Diappearance of Coronary Artery Stenosis Murmur after Aortocoronary Bypass

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A woman with a diastolic coronary artery stenosis murmur which disappeared immediately after aortocoronary bypass surgery is reported. Two months post-operation, the murmur reappeared coincidental with acute anteroseptal myocardial infarction, presumably secondary to occlusion of the bypass graft. Ten months following surgery, when coronary arteriography showed occlusion of the bypass graft and the affected coronary artery, the murmur was no longer present.

A diastolic murmur due to stenosis of a coronary artery is distinctly uncommon,1 and only a handful of cases has been reported.2-5 Of the patients with coronary murmurs described, the murmur has disappeared3,5 or become difficult to hear4 in those having subsequent coronary occlusion and myocardial infarction. Recent advances in coronary artery surgery utilizing saphenous vein grafts have permitted bypass of a localized stenosis, thereby relieving cardiac pain6,8 and improving ventricular performance.9,10 This paper describes the disappearance of a coronary artery diastolic murmur following aortocoronary bypass surgery.

*From the Division of Cardiology, Department of Medicine, University of Oregon Medical School, Portland. Supported by Program Project Grant HL 06336 and Graduate Training Grant HL 05791 of the National Heart and Lung Institute.

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Figure 1. Phonocardiogram, indirect carotid pressure pulse and electrocardiogram (A) before aortocoronary bypass, (B) immediately after bypass, (C) following myocardial infarction two months later, and (D) ten months following surgery. In (A) an early systolic click (SC) is present, as well as systolic (SM) and diastolic (DM) murmurs. In (B) the systolic click and diastolic murmur have disappeared. In (C) the systolic click and diastolic murmur have reappeared. In (D) the systolic click occurs a little later than previously and the diastolic murmur has disappeared.
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enlarged. There was a grade 1/6 systolic ejection murmur in the second right intercostal space at the sternal edge. A grade 2/6 high-pitched early and mid-diastolic murmur, decreasing in intensity during inspiration, was present in the second and third interspaces at the left sternal border. Phonocardiogram (Fig 1A) showed the diastolic murmur beginning about 0.02 sec after the aortic valve closure sound and terminating 0.08 sec before the first heart sound. An early systolic click followed the first heart sound by 0.05 sec. The electrocardiogram displayed ST coving and T inversion in leads V1-V5 indicating myocardial ischemia. No cardiomegaly was evident on the chest x-ray film. Serum creatine phosphokinase (CPK), glutamic oxaloacetic transaminase (SGOT), lactic dehydrogenase (LDH) and hydroxybutyric dehydrogenase (HBDH) levels were within normal limits.

Selective coronary, left ventricular and thoracic aortic angiography showed normal left ventricular size and contractility and no mitral or aortic valvular disease. Injection of contrast agent into the aortic root showed no aortic incompetence, and the sinuses of Valsalva were normal. There was 95 percent stenosis of the anterior descending coronary artery immediately proximal to the diagonal branch (Fig 2), with excellent distal run-off. The circumflex and right coronary arteries were normal.

Because the patient was experiencing intractable crescendo angina, emergency coronary surgery was done. An aortocoronary saphenous vein bypass graft was anastomosed to the anterior descending artery distal to the stenosis. Postoperatively, the patient was relieved of angina, and the diastolic murmur and systolic click were no longer evident (Fig 1B).

Two months after surgery the patient experienced severe substernal chest pain, and an immediate diastolic murmur was heard again together with the early systolic click (Fig 1C). The electrocardiogram showed evidence of acute anteroseptal infarction, and the maximum serum SGOT, LDH and HBDH levels were 126 (normal 8-40), 171 (normal 60-160) and 398 (normal 90-270) units, respectively. Inasmuch as the murmur persisted although there was evidence of myocardial infarction, it was surmised that the vein graft had become occluded.

Ten months postoperation, selective angiography disclosed occlusion of the aortocoronary vein graft. The left anterior descending coronary artery was occluded proximally and filled retrogradely from the right coronary artery. At this time, the diastolic murmur was absent and the systolic click occurred a little later after the first heart sound (0.07 sec) (Fig 1D).

DISCUSSION

It is of interest that all of the reported instances of diastolic murmur due to coronary disease have been characterized by stenosis of the left coronary artery, usually involving the proximal anterior descending branch.2-5 The diastolic timing is consistent with the period of maximum blood flow into the left coronary artery.

The diagnosis of coronary artery stenosis was suspected in the patient reported here because of the coincidence of the murmur with the occurrence of chest pain, the early and mid-diastolic timing, and the absence of signs of aortic valve incompetence and dilatation or paradoxical motion of the heart. Coronary arteriography confirmed the coronary stenosis and ruled out coronary arteriovenous fistula. Aortography excluded aortic valve incompetence, aortic dissection, and ruptured sinus of Valsalva aneurysm. Left ventricular angiography eliminated the possibility of a ventricular aneurysm.

Although disappearance or diminution of a diastolic murmur of coronary stenosis has been described after myocardial infarction,2-5 this report documents the loss of the murmur as a result of coronary artery bypass surgery. A peculiar feature of this case is the reappear-

FIGURE 2. Selective coronary arteriograms. The left coronary system is shown on the left, displaying 95 percent stenosis of the anterior descending artery immediately proximal to the diagonal branch, with excellent distal run-off. The circumflex artery is normal. The normal and large right coronary artery is shown on the right.

CHEST, VOL. 63, NO. 3, MARCH, 1973
Tachy-Brady Syndrome following Open Heart Surgery*

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A 17-year-old patient is discussed who had the tachycardia-bradycardia syndrome following closure of a sinus venous atrial septal defect. Cardiac standstill up to 7.2 seconds long occurred, but placement of a permanent demand pacemaker was avoided. This unique case demonstrates that permanent pacemaker may not always be necessary in the tachycardia-bradycardia syndrome, especially when it occurs in a young postsurgical patient.

The tachycardia-bradycardia (tachy-brady) syndrome is now well described and usually occurs in elderly patients with coronary artery disease.1-4 The treatment usually recommended is the implantation of a permanent demand ventricular pacemaker.1-3,7 This paper describes a unique patient who developed the tachy-brady syndrome following pericardial patch closure of a sinus venous atrial septal defect and was successfully treated without the use of a permanent pacemaker.

Case Report

The patient is a 17-year-old white male who presented to Walter Reed General Hospital Cardiology Clinic for evaluation of a heart murmur and abnormal findings on electrocardiogram and chest roentgenogram. The patient was asymptomatic and participated in strenuous athletics without limitation. Family history was negative for heart disease.

Physical examination revealed the patient to be 74½ inches tall; weight was 185 pounds. His blood pressure was 136/77 mm Hg, pulse 80/minute and regular and respirations 12/minute. The abnormal findings were limited to the cardiovascular examination. The carotid pulsations were normal. The thorax was normal on inspection and there were no abnormal heaves, thrills, or pulsations on palpation. The first heart sound was normal and the second was fixed in its splitting. There was no gallop rhythm. There was a grade 2/6, short, systolic ejection murmur along the left sternal border most intense at the pulmonic area. There was also a diastolic flow rumble at the fifth intercostal space at the left sternal border.

Electrocardiogram revealed complete right bundle branch block pattern with an electrical axis of -30 degrees (Fig. 1). The P waves were inverted in leads 2, 3, and aVF. Chest x-ray film showed the overall heart size to be normal with a density along the right cardiac border suggestive of a pulmonary vein draining into the right atrium. The pulmonary vasculature was prominent. Phonocardiogram confirmed fixed splitting of the second heart sound.

Cardiac catheterization was carried out and documented the presence of an atrial septal defect with mean right atrial pressure of 8 mm Hg, right ventricular pressure 29/0-8.5 and pulmonary artery mean pressure 18. Pulmonic-to-systemic blood flow ratio was 2.8:1. An anomalous right upper lobe

REFERENCES


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