Echocardiographic Evaluation of Ventricular Septal Aneurysms

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SUMMARY The spontaneous closure of ventricular septal defects is frequently associated with septal aneurysm formation. In this paper we discuss the M-mode and two-dimensional echocardiographic findings in nine children with aneurysms of the ventricular septum in association with ventricular septal defects. In all patients the diagnosis was confirmed by angiography. The ventricular septal aneurysms were detected by both M-mode and two-dimensional echocardiography. With M-mode echocardiography, septal aneurysms could be recognized by a pattern of multiple systolic echoes within the right ventricle. With two-dimensional echocardiography, the protrusion of the septal aneurysm into the right ventricle could be seen from several views and the location and the relative size of the aneurysm assessed. Echocardiographic techniques useful in the detection of ventricular septal aneurysms are discussed and examples presented.

RECENT STUDIES of the natural history of ventricular septal defects suggest a continuing rate of spontaneous closure throughout childhood.1-4 Diminution and spontaneous closure of membranous ventricular septal defects through aneurysm formation have been well documented.5-7 The formation of a ventricular septal aneurysm is frequently the prelude to complete closure of the septal defect or partial closure with diminution in the size of left-to-right shunt.5-7 Therefore, if a ventricular septal aneurysm can be detected reliably by a noninvasive technique, repeated cardiac catheterization may be avoided. In this paper we present the M-mode and two-dimensional echocardiographic findings of nine patients with ventricular septal aneurysm in association with ventricular septal defect. The reliability of both echocardiographic techniques is assessed.

Materials and Methods

Nine patients with ventricular septal aneurysm in association with ventricular septal defect were studied by M-mode and two-dimensional echocardiography. They ranged in age from 1 week to 13 years. All patients underwent cardiac catheterization for evaluation of the severity of the ventricular septal defect. Complete cardiac catheterization data, including oximetric shunt determinations and biplane right and left ventricular cineangiograms, were available for all patients. The catheterization and angiographic studies were done within 2 weeks of the echocardiograms. The diagnosis of ventricular septal aneurysm was based on the angiographic demonstration of tissue with distinctive margins protruding from the membranous ventricular septum into the right ventricle with each systole8,9 (fig. 1).

All patients had M-mode and two-dimensional echocardiographic studies before cardiac catheterization. The echocardiograms were interpreted by two observers who did not know the angiographic results. M-mode echocardiograms were performed using an Ekoline 20A ultrasonoscope interfaced with a strip chart recorder. A variety of transducers of differing focal length and frequency appropriate for the patient’s size were used. Recordings were performed with the patient in the left lateral decubitus position, with the transducer positioned in the third or fourth intercostal space at the left sternal border. The ultrasound beam was directed to obtain the left ventricular, mitral valve and aortic root echoes in the usual manner and then angled to the patient’s right to obtain echoes in the plane of the tricuspid valve.10 From this position the transducer was then angled toward the left shoulder to obtain a sweep of the right ventricular outflow tract.

All patients were studied by two-dimensional echocardiography with a prototype of a Varian Associates 32-element, phased-array, wide-angle (80°) sector scanner, now commercially available. The patients were examined in the left lateral decubitus position with the transducer in the second or third intercostal space. Two-dimensional images were obtained in the long-axis, or sagittal, plane by directing the tomographic plane between the apex and base of the heart. Short-axis, or transverse, views were obtained by directing the plane of sweep along a line drawn between the right hip and left shoulder, perpendicular to the long axis of the left ventricle.11 The apical four-chamber view was also obtained in all patients. For this view the transducer was placed over the cardiac apex and angulated in such a way as to visualize simultaneously all four of the cardiac chambers.12 The two-dimensional echocardiographic images were permanently recorded on videotape for future analysis. The illustrations presented here were obtained from Polaroid photographs of stop-action, single-frame, scan images made from the videotape recordings. This photographic process results in a marked reduction in image quality, as well as a loss of the visual appreciation of motion normally present in these phased-array, real-time recordings.
from the septal aneurysm appear anterior to the closed systolic tricuspid valve echo and show a pattern of abrupt systolic motion similar to the systolic anterior motion of the mitral valve seen in patients with idiopathic hypertrophic subaortic stenosis. Figure 2 is a representative M-mode echocardiogram illustrating this pattern. The systolic anterior motion of the ventricular septal aneurysm was found in all nine patients. Often, abrupt systolic anterior motion of the echoes from the septal aneurysm permitted its differentiation from the more gradual sloping systolic motion of the closed tricuspid valve echoes. With respiratory variation in right ventricular cavity size, we observed an increase in the excursion of the septal aneurysm into the right ventricle with inspiration. The multiple echoes from the ventricular septal aneurysm consistently showed a high-frequency, low-amplitude flutter (fig. 3).

Two-dimensional Echocardiography of Ventricular Septal Aneurysms

Long-Axis View of Ventricular Septal Aneurysms

Using the long-axis view of the heart, in seven of nine patients we visualized a saccular protuberance with a rapid flicking motion extending into the right ventricle in systole and realigning with the ventricular septum in diastole. This pattern was felt to represent a ventricular septal aneurysm. Figure 4 is a systolic stop-frame image illustrating the ventricular septal aneurysm extending into the right ventricle from the high ventricular septum, just beneath the aortic valve.

Apex Four-Chamber View of Ventricular Septal Aneurysms

With cranial angulation from the apical four-chamber view, the membranous interventricular septum is visualized. The systolic bulging of the membranous septal aneurysms into the right ventricle was visualized in all nine patients with this modified apex four-chamber view. In diastole the aneurysm realigns with the rest of the septum. This systolic expansion and diastolic realignment of the septal aneurysm was seen in all patients with the apex four-chamber view. Figure 5 illustrates the two-dimensional echocardiographic appearance of a saccular aneurysm of the membranous septum as seen from the apex four chamber view in a 5-year-old child.

Short-Axis View of Ventricular Septal Aneurysms

With the short-axis view of the heart at the level of the aortic valve, the membranous interventricular septum is viewed in cross section along with portions of the right ventricular outflow tract. As the septal aneurysm moved out of this plane in systole, the short-axis view permitted visualization of only four of the nine membranous septal aneurysms bulging into the right ventricular outflow tract. The protrusion of the septal aneurysm into the right ventricle was seen with this view, but the point of origin of the aneurysm from the interventricular septum could not be appreciated.
Table 1. Clinical, Electrocardiographic, and Catheterization Data in Nine Patients with Ventricular Septal Aneurysm

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>ECG</th>
<th>Diagnosis by catheterization</th>
<th>Hemodynamic data</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Recent/Qp/Qs/PA</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Recent/Previous</td>
</tr>
<tr>
<td>JR</td>
<td>1</td>
<td>WNL</td>
<td>VSD, VSA</td>
<td>2.5/26/14,20</td>
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<tr>
<td>DD</td>
<td>13</td>
<td>WNL</td>
<td>VSD, VSA</td>
<td>1.3/20/8,14</td>
</tr>
<tr>
<td>AJ</td>
<td>5</td>
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<td>VSD, VSA</td>
<td>2.7/24/8,15</td>
</tr>
<tr>
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<td>1 week</td>
<td>WNL</td>
<td>VSD, VSA</td>
<td>1.3/45/20,35</td>
</tr>
<tr>
<td>DW</td>
<td>13</td>
<td>LAD, CC loop RVCD, cannot rule out RVH</td>
<td>VSD, VSA</td>
<td>2.2/75/40,50</td>
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<td>PH</td>
<td>12</td>
<td>LAD, BAE, LAH, 1-TGA, VSD, severe PS, VSA</td>
<td>1.0/20/10,14</td>
<td></td>
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<tr>
<td>DP</td>
<td>11</td>
<td>WNL</td>
<td>VSD, PDA, mild PS, VSA</td>
<td>3.3/30/8,15</td>
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<tr>
<td>MM</td>
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<td>RVH</td>
<td>ASD, VSD, VSA, reactive PAH</td>
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<tr>
<td>TW</td>
<td>9</td>
<td>WNL</td>
<td>MS, VSD, VSA, post-op coarctation repair</td>
<td>1.0/44/12,30</td>
</tr>
</tbody>
</table>

Abbreviations: ASD = atrial septal defect; BAE = bialtrial enlargement; CC loop = counterclockwise vector loop; LAD = left-axis deviation; LAH = left anterior hemiblock; 1-TGA = 1-transposition of great arteries; LV = left ventricular; MS = mitral stenosis; PA = pulmonary artery; PAH = pulmonary artery hypertension; PDA = patent ductus arteriosus; PS = pulmonary stenosis; Qp/Qs = pulmonic-to-systemic blood flow ratio; RVCD = right ventricular conduction delay; RVH = right ventricular hypertrophy; VSA = ventricular septal aneurysm; VSD = ventricular septal defect; WNL = within normal limits.

In none of our patients did the septal aneurysm occlude the right ventricular outflow tract.

Discussion

Aneurysms of the membranous interventricular septum are small conical projections of thin membrane which arise from the margins of ventricular septal defects. These aneurysms are generally small and bulge 1-2 cm into the right ventricle in systole. The length of these aneurysms, however, varies greatly, and some have been reported to extend considerable distances into the right ventricle and produce some obstruction to the right ventricular outflow tract. Aneurysms of the membranous ventricular septum were initially believed to be congenital. There is now evidence to suggest that aneurysms may develop in the course of spontaneous partial or complete closure of...
ventricular septal defects, although their exact anatomic basis is controversial. Several series have reported that aneurysm formation is associated with diminution in the functional size of membranous ventricular septal defects, and it appears that the formation of aneurysms of the membranous septum is one mechanism of closure of ventricular septal defect.3, 5, 6

Although anatomic closure of a ventricular septal defect is not assured by the formation of a septal aneurysm, natural history studies of these aneurysms suggest that they are usually associated with a benign asymptomatic course and left-to-right (Qp/Qs) shunts of less than 2.0.7

Our series includes only those patients who were

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**Figure 3.** M-mode echocardiogram from the region of the tricuspid valve illustrating the coarse systolic flutter of the echoes from the ventricular septal aneurysm. The systolic excursion of the aneurysm is not clearly seen in this example. TV = tricuspid valve; VSA = ventricular septal aneurysm.

**Figure 4.** Systolic, stop-frame image of the heart in the long axis. Note the ventricular outflow tract. The aneurysm originates from the membranous interventricular septum just below the aortic root. Ao = aorta; LA = left atrium; LV = left ventricle; MV = mitral valve; RV = right ventricle; Sept = septum; VSA = ventricular septal aneurysm.
catheterized and, therefore, more likely to have hemodynamically significant left-to-right shunts. Four of the nine patients with ventricular septal defect and ventricular septal aneurysm had several associated cardiac lesions (table 1). Of the five patients with isolated ventricular septal defect and ventricular septal aneurysm, only one patient (DW) had significantly elevated pulmonary artery pressures. This child also had Down's Syndrome, which may have contributed to the elevated pulmonary artery pressure. Two of the five patients with isolated ventricular septal defect had evidence on serial cardiac catheterization of decreasing ventricular shunt size and decreasing pulmonary artery pressures accompanied by septal aneurysm formation. Complete closure of a ventricular septal defect by septal aneurysm formation was not observed in our patients. Therefore, we do not know the echocardiographic appearance of the ventricular septal aneurysm once complete closure has occurred. The echocardiographic appearance of the ventricular septal aneurysm in older children (DD, TW) with near complete closure of the ventricular septal defect was similar to that of the younger children in the series.

Echocardiography can be an accurate noninvasive method of detecting ventricular septal aneurysms. Assad-Morell and colleagues reported a large aneurysm of the membranous interventricular septum detected by M-mode echocardiography. Sapire and Black reported a series of seven aneurysms of the membranous ventricular septum that were detected by M-mode echocardiography. In their series echocardiography was 100% accurate, neither missing nor erroneously diagnosing a single case. We believe that this important early work was too optimistic in estimating the sensitivity of M-mode echocardiography as a diagnostic tool in the detection of ventricular septal aneurysms. The previously published echocardiograms of Sapire and Black and our own examples (figs. 2 and 3) show that there could be several pitfalls to accurate interpretation. Lateral resolution errors introduced by the M-mode beam width could easily lead to misinterpretation of echoes from the aortic root, tricuspid valve apparatus or the septum itself, for true septal aneurysms. In our experience the most consistent M-mode finding of a ventricular septal aneurysm is the presence of coarse fluttering echoes with abrupt systolic anterior motion anterior to the tricuspid valve echo. However, we have observed these M-mode patterns in four patients who did not have a ventricular septal aneurysm. In these patients, the systolic motion of the aortic root or chordae tendineae mimicked septal aneurysms; however, the characteristic coarse flutter was not noted. These errors may be minimized if a careful right ventricular outflow tract sweep is performed, because the bulging septal aneurysm is most easily separated from the tricuspid valve in the region of the right ventricular outflow tract. We have also found that an expanded view, as illustrated by figure 3, and rapid paper speed are helpful in differentiating systolic tricuspid valve from the bulging systolic septal aneurysm. A 3.5 MHz non-focused transducer is of further help in accurately delineating these anterior structures. Although the M-mode echocardiogram may misdiagnose ventricular septal aneurysms, it is nonetheless a useful screening technique for their detection.

Two-dimensional echocardiography, because of its real-time format and spatial anatomic display, provides an even more complete noninvasive means of detecting ventricular septal aneurysms. We found two-dimensional echocardiography to be a sensitive technique, enabling us to detect all patients with a ven-
tricular septal aneurysm found at cardiac catheterization. We have observed the systolic bulging of the septum in all cases of proven ventricular septal aneurysms.

We believe, for maximum diagnostic accuracy, multiple two-dimensional echocardiographic views must be used. The thinness and rapid motion of the septal aneurysms and overall cardiac motion may cause a single view to miss the aneurysm. We have found the short-axis and apical four-chamber views to be the most helpful in visualizing the area of the membranous septum. Careful cranial transducer angulation from the long-axis and apical views will allow the best identification of the membranous septum. The location of the aneurysm and the dynamic nature of its bulging into the right ventricle during systole can be appreciated with this technique.

The specificity of two-dimensional echocardiography is difficult to assess from our series. A potential source of error we have encountered is the rare tricuspid pouch lesion. Tricuspid pouches in association with partial endocardial cushion defects may produce a pattern of septal motion indistinguishable on M-mode and two-dimensional echocardiography from membranous septal aneurysms. These pouches, however, can usually be distinguished from septal aneurysms associated with simple ventricular septal defects by virtue of the associated findings of endocardial cushion defect. Figure 6 illustrates a tricuspid valve pouch found in a patient with partial endocardial cushion defect.

With the addition of two-dimensional echocardiography to supplement M-mode techniques, the diagnosis of ventricular septal aneurysms can be accomplished noninvasively, eliminating the need for repeated invasive studies. This technique should be valuable not only in the assessment of the individual patient with ventricular septal defect, but also in the longitudinal study of this defect.

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References

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Effects of Nitroglycerin on Echocardiographic Measurements of Left Ventricular Wall Thickness and Regional Myocardial Performance During Acute Coronary Ischemia

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SUMMARY The effects of nitroglycerin on regional left ventricular performance, assessed by echocardiographic techniques, were investigated in anesthetized, open-chest dogs during acute myocardial ischemia. During transient occlusion of the left anterior descending coronary artery, there was end-diastolic thinning and marked reduction in systolic thickening in the central ischemic zone. Similar changes of lesser degree were noted in the border zone. The normal zone was unaffected. Infusion of nitroglycerin during ischemia in dosages of 2.5–50 μg/kg/min reduced left ventricular end-diastolic pressure without changing the abnormalities of systolic wall thickening. Effects of bolus injections of 20 and 50 μg/kg of nitroglycerin were similar, although this also lowered aortic pressure. In a subgroup of animals in which nitroglycerin infusion was unaccompanied by tachycardia, there was also no evidence that ischemic dysfunction was altered. We conclude that nitroglycerin does not improve regional myocardial performance in acutely ischemic canine myocardium. The decrease in preload is probably entirely due to the peripheral effects of the agent.

NITROGLYCERIN HAS BEEN USED in the treatment of angina pectoris for longer than a century. Recently, however, many clinical and experimental studies have suggested that the agent may be beneficial in the treatment of left ventricular failure due to both acute myocardial infarction1–8 and chronic ischemic heart disease.9–18 These studies have shown that nitroglycerin causes a reduction in left ventricular filling pressure, a decline in arterial pressure of varying degree (depending on route and speed of drug administration), variable changes in cardiac output, and in many of the studies, evidence of improved performance of areas of ischemic left ventricular dysfunction. The mechanism for these changes is not known, but has been attributed to increased venous capacitance, afterload reduction and favorable effects on myocardial oxygen supply/demand ratio.

Our study was designed to test whether nitroglycerin directly affects segmental myocardial performance in a canine myocardial ischemia preparation. Performance was assessed by examining left ventricular systolic and diastolic wall thickness using ultrasound techniques during transient occlusion of the left anterior descending coronary artery. The results show that ischemic segment dysfunction is not affected by nitroglycerin, and suggest that improved overall left ventricular performance in this preparation is the result of the peripheral vascular effects of the agent.

Materials and Methods

The study was performed using 24 mongrel dogs weighing 21.5 ± 0.9 kg (SEM). The animals were anesthetized with a mixture of chloralose 30 mg/kg and urethane 450 mg/kg intravenously, after premedication with morphine sulphate 3 mg/kg given subcutaneously. One hour later, a second dose of morphine (1.5 mg/kg) was injected subcutaneously. The animals were ventilated using a 40% oxygen-air mixture through a Bennett respirator. A #9 NIH catheter was introduced through the femoral artery into the ascending aorta for continuous measurement
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