachial artery. On return to the ward control observations of systemic arterial pressure (SAP), pulmonary artery pressure (PAP), pulmonary artery "wedge" pressure (PAW), right atrial pressure (RAP), and cardiac output (in triplicate) were made at 15-minute intervals for one hour. Nifedipine 30 mg (cases 1 and 2) or 20 mg (cases 3-6) was given orally. Observations were then repeated at 15-minute intervals for the first hour and hourly for five hours.

Haemodynamic changes were detected within 15 minutes, peaked at 30 to 45 minutes, and persisted up to five hours (table). In all patients SAP fell by an average of 30%, PAP by 29%, and PAW by 25%. Systemic vascular resistance was reduced by an average of 47%. Cardiac output increased in three patients. In cases 2 and 6, the main subjects of this report, severe hypotension accompanied a fall in cardiac output. In case 2 the patient received 30 mg nifedipine. After 15 minutes he became cyanosed and confused, SAP fell progressively from 130/70 to 50/30 mm Hg, PAP from 80/30 to 50/24 mm Hg, and PAW from 34 to 25 mm Hg. Cardiac output declined from 4·8 to 2·4 l/min and stroke volume from 45 to 31 ml. In case 6 the patient received 20 mg nifedipine, and a similar reaction developed; SAP fell from 110/65 to 65/35, PAP from 70/35 to 40/22, and PAW from 35 to 22 mm Hg. Cardiac output decreased from 4·1 to 3·1 l/min and stroke volume from 57 to 52 ml.

Both patients responded to intravenous injection of 20 ml 10% calcium chloride followed by 10 ml 1:10 000 adrenaline. Cardiac output rose and systemic and pulmonary arterial pressures returned to their control levels. There were no further complications.

Comment

Vasodilators may cause hypotension by producing an excessive fall in peripheral resistance, and this leads to a reduced left ventricular filling pressure and volume. In case 2 peripheral resistance fell more than in any other patient and the resulting hypotension was aggravated by a concomitant reduction in cardiac output. In case 6 the decrease in peripheral resistance was modest and the fall in cardiac output was a major factor in the resulting circulatory collapse. In both the reduction in left ventricular filling pressure was substantial but no more than in case 4, where cardiac output was augmented. Furthermore, in previous studies in which filling pressure was reduced to a comparable extent cardiac output invariably rose.[1 2] This, and a recent report of a patient in whom pulmonary oedema was precipitated by nifedipine,[3] raises the possibility that in certain patients the drug can depress myocardial contraction. That this effect is not usually seen in man is ascribed to reflex beta-adrenergic stimulation from activation of the baroreceptor reflex. The fall in heart rate in our patients despite the extreme hypotension is therefore interesting.

Despite earlier promising reports nifedipine cannot be recommended unreservedly for the treatment of cardiac failure and it should be used cautiously in patients with poor left ventricular function.

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Cardiac Department, London Chest Hospital, London E2 9JX
NICHOLAS BROOKS, MD, MRCP, clinical lecturer and honorary senior registrar
MARTIN CATTELL, MB, MRCP, clinical lecturer and honorary senior registrar
JOHN PIDGEON, MB, MRCP, registrar
RAPHAEL BALCON, MD, FRCP, consultant cardiologist

Haemodynamic observations before (control) and 45 minutes after nifedipine (cases 1, 3, 4, 5) or at peak effect before intervention (cases 2, 6)

Case No	Age (years)	Diagnosis	Concomitant treatment (mg/day)	Condition	Heart rate (min)	Pressures (mm Hg)				CO (l/min)	S Vol (ml)	SVR units	PVR units
						SAP	PAP	PAW	RA				
1	63	CAD	Frusemide 120 Amiloride 15	Control Nifedipine 30 mg	98 95	119 75	30 19	25 14	5 3	3·2 3·4	33 36	36 19	1·5 1·5
2	55	CCM	Frusemide 160 Amiloride 20	Control Nifedipine 30 mg	107 92	102 35	44 30	34 25	14 16	4·8 2·9	45 32	18 7	2·1 1·7
3	58	CCM	Frusemide 120 Amiloride 20 Digoxin 0·375	Control Nifedipine 20 mg	63* 65*	80 75	33 32	22 21	13 11	3·5 5·4	56 83	19 12	3·1 2·0
4	52	CCM	Frusemide 250 Spironolactone 100 Digoxin 0·25	Control Nifedipine 20 mg	120 115	100 82	46 33	37 25	14 11	4·3 5·1	36 44	20 14	2·1 1·6
5	61	CAD	Frusemide 120 Amiloride 10	Control Nifedipine 20 mg	83 80	140 127	50 40	40 35	4 4	5·0 6·8	60 85	27 18	2 0·7
6	59	CAD	Frusemide 120 Slow-K 3600	Control Nifedipine 20 mg	72 60	80 50	48 30	35 22	20 20	4·1 3·1	57 52	15 10	3·2 2·5

*Atrial fibrillation.
Abbreviations SAP = mean systemic arterial pressure. PAP = mean pulmonary artery pressure. PAW = mean pulmonary artery "wedge" pressure. RA = right atrial pressure. CO = cardiac output. S Vol = stroke volume. SVR = systemic vascular resistance. PVR = pulmonary vascular resistance. CAD = coronary artery disease. CCM = congestive cardiomyopathy.