Aortic Stenosis with Coronary Artery Disease: Dilemma of Management

Phillip Dawkins, M.D.*

This patient is a 53-year-old Caucasian man admitted to Methodist Hospital, Indianapolis, on July 15, 1973. He had been retired from his custodial job for two years because of his illness. He was treated since March, 1971 for congestive heart failure secondary to what was thought to be arteriosclerotic heart disease and had been hospitalized twice in 1972, with congestive heart failure. He had always responded to digitalis and diuretics, but remained dyspneic on less than normal activity. His symptoms, which had not changed significantly during the past year, were dyspnea on walking one block, waking two to three times per night with a cough, and a vague chest pain which is substernal, nonradiating, not related to exertion, and not accompanied by shortness of breath. He denied edema, nocturia, or syncopal episodes.

Previous medical history indicated that he had never been hospitalized for a myocardial infarction and had never been told that he had high blood pressure. He does have a history of adult onset diabetes mellitus which was discovered about two years ago. There is no history of rheumatic fever or heart murmur. He had approximately a 20 pack a year smoking history, and he is a nondrinker. His mother died at age 73. The etiology of her death is unknown but she had diabetes mellitus and was on oral hypoglycemics. He has one brother who is living and has had surgery for an abdominal aortic aneurysm. His only surgery was a tonsillectomy. His medications at the time of this admission were digoxin, 0.1 mg QD, furosemide, 40 mg BID, dyazide 1 tablet BID.

Results of physical examination were blood pressure, 100/80; pulse, 110 and regular; temperature, 37.6°C; respiratory rate, 20 per minute.

The chest contained minimal bilateral basilar rales. There was no jugular venous distention. The carotids were equal bilaterally, but were too weak to evaluate the upstroke well.

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Cardiovascular: Apical impulse was in the fifth intercostal space and 9 cm from the MSL. Carotids, radial and femoral pulses were present and equal. There was a palpable gallop and a summation gallop was heard; his rate was too fast to determine splitting of S2; a 1/6 systolic ejection murmur was heard best at the base; a 1/6 blowing pansystolic murmur was noted at the apex.

The abdomen was not distended; the liver was one fingerbreadth below the inferior costal margin and no hepatojugular reflux was present. The extremities were free of edema.

The electrocardiogram showed left bundle branch block. Chest x-ray picture (Fig 1) indicated slight cardiomegaly with mild passive congestive changes in both lung fields. Cardiac fluoroscopy disclosed some calcium in the region to the left ventricular outflow tract, believed present within a coronary artery.

FIGURE 1. Chest x-ray film at the time of hospital admission.
FIGURE 2. Phonocardiogram displays a very soft systolic murmur (SM) seen at the pulmonic area (PA). Reversed splitting of the second sound is noted with a very loud pulmonic component (P₂). The carotid pulse is not particularly revealing.

Phonocardiogram (Fig 2) showed a soft systolic ejection murmur and reversed splitting of the second sound, thought to be attributable to left bundle branch block. The carotid pulse did not appear slowly rising and the ejection time (0.21 second) was normal for rate (120 per minute). A single diastolic gallop was present, believed to be a summation gallop.

Echocardiogram (Fig 3) demonstrated a dilated left ventricle and left atrium and a thickened aortic valve. The thickness of the left ventricular wall was normal (1 cm).

Cardiac catheterization was performed and revealed an elevated left ventricular end-diastolic pressure (30 mm Hg) with a left ventricular systolic pressure of 92. Aortic pressure was 76/64, disclosing a 16 mm Hg systolic pressure gradient across the aortic valve. Pulmonary artery pressure was 60/10. Ventriculography showed marked diminution in left ventricular contraction, with symmetrical involvement. Cardiac output was 2.1 L/min (index 1.1 L/min/M²); arteriovenous O₂ was 13 volumes percent. Aortic valve area was calculated at 0.82 cm². Coronary cineangiography disclosed total obstruction of both the left anterior descending and the right coronary artery near their origins. There was 90 percent obstruction of the obtuse marginal branch of the circumflex artery. No aortic insufficiency was noted.

How would you manage this patient? Is there any place for surgery, and if so, what would it be?

COMMENTS BY ROMAN W. DE SANCTIS, M.D.**

Briefly, this 53-year-old man presents with end stage heart disease manifested primarily by left ventricular failure with total occlusions of the right and left anterior descending coronary arteries and a 90 percent occlusion of the left circumflex marginal coronary artery with a small gradient across the aortic valve. He is on a nearly maximum medical program, and the only real question from a therapeutic standpoint is whether or not he should undergo cardiac surgery, and if so what kind.

I should first like to comment on possible pathophysiologic mechanisms for his congestive failure. He does have severe trivessel coronary artery disease without any history of myocardial infarction, and the left bundle branch block pattern presumably obscures the possibility of diagnosing infarction from the electrocardiogram. The term “ischemic cardiomyopathy” has been used to refer to patients who present with the picture of a diffuse congestive cardiomyopathy with severe coronary artery disease. I would distinguish between this type of left ventricular failure and that which is clearly the result of documented myocardial infarction. Whether or not coronary ischemia without infarction can lead to the picture of cardiomyopathy is not yet clear. There is no doubt that occasional patients present with the picture of a cardiomyopathy and with severe coronary disease without any history of angina or prior myocardial infarct. On the other hand, equally impressive is the large number of patients with extremely severe coronary disease, often involving all three coronary arteries, with excellent left ventricular function. Coronary disease is common, and cardiomyopathy is also relatively frequent. In my own mind, when we see the combination of a patient with the picture of cardiomyopathy and simultaneous severe coronary disease, I am not sure whether we are witnessing a cause-effect relation-

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ship or simply the coincidence of the two diseases. Surely, coexistent coronary disease does not help the patient with cardiomyopathy, but it is still not entirely proved to my satisfaction that coronary disease without infarction leads to cardiomyopathy. The point is of more than academic interest, because from a therapeutic standpoint, if ischemia leads to chronic left ventricular failure, then improvement might be expected from revascularization. The other major point in this particular individual is the question of the severity of his aortic valve disease. He only exhibited a 16 mm Hg systolic gradient across the aortic valve, but with a cardiac index of 1.1 L/min/M², the significance of this gradient is hard to evaluate. In our own unpublished observations on patients with aortic gradients of less than 30 mm Hg in the presence of a cardiac index of less than 1.5 L/min/M², about 50 percent of patients turned out to have significant aortic stenosis, and the other half did not. The smallest gradient that I have ever seen in the patient with end stage aortic valve disease and significant aortic stenosis was 22 mm Hg. On the other hand, even mild to moderate aortic stenosis with a stiff aortic valve can provide a significant obstacle to left ventricular emptying in a heart that is obviously as bad as this gentleman’s is. I will say that the normal left ventricular wall thickness on echocardiography would argue against significant aortic stenosis.

Since the prognosis is so limited in this patient, I would favor surgery accepting an exceedingly high mortality and uncertainty as to the results. At our institution, we would first institute intra-aortic balloon counterpulsation in order to allow for safe induction of anesthesia as well as to assist in trying to get the patient off cardiopulmonary bypass and through the immediate postoperative period. The aortic valve would first be explored in order to assess the severity of aortic stenosis. Even a mild degree of aortic stenosis would justify aortic valve replacement under these circumstances. Our surgeons would then proceed to the construction of triple bypass grafts (I assume the arteries are angiographically bypassable—the protocol is not clear on this point). The risk of surgery would probably be in the range of 50 percent to 60 percent, and the results uncertain. I suppose that alternatively one might consider cardiac transplantation. However, I do not believe transplantation should be performed in any individual in whom there is even a remote possibility that conventional surgical methods might effect an improvement.

Department Editor’s Note
Because of the extremely poor clinical status and apparent poor cardiac contraction noted on left ventricular cineangiogram, the patient was thought to represent a prohibitive surgical risk in our institution. Surgery was, therefore, not performed, and the patient died four months later from myocardial failure. One often encounters end-stage cardiac disease which, although containing one or more potentially operable lesions, obviously has passed into the realm of extremely high surgical risk. Clinical management is then painful to both patient and physician alike. The temptation to intervene surgically under such conditions is usually almost irresistible. In many cases, the choice may be simply stated: either one must allow the patient to die of his disease, or he must perform a procedure, which in itself, would almost certainly be fatal. Our decision to resist the aggressive approach did prove to be fatal. Dr. DeSanctis’s decision may have been better, or may have been just as fatal—earlier. I suspect there were no “correct” decisions in such a case. Ultimately, perhaps, cardiac transplantation will offer a much better alternative, but as yet, this is not generally available.