Table I. Hemodynamic data

	No therapy	*Nifedipine, 6 × 20 mg/day orally for 3 days*	*Nifedipine, 6 × 20 mg/day orally for 6 mo*
Pulmonary artery (mm Hg)			
Systolic	85	50	40
Diastolic	40	25	15
Mean	55	35	30
Pulmonary capillary wedge pressure (mm Hg)			
Mean	10	12	10
Systemic arterial pressure (mm Hg)			
Systolic	110	100	105
Diastolic	65	60	60
Mean	80	75	75
Resistances (dynes-sec-cm^{-5})			
Pulmonary arteriolar	857	312	320
Total systemic	1428	1003	1168
Cardiac output (L/min)	4.2	5.9	5.0
Heart rate (bpm)	80	90	90

beneficial effects of diltiazem were followed by an associated subjective improvement over an 11-month period.[4]

In this report we describe a late-stage patient who sustained a significant improvement with nifedipine over a 6-month period. This improvement was associated with a significant change in hemodynamic data measured repeatedly. A white female patient (age 27 years) was admitted for evaluation of progressive dyspnea and syncope on exertion. At the age of 20 years she was known to have cardiomegaly. Her blood pressure was 115/75 mm Hg. There was clinical evidence of right ventricular hypertrophy. On auscultation a loud pulmonic closure sound was followed by a long early diastolic murmur of pulmonary regurgitation. Routine laboratory examinations were normal. The ECG showed right axis deviation and right ventricular hypertrophy. The chest x-ray showed cardiomegaly and central pulmonary arterial dilatation with distal vessel narrowing. A pulmonary angiogram revealed no signs of pulmonary emboli.

The most important hemodynamic data are shown in Table I. The pulmonary capillary wedge pressure was normal. After treatment with nifedipine, 20 mg six times a day, significant improvement of hemodynamic data was seen after 3 days. During the follow-up period, symptoms markedly improved. No side effects occurred. Repeated catheterization after 6 months of continued oral therapy showed a sustained beneficial effect (Table I). The results in our patient clearly indicate that further evaluation of nifedipine use in primary pulmonary hypertension is warranted.

REFERENCES

1. Braunwald E: Heart disease: A textbook of cardiovascular medicine. Philadelphia, 1980, WB Saunders Co, p 1636.
2. McMurtry IF, Davidson AB, Reeves JT, Grover RF: Inhibition of hypoxic pulmonary vasoconstriction by calcium antagonists in isolated rat lungs. Circ Res **38**:99, 1976.
3. Camerini F, Alberti E, Klugman S, Salvi A: Primary pulmonary hypertension: Effects of nifedipine. Br Heart J **44**:352, 1980.
4. Kambara H, Fujimoto K, Wakabayashi A, Kawai C: Primary pulmonary hypertension: Beneficial therapy with diltiazem. AM HEART J **101**:230, 1981.

Ventricular tachycardia associated with a left ventricular apex sump aneurysm in an adolescent

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Recently we reported a group of children who were surgically treated for congenital heart disease and who developed left ventricular (LV) apical aneurysms after venting of the left ventricle at the time of cardiopulmonary bypass.[1] We speculated that the aneurysms, although small, might provide a focus for ventricular arrhythmias. This report describes a case of potentially life-threatening ventricular arrhythmia developing postoperatively in a patient with residual subaortic stenosis. Location of ven-

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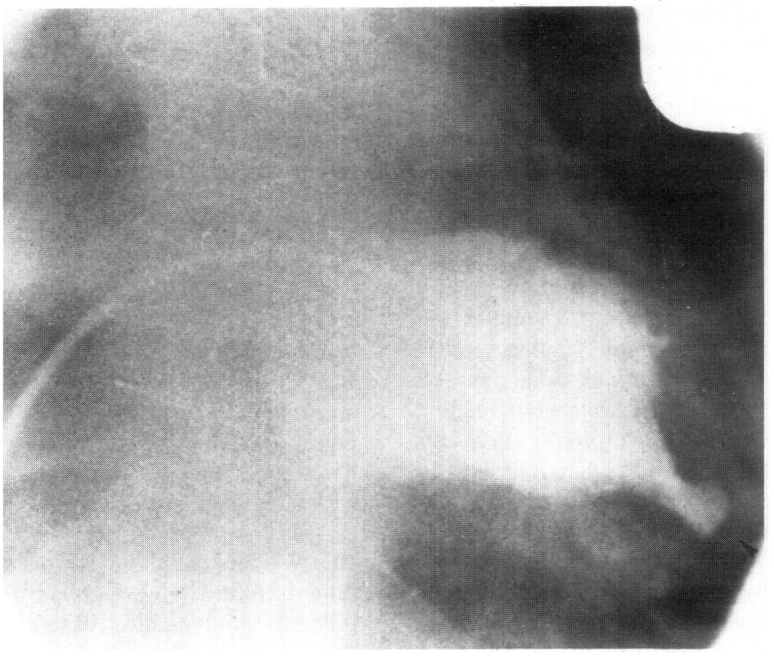

Fig. 1. LV cineangiogram demonstrating small sump aneurysm *(arrow)*.

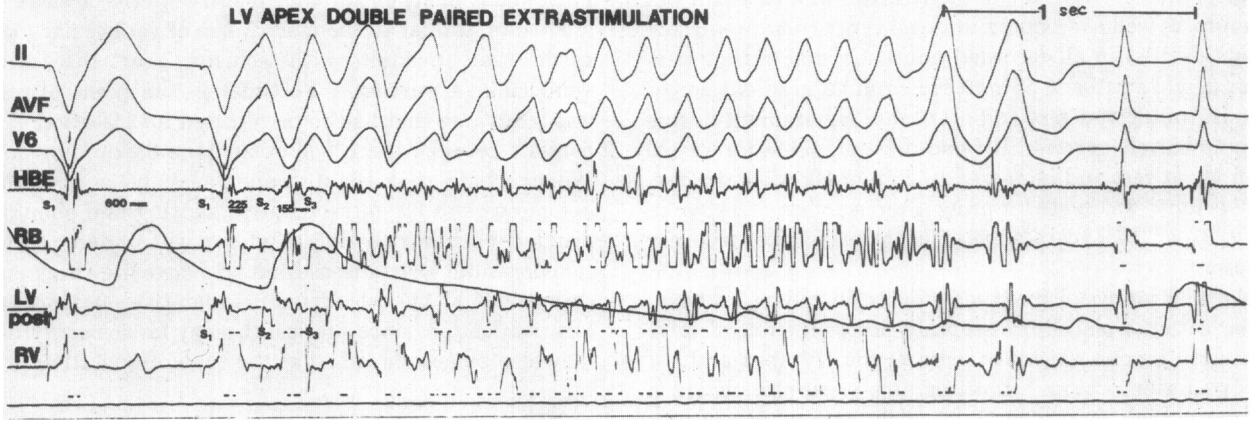

Fig. 2. Tracing of double paired extrastimuli at LV apex resulting in nonsustained ventricular tachycardia (VT). Endocardial mapping demonstrated that the first beat of the VT (fifth beat of tracing, VT) began in the left ventricle (LV posterior). This continued until the last two beats of the VT, when an abrupt slowing, change in the QRS morphology (left bundle branch block from right bundle branch block), and earlier activation of the right ventricle appeared, induced by right ventricular catheter manipulation, terminating the VT. *II, AVF,* and V_6 = Standard ECG leads; *HBE* = His bundle tracing (electrogram); *LV post* = intracardiac tracing in the region of the LV posterior septum; *RB* = intracardiac tracing in region of right bundle and right ventricular septum; *RV* = intracardiac tracing from the right ventricular apex; and S_1, S_2, S_3 = first, second, and third extrastimuli.

tricular arrhythmia to the area of an LV apex aneurysm was confirmed by both preoperative and intraoperative programmed stimulation and electrophysiologic mapping. At the time of reoperation to reduce residual LV hypertension by placement of an conduct from the LV apex to the descending aorta, the apical aneurysm was excised and the arrhythmia ceased.

A 19-year-old man was admitted to the C. S. Mott Children's Hospital at the University of Michigan for syncopal episodes. His first catheterization at another hospital was at 5 years of age and showed a 60 mm Hg peak systolic gradient across the subaortic area. By 10 years of age, symptoms of syncope developed, and a resection of a discrete subaortic membrane was performed

at our institution. The patient did well postoperatively until 14½ years of age, when he again had two episodes of syncope accompanied by decreased exercise tolerance. A second catheterization at that time revealed LV pressure of 208/18 mm Hg and aortic pressure of 140/90 mm Hg. One month later, he underwent resection of subaortic membrane and a hypertrophic muscle band. He was improved following this second operation, and at 17½ years of age he underwent a postoperative catheterization that demonstrated a 50 mm Hg residual LV outflow gradient, trivial aortic insufficiency, and a small apical aneurysm (Fig. 1). He remained without symptoms until 1 month prior to admission, when he had three syncopal episodes, two during light exertion and one while reading. He denied other cardiovascular symptoms. Pertinent physical findings included a thrill, a harsh grade 4/6 systolic ejection murmur at the right upper sternal border radiating to the carotid arteries, and a grade 1/4 diastolic decrescendo murmur at the left lower sternal border. A resting ECG showed LV hypertrophy with nonspecific ST segment and T wave changes in the lateral precordial leads. A graded treadmill exercise test using the Bruce protocol showed no ischemic changes; Lown's grade 1B ventricular ectopy was present in the early stages of exercise.[2] A 24-hour Holter monitor demonstrated one short burst of ventricular tachycardia at 172 beats per minute, as well as frequent unifocal ventricular premature beats (VPBs) in all recorded hours. Cardiac catheterization demonstrated a 55 mm Hg residual gradient across the LV outflow at rest and a 110 mm Hg gradient during supine bicycle exercise. LV end-diastolic pressure was 20 mm Hg at rest and increased to 25 mm Hg with exercise. No ectopy was noted during exercise, but a salvo of ventricular tachycardia was seen during the recovery phase.

An electrophysiologic study was conducted with a hexapolar catheter placed transseptally across the mitral valve and with bipolar electrode pairs at the LV apex and LV midposterior septum.[3] Electrograms were also recorded from the high right atrium, low right atrium, His bundle region, right septal region, and right ventricular apex. Programmed extrastimulation of the right ventricular apex, right ventricular outflow, and LV midposterior septum failed to evoke repetitive beats. During LV apex pacing (S_1S_1) at a basic drive rate of 100 bpm (C_1 = 600 msec), single paired (S_1S_2 = 230 msec) extrastimuli consistently resulted in repetitive beats originating near the apex. Double paired extrastimuli at the LV apex also resulted in single repetitive beats originating at the apex. At an S_1-S_2 interval of 225 msec and an S_2-S_3 interval of 185 msec, ventricular tachycardia at 275 bpm, accompanied by an acute blood pressure decline, was initiated (Fig. 2). Catheter-induced VPBs converted the tachycardia to supraventricular; spontaneous conversion to normal sinus rhythm then followed. One week later the patient underwent construction of a conduit between the LV apex and the descending aorta, and a 20 mm Hancock valved prosthesis was inserted.[4] Intraoperative endocardial mapping confirmed ectopic beats originating on the posterior septal side of the sump aneurysm. Programmed single extrastimulation at this site resulted in three repetitive beats.[3] This area was resected during conduit placement. Programmed extrastimulation at this site failed to evoke repetitive beats following excision. Postoperative Holter monitoring (48 hours) revealed no ventricular ectopy, and the patient has remained asymptomatic since his operation.

At our institution during a 1-year period, 50 children underwent routine cardiac catheterization following open heart surgery in which the LV sump venting technique was used; 32% had small and usually intramural LV apical aneurysms because of intraoperative venting of the LV apex.[1] We were concerned that this aneurysm might become a source of ventricular arrhythmias, particularly with advancing age. This patient represents the first such case. Using programmed stimulation techniques, we were able to locate the site of the origin of ventricular ectopy to the intramural defect caused by the aneurysm. The potential for hemodynamically unstable ventricular arrhythmia was clearly demonstrated, probably correlating with the reported symptoms. Although the precise mechanism of the ventricular tachycardia could not be absolutely determined,[5] the response to extrastimuli suggested that the most likely mechanism was reentry in the area of the aneurysm, similar to the mechanism of ventricular tachycardia seen in adults with ischemic heart disease and ventricular aneurysm.[5,6] Fortunately the point of origin was in an area that had to be resected for insertion of the conduit between the LV apex and the descending aorta. We conclude that the LV apex may be a potentially dangerous site for intraoperative venting and should be avoided whenever possible. Children who undergo surgery for congenital heart disease and who have life-threatening arrhythmia should also have preoperative electrophysiologic evaluation, since surgical therapy for the arrhythmia may also be accomplished at the time of operation.

REFERENCES

1. Weesner KM, Byrum C, Rosenthal A: Left ventricular aneurysms associated with intraoperative venting of the cardiac apex in children. AM HEART J **101**:622, 1981.
2. Lown B: Sudden cardiac death—1978. Circulation **60**:1593, 1979.
3. Josephson MD, Harken AH, Horowitz LN: Endocardial excision: A new surgical technique for the treatment of recurrent ventricular tachycardia. Circulation **60**:1430, 1979.
4. Norman JD, Cooley DA, Hallman GL, Nihill MR: Left ventricular apical-abdominal aortic conduits for left ventricular outflow tract obstruction. Circulation **56**:(suppl II):62, 1977.
5. Rosen MR, Reder RF: Does triggered activity have a role in the genesis of cardiac arrhythmias? Ann Intern Med **94**:794, 1981.
6. Josephson ME, Horowitz LN, Farshidi A, Kastor J: Recurrent sustained ventricular tachycardia. I. Mechanisms. Circulation **57**:431, 1978.