artery, and it has been suggested that these patients should be treated by bypass surgery.

The mechanism for sudden death is not known. Theories include kinking of the anomalous artery and occlusion by a flap-like closure of the ostium as the aorta expands. However, sudden death, infarction, and ischemia may occur without regard to the course of the anomalous left coronary artery.

In the past six years there have been nine cases of an anomalous left coronary artery from the right sinus of Valsalva or right coronary artery out of 7,893 adult cardiac catheterizations performed at Emory University Hospital and Grady Memorial Hospital. The course of the anomalous artery varied — posterior to the aorta, anterior to the aorta, and anterior to the pulmonary artery. In only one case, posterior to the aorta, did the patient have a previous infarction by ECG and abnormal ventriculogram in the distribution supplied by the anomalous artery in the absence of any demonstrable fixed obstruction.

The entire spectrum of the nonatherosclerotic anomalous left coronary artery, regardless of its course, presents a difficult clinical decision in the patient with chest pain. Chaitman et al. summarize the dilemma best by suggesting lactate studies during pacing, coronary sinus blood flow during exercise and exercise testing of these patients.

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**References**

_The author replies:_
To the Editor:
We have no basic disagreement with Dr. Rossner's letter. In our discussion of the hemodynamic significance of origin of the left coronary artery (LCA) from the right aortic sinus with subsequent passage between the aorta and right ventricular infundibulum, we reviewed the study by Cheitlin et al. which incontrovertibly proved the clinical danger of such anomalies. Like Dr. Rossner, we alluded to the paper by Chaitman et al. which documented the occurrence of myocardial infarction in two patients without coronary atherosclerosis whose LCA also arose from the right aortic sinus but which passed either anterior to the pulmonary artery or posterior to the aorta, rather than between the two great arteries. The case described by Rossner, in which a patient with previous myocardial infarction but no coronary atherosclerosis was found to have a LCA originating from the right aortic sinus and passing behind the aorta, would appear to be a third example of this phenomenon. The etiology of the infarcts in these cases is certainly unclear, since there is no apparent source of compression of these anomalous arteries, as there is when the vessel passes between the aorta and pulmonary artery. The experience is too limited to determine whether these patients have myocardial perfusion deficits related to their anomalies or whether they fall into the larger and well-known but still poorly-understood category of patients with angina or previous myocardial infarction and angiographically "normal" coronary arteries.

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**References**

**Echo Diagnosis of Ruptured Aortic Valve Leaflet**

To the Editor:
We read with interest the paper by Ramirez, Guardiola and Flowers entitled, "Echocardiographic Diagnosis of Ruptured Aortic Valve Leaflet in Bacterial Endocarditis" (Circulation 57: 634, 1978).

Diagnosis of ruptured left coronary cusp was made by the presence of dense, shaggy echoes appearing anteriorly in the aortic valve area during diastole and protruding into the left ventricular outflow tract in an otherwise normal-appearing aortic valve.

In the presence of heavy left and right coronary cusp vegetations, however, dense, shaggy echoes in the middle of the aortic lumen throughout diastole and systole may not be present in this case. Abnormal diastolic echoes in the aortic valve area were recorded not only immediately but also in the midposition (fig. 2). We agree that echocardiographic diagnosis of ruptured aortic valve leaflet in bacterial endocarditis can be made in this case, but not further determination is possible of aortic valve anatomy.

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**References**

_The authors reply:_
To the Editor:
We thank Drs. van Leeuwen and Fast for their interest in our paper.

The presence of heavy left and right coronary cusp vegetations were documented later by the macroscopic examination of the leaflets at the time of surgery. However, they were not demonstrated at the time of the echocardiogram illustrated in figure 1. We agree with Drs. van Leeuwen and Fast that abnormal diastolic echoes are seen in the midportion of the aortic valve area; however, the anatomic portion from which it is recorded corresponds to the transition between the aortic root and the left ventricular outflow
Echo diagnosis of ruptured aortic valve leaflet.
K van Leeuwen and J H Fast

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