Left ventricular aneurysms associated with intraoperative venting of the cardiac apex in children

Left ventricular (LV) apical aneurysms were observed in 16 of 50 (32%) children (average age 8 years) consecutively catheterized after surgical repair of congenital heart disease. The LV apex was vented by a sump during cardiopulmonary bypass in each. The aneurysms varied in size, but were generally small. Average dimensions were 7.5 × 6.8 mm in the anteroposterior projection and 8.9 × 5.7 mm in the left anterior oblique projection. The LV apex wall was thinner in patients with aneurysms than in age- and lesion-matched controls. All of the LV aneurysm patients were asymptomatic during average follow-up of 4 years. Nevertheless, such aneurysms are anticipated to represent a potential source of cardiovascular complications and, when possible, alternate methods for venting the left ventricle are recommended. (Am Heart J 101:622, 1981.)

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In some institutions, including our own, a sump is placed in the apex of the left ventricle (LV) to vent the chamber during open heart surgery. To determine if the procedure results in any subsequent structural or functional LV abnormality, we reviewed the clinical, hemodynamic and LV cineangiographic findings in 50 consecutive postoperative patients with congenital heart disease. This report describes the incidence, size, and clinical correlates of the observed LV apical aneurysms.

METHODS

Patients. The cineangiograms of all patients catheterized at our institution for postoperative evaluation between July 1, 1978, and June 30, 1979, were reviewed. The medical records including operative report and cardiac catheterization data of all patients with an LV apex abnormality were then obtained and examined. All the surgical procedures were performed utilizing cardiopulmonary bypass with LV venting done through the LV apex. The metalic sump used for venting varied in size from 10 mm to 12 mm. The apical hole was closed with a purse-string suture of Tevdek and the repair usually reinforced by pledgets or deep mattress sutures.

Aneurysm characterization. Dimensions of the LV apical aneurysms were measured from the anterior-posterior (AP) and lateral, or left anterior oblique (LAO), projections of the LV cineangiogram using a magnification grid obtained at the time of cardiac catheterization. The largest dimension (L-axis) and the dimension at a perpendicular axis to the L-axis were recorded. LV apical wall thickness was measured at the thinnest portion of the aneurysm using the same grids. For comparison, apical wall thickness was measured in a similar manner in 26 age-and cardiac lesion-matched controls. Statistical analysis was performed by group t statistic.

RESULTS

Incidence. LV apical aneurysms were observed in 16 of 50 (32%) patients in whom a sump had previously been placed in the LV apex during surgical repair. Mean age at repair of the cardiac lesion was 3.8 years (range 7 months to 9 years) and interval between operation and postoperative cardiac catheterization was 3.7 years (range 9 months to 12.5 years). Cardiac diagnoses in the 16 patients with apical aneurysm included ventricular septal defect (six), tetralogy of Fallot (five), aortic stenosis (two), and miscellaneous (three). No aneurysms or diverticula were noted in any of the patients on preoperative LV cineangiograms. Among the 34 patients without LV apex aneurysm, mean age at repair was 3.6 years (range 3 days to 6.9 years) and follow-up period was 5.6 years (range 1 to 16 years). Distribution of cardiac diagnosis was similar to the group with LV apex aneurysms.

Configuration. The aneurysms varied in shape from

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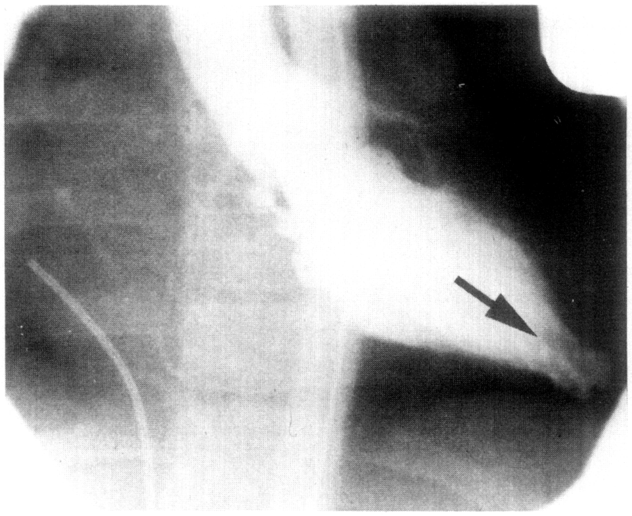

Fig. 1. LV apex sump aneurysm *(arrow)* during systole in an 11-year-old boy, 3 years after surgical repair of a ventricular septal defect.

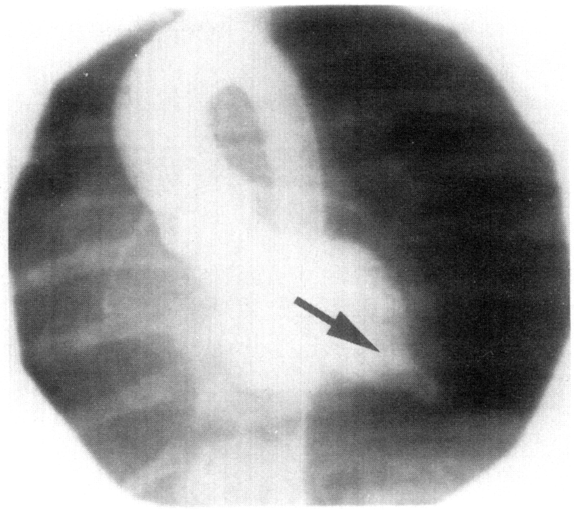

Fig. 2. LV cineangiogram during systole in a 4-year-old child, 2½ years after repair of tetralogy of Fallot. The LV sump aneurysm *(arrow)* measured 6.3 × 6.0 mm.

Table I. Morphology of LV apical sump aneurysms

Age at surgery (yr)	*Age at PO cath (yr)*	*LVAS dimensions* (mm)*	*LVP S/ED (mm Hg)*	*LVAS wall thickness (mm)*
0.6	2.3	6.4 × 5.9/6.5 × 6.2	115/8	5.6
1.2	7.5	7.0 × 7.0/10.8 × 7.4	108/9	5.2
1.5	2.5	3.5 × 2.2/–	99/6	3.8
1.8	3.1	6.4 × 6.0/7.6 × 5.0	101/10	5.0
2.0	3.5	6.9 × 9.3/11.1 × 6.2	106/17	1.3
2.3	3.3	4.6 × 5.4/6.4 × 4.0	127/–	4.0
3.0	10.0	12.2 × 7.8/10.0 × 5.0	110/15	3.5
4.0	5.0	6.4 × 6.0/–	110/8	4.4
4.0	6.0	–	147/14	–
4.0	6.5	8.7 × 3.0/12.4 × 4.8	120/10	2.0
5.0	15.0	–	100/–	–
5.5	17.0	9.9 × 7.8/10.0 × 3.0	110/5	5.8
7.5	11.0	7.8 × 10.0/7.5 × 9.3	111/11	3.0
9.0	10.0	–/8.0 × 5.2	120/7	2.5
12.0	12.0	9.2 × 10.8/–	150/20	4.8

LVAS = left ventricular apical sump aneurysm during systole; LVP = left ventricular pressures; S = peak systole; ED = end-diastole; PO cath = postoperative catheterization.
*AP dimensions/lateral dimensions during systole.

round to oblong fingerlike projections (Figs. 1 to 3). Paradoxical motion during systole was observed in each. Adequate measurements of dimensions and wall thickness of the aneurysm were possible in 14 of 16 patients (Table I). Average size of the aneurysm in the AP, projection was 7.5 × 6.8 mm (range 3.5 × 2.2 to 12.2 × 7.8 mm) and in the lateral or LAO projection it was 8.9 × 5.7 mm (range 6.4 × 6 to 12.4 × 4.8 mm). The thinnest portion of the LV wall at the aneurysm averaged 3.9 mm (range 1.3 to 5.8 mm). In contrast, the thinnest portion of the LV apex in age-and cardiac lesion-matched controls (Table II) was 9.2 mm (range 4.1 to 23.9 mm) ($p < 0.0002$).

LV pressures. At postoperative cardiac catheterization in the 16 patients with LV apical aneurysm (Table I), LV peak systolic pressure averaged 116 mm Hg (range 99 to 150 mm Hg) and simultaneous LV end-diastolic pressure averaged 11 mm Hg (range 5 to 20 mm Hg). In the control group (Table

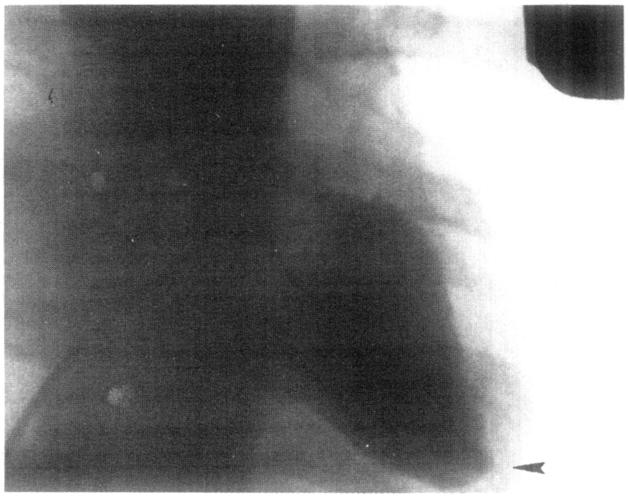

Fig. 3A. Selective LV cineangiogram in a 17-year-old boy, 5 years after aortic valvulotomy. Note absence of aneurysm at the LV apex at end-diastole *(arrow).*

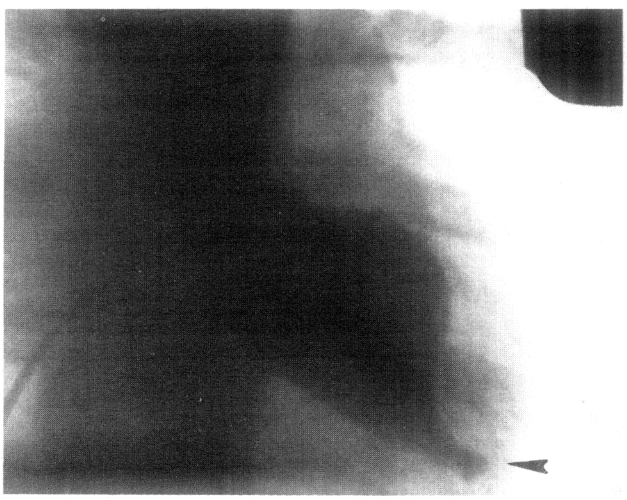

Fig. 3B. LV cineangiogram during systole in the same patient as in Fig. 3A, demonstrating a small sump aneurysm at the apex of the LV *(arrow).*

II), LV peak systolic pressure was 108 mm Hg (range 90 to 255 mm Hg) and LV end-diastolic pressure was 10 mm Hg (range 5 to 18 mm Hg).

Clinical observations. None of the patients had any signs or symptoms related to the aneurysm. No ventricular ectopic beats or myocardial infarction pattern were present on standard electrocardiograms. There were no rhythm disturbances on 24-hour ECG recordings in the six patients in whom the test was performed. M-mode echocardiography was unable to identify or delineate the aneurysms.

DISCUSSION

Causes of LV aneurysms. LV aneurysms may be congenital in origin,[1-3] or may occur as a consequence of myocardial infarction,[4] penetrating chest trauma,[7] or an infectious process.[2] LV aneurysms as a complication of open heart surgery have been reported after mitral valvulotomy, mitral valve replacement, and myotomy performed for relief of idiopathic subaortic stenosis.[2, 5, 6] Large false aneurysms resulting in symptoms have been described following venting of the LV in children with congenital heart disease.[5] However, to our knowledge, there have been no studies on the frequency and characteristics of LV true aneurysms produced by venting the LV with a sump.

Table II. LV apical wall thickness during systole in matched controls

Age group (yr)	*Patients (no.)*	*Range (mm)*	*Mean (mm)*
1- 2	5	4.8- 7.4	6.3
3- 4	6	4.1-11.8	7.7
5- 8	6	7.9-14.8	10.8
9-13	5	6.9-23.9	11.5
13-18	4	8.8-15.2	10.8

Morphology and related features. Our study indicates that 32% of patients in whom an LV sump is used may be expected to develop an aneurysm. The aneurysm is generally small and its size seems to be independent of the type of cardiac lesion, LV pressure, age at initial surgery, or duration of follow-up. The wall of the aneurysm appears to be considerably thinner than the LV apex in matched controls. During systole there is paradoxical motion of the LV apex.

Potential complications. Follow-up of our patients shows no adverse effects from the LV apical aneurysms. However, potential complications which may develop with advancing age are arrhythmias, rupture, thromboembolism, and endocarditis. Progressive enlargement later in life might be anticipated in instances of occurrence of an additional disorder, such as ischemic heart disease. These potential complications are similar to those reported with LV aneurysms from other causes.[3, 4, 7, 8] Several reports have described the surgical removal of an LV apex pseudoaneurysm in patients who became symptomatic.[2, 5, 9]

Pathogenesis. LV sump aneurysms may occur as a result of focal infarction between interlocking sutures or tearing of lightly placed stitches.[6, 9, 10] Localized weakness or scar of the LV apex at the myotomy site may gradually enlarge and thin out. Although we have no pathologic studies of the aneurysms, the morphology on cineangiogram and clinical course of the patients suggest that the aneurysms observed are true rather than false or pseudoaneurysms. Whereas LV false aneurysms may be rapidly progressive, result in symptoms, and require reoperation, these true sump aneurysms are usually small and incidental at postoperative cardiac catheterization. It is anticipated that recognition may be facilitated with two-dimensional echocardiography and or Doppler technique.[9] In postoperative patients with ectopy and a sump aneurysm, electrophysiologic studies may be useful in determining if the aneurysm is the source of such ectopy.

Conclusions. Since the incidence of LV sump aneurysms appears to be relatively high following the use of LV sumps, it would seem advisable to use an alternate method of venting the LV when possible. The left atrium or pulmonary veins may be vented directly if venting is deemed necessary. While no serious complications of true LV sump aneurysms have been reported to date, these aneurysms are a potential source for cardiovascular complications with advancing age.

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