vascular malformations must be confirmed angiographically and should be treated definitively. This is the first reported case in which a nonsurgical approach has been used in the treatment of SAPA fistulae. Embolization is an alternative to surgery and should be considered in most cases.

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Rapid Attenuation of Response to Nifedipine in Primary Pulmonary Hypertension

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In a 22-year-old woman with primary pulmonary hypertension resistant to all previous attempts to reduce the pulmonary vascular resistance, there was dramatic improvement after the first dose of nifedipine, 20 mg, which was not sustained with subsequent doses. While there was a persistent reduction in systemic vascular resistance, the initial drug-related reduction in pulmonary vascular resistance was progressively attenuated with the subsequent four doses of nifedipine, 20 mg.

Primary pulmonary hypertension may be caused by pulmonary vasoconstriction. Attempts to alleviate vasoconstriction have included the use of oxygen, acetylcholine, isoproterenol, phentolamine, diazoxide, tolazoline, prostacyclin, hydralazine, dilatiazem, verapamil, and nifedipine.* We report a patient with primary pulmonary hypertension in whom nifedipine caused a dramatic reduction in pulmonary hypertension initially. However, tachyphylaxis developed with subsequent doses.

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CASE REPORT

A 22-year-old black woman was first admitted to this hospital in February, 1981 for progressive dyspnea on exertion and near syncopal episodes. These had begun approximately five months prior to her admission when she noted dyspnea on climbing stairs. Physical examination revealed the presence of a right ventricular heave and a grade 3/6 holosystolic heart murmur loudest at the upper left sternal border with radiation down the left sternal border toward the left mid-clavicular line. The lungs were clear to percussion and auscultation. There was moderate jugular venous distension without peripheral edema. There was RVH noted on the electrocardiogram, and cardiac enlargement noted on the chest x-ray film without any pulmonary parenchymal abnormalities. A ventilation/perfusion lung scan gave normal findings. Echocardiogram showed a small left ventricle with a hypokinetic septum and decreased systolic function, reduced EF slope and an enlarged right ventricle.

The patient underwent left and right heart catheterization. Right atrial (RA) pressures (mm Hg) were elevated to a = 25, v = 18 (mean RA-17); right ventricular pressures, 90/6; pulmonary artery pressures (PAP), 90/60 with a mean of 65. Aortic pressures were 140/80; left ventricular pressures (LVP), 140/80; mean pulmonary capillary wedge (PCW), pressure 8. Peripheral pulmonary artery angiogram with hand injection demonstrated the "pruned tree" effect found with primary pulmonary hypertension.

The patient failed to respond to a variety of therapeutic agents including 100 percent oxygen, 60 mg of tolazoline IV, 0.4 mg nitroglycerin SL, and prazosin 1 to 5 mg and hydralazine, 25 and 50 mg orally.

The patient was re-admitted to the hospital on September 30, 1981. Results of the physical examination had changed from the previous admission with the development of marked jugular venous distention to the angle of the mandible while sitting upright, hepatomegaly with a span of 18 cm in the right midclavicular line, a palpable splenic tip and 2+ pitting edema in the lower extremities extending to the mid-thighs. The cardiac examination, electrocardiogram and chest x-ray film were unchanged. Radionuclide gated wall motion study showed a markedly enlarged
right ventricle with global hypokinesis and a small normally contracting left ventricle. Right sided cardiac catheterization with a Swan-Ganz thermodilution catheter revealed the following pressures: RAP 12; PAP, 100/60 with a mean of 75; PCWP, 13; and a cardiac output of 2.7 liters per minute.

The patient subsequently received in all seven doses of nifedipine, three of 10 mg and four of 20 mg. A summary of the hemodynamic effects of nifedipine after the first dose of 10 mg and following the first, second and fourth dose of 20 mg are tabulated in Table 1. After the administration of the first 20 mg dose of nifedipine, there was a marked reduction in PAP and pulmonary vascular resistance which, however, became progressively less with each subsequent dose of 20 mg. Following the first dose of nifedipine 20 mg, she became lightheaded and apprehensive, but administration of the subsequent doses of nifedipine was uneventful. Nifedipine also reduced the systemic vascular resistance, but in a dose-related manner with persistent drug effect all doses. The reduction in PAP was not associated with a significant reduction in right atrial pressure eliminating a role for systemic venodilation alone in the etiology of the reduction in pulmonary vascular resistance.

**DISCUSSION**

Nifedipine is a potent blocker of the slow inward calcium current and results in systemic vasodilatation and reflex beta adrenergic activity that blocks its potential negative inotropic, chronotropic and dromotropic effects.

It has been found to lower the pulmonary artery pressure and pulmonary vascular resistance in a number of conditions, including congestive heart failure, hypoxic pulmonary vasoconstriction, and pulmonary hypertension. Their patient responded acutely to the administration of the medication and showed a persistent ability to respond at the time of repeat hemodynamic evaluation after three months of treatment.

The response of our patient to nifedipine was markedly different from that previously reported. She showed a modest reduction in systemic and pulmonary artery pressures after the first 10 mg dose. After nifedipine, 20 mg, however, she experienced a precipitous fall in her pulmonary artery pressure and resistance and a lesser fall in systemic blood pressure and resistance. These effects lasted approximately three hours. In contrast to most other patients' reported responses to nifedipine, whether they had increased pulmonary artery pressures secondary to primary pulmonary hypertension or congestive heart failure, this patient did not increase her cardiac output or her heart rate concomitantly with her fall in systemic and pulmonary pressures. The reason for these differences in response is unclear. It is also noteworthy that the patient failed to show changes from baseline in her hemodynamics after further doses of 10 and 20 mg of nifedipine except for modest increases in her cardiac output. It was decided not to continue the patient on nifedipine because of the greater and excessive effect on systemic vascular resistance.

The results of this trial of nifedipine in the treatment of primary pulmonary hypertension appear to bear out the caveat of Camerini and colleagues that "in primary pulmonary hypertension the response to this, as well as to other vasodilators, can be extremely variable from time to time and from one subject to another." The marked but transient reduction in pulmonary vascular resistance in this patient receiving nifedipine after failure of other agents may suggest a role for calcium antagonists in the treatment of some patients with primary pulmonary hypertension. Further observations are needed to confirm this clinical observation.

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**Disappearance of Exercise-Induced ST-Segment Depression Associated with Transient Left Anterior Hemiblock**

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To our knowledge, this is the first case reported of total disappearance of exercise-induced ST-segment depression in the presence of transient left anterior hemiblock at peak exercise. An unusual paradoxic increase in T-wave amplitude was also noticed. The significant ST-segment displacement reappeared when the transient

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