

**Demonstration of Improved Myocardial Perfusion
following Aortic Implantation of Anomalous
Left Coronary Artery**

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An adult with anomalous origin of the left coronary artery (originating from the pulmonary artery) had implantation of the anomalous vessel into the aorta. Exercise myocardial perfusion imaging with thallium-201, performed before and after the operation, disclosed a marked improvement in anterior-wall Tl-201 uptake. Postoperative isotope ventriculogram showed improved regional contraction in the revascularized anterolateral wall of the left ventricle. Thus, it has been demonstrated that implantation of an anomalous left coronary into the aorta improves regional myocardial perfusion and regional left-ventricular motion.

J Nucl Med 19: 1032-1035, 1978

Anomalous left coronary artery originating from the pulmonary artery is a rare and often lethal congenital lesion. Eighty-eight percent of the patients with this anomaly die within the first year of life (1). Although diverse palliative surgical techniques have been used in the management of anomalous coronaries, only recently has a truly anatomic correction been developed (2), and its initial results seem promising. However, it has yet to be demonstrated that this operation, which is usually performed in patients already having a myocardial infarct, improves myocardial perfusion or segmental ventricular function.

This report describes an adult patient with anomalous left coronary artery originating from the pulmonary artery, who underwent implantation of the left coronary artery into the aorta. Postoperative cardiac imaging studies demonstrated improved myocardial perfusion and segmental ventricular function.

CASE REPORT

The patient is a 28-year-old white woman known to have a heart murmur since age 20 but otherwise having been relatively asymptomatic. Three months before admission, she experienced transient blurred vision and fatigue. As part of the investigation of this problem, an ECG was taken and revealed an old antero-septal myocardial infarct. A subsequent detailed history revealed that over a period of 5 yr she had had occasional episodes of substernal chest discomfort, radiating to the left arm and brought on by strenuous exercise.

Physical examination revealed a well-developed, slender woman in no distress. Vital signs: blood pressure, 115/55 mm Hg; pulse, 88 regular beats per minute; respiration, 16 per minute. Carotid pulses were normal, and jugular venous pressure and venous pulse were normal. The lungs were clear

to percussion and auscultation. Precordial palpation revealed a slightly exaggerated ventricular impulse in the 5th left intercostal space, 1 cm lateral to the midclavicular line. No precordial thrills were felt. The first and second heart sounds were normal and there were no third or fourth heart sounds. A grade 2/6 blowing systolic murmur was heard at the apex and left sternal border, with faint radiation to the left upper back and poor radiation to the left axilla. There was also a grade 2/6 high-pitched, early diastolic, blowing murmur at the left lower sternal border, without respiratory variation. The remainder of the physical examination was normal.

A chest x-ray showed a normal cardiothoracic ratio and straightening of the left upper cardiac border, probably caused by slight prominence of the main pulmonary artery; there was no evidence of chamber enlargement. The electrocardiogram showed an anteroseptal infarction and left atrial abnormality, and an R wave of 1.2 millivolts in aVL, suggesting left ventricular hypertrophy. An echocardiogram showed mild left atrial enlargement (left atrial dimension = 4.4 cm). The interventricular septum moved normally but failed to thicken significantly in systole.

Cardiac catheterization revealed normal right-sided pressures, mild elevation of the mean pulmonary-artery wedge pressure, and a left-ventricular end-diastolic pressure of 16 mm Hg (see Table 1). There was a small and insignificant O₂ step-up (1.5 volumes %) from the superior vena cava to the main pulmonary artery. Increases of this magnitude

Received Nov. 8, 1977; revision accepted March 8, 1978.

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TABLE 1. HEMODYNAMIC RESULTS

Site	Pressures (mm Hg)	O ₂ content (vol %)
SVC	—	13.6
IVC	—	15.4
RA	a = 8, v = 7 (6)	14.8
RV	34/8	14.3
PA	34/12 (18)	15
PAW	a = 16, v = 17 (15)	—
Av	95/60 (80)	—
LV	100/16	17.1

C · O · (Fick) = 5.9 l/min (3.5 l/min·m²).
PVR = 0.5 units; SVR = 12.4 units.
EDV = 157 ml; ESV = 86 ml; SV = 71 ml.
EF = 0.45.

Abbreviations: SVC = superior vena cava; IVC = inferior vena cava; RA = right atrium; RV = right ventricle; PA = pulmonary artery; PAW = pulmonary artery wedge; AO = aorta; LV = left ventricle; CO = cardiac output; PVR = pulmonary vascular resistance (total); SVR = systemic vascular resistance; EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction.

can be observed normally (3). The cardiac output was within normal limits. A left ventriculogram in the right anterior oblique projection showed a normal-sized left ventricle, but there was severe hypokinesis of the anterior wall and apex. Mild mitral regurgitation was observed. The left-ventricular ejection fraction was 45%. In our laboratory, the average ejection fraction in 18 normal young men was 66 ± (s.e.m.) 3%. Selective coronary arteriography showed the right coronary to be the only vessel coming off the aorta. The right coronary was an enormous, tortuous vessel, supplying a large posterior descending artery with multiple, large, collateral vessels retrogradely filling a dilated anterior descending and a small circumflex artery (Fig. 1). The main left coronary originated from the proximal main pulmonary artery; the latter was opacified by contrast flowing from the former.

On May 10, 1977, the patient had implantation of the left coronary artery into the aorta. At operation, the anterior wall of the left ventricle showed two scars, each about 1.4 cm in diameter. There was a continuous thrill palpable over

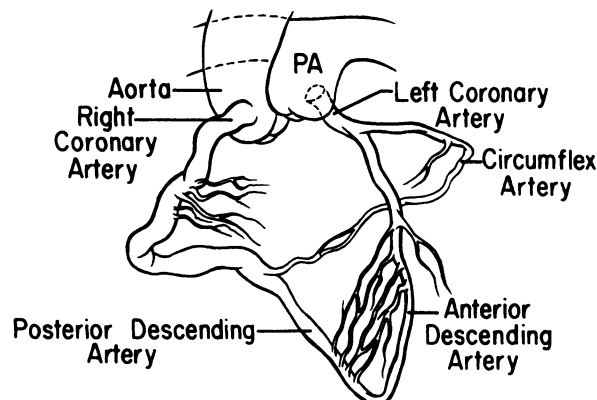


FIG. 1. Composite drawing of anomalous left coronary artery, which filled retrogradely from the right coronary, causing contrast to spill into pulmonary artery. Abbreviation: AO = aorta; RCA = right coronary artery; LCA = left coronary artery; and PA = pulmonary artery.

the left coronary artery and in the pulmonary artery. The left coronary was dissected distally to its origin from the pulmonary artery. An incision was made in the pulmonary artery around the origin of the left coronary, retaining a generous button of the pulmonary arterial wall so that the anastomosis of the coronary artery would be large. The defect in the pulmonary artery was then closed with a patch of pericardium. The aorta was then separated from the pulmonary artery. A longitudinal incision was made in the aorta and the left coronary artery directly anastomosed to it in end-to-end fashion.

The postoperative course was uneventful. When seen 3 mo postoperatively, the patient was asymptomatic. Cardiac auscultation revealed a grade 2/6 early-midsystolic murmur at the apex and left lower sternal border, but the diastolic murmur was no longer heard. Her resting ECG was similar to the preoperative tracing.

Tl-201 myocardial perfusion imaging (MPI). A Tl-201 scintigram was performed the day before the operation and again 3 mo later. The same graded treadmill protocol was used for both. Thallium-201 chloride (1.5 mCi) was injected through a previously placed cannula at the peak of exercise, as determined by severe fatigue, and then flushed with 5 cc of saline. After the injection the patient continued the same level of exercise for 1 additional minute. During the preoperative graded treadmill exercise, the peak heart rate achieved was 150 beats per minute and peak double product (heart rate × systolic blood pressure) was 19,500 beats × mm Hg. The patient had mild precordial and left-arm pain associated with flat 1.5-mm ST depression in V₄ and V₅. The total duration of exercise was 7 min. In the postoperative test the patient was able to exercise for 12 min without symptoms. The peak heart rate achieved was 170 per minute and the peak double product was 24,600 beats × mm Hg. There were no ST abnormalities to suggest ischemia.

Imaging was started 10 min after the injection using a scintillation camera with low-energy general-purpose collimator. Counting was done with two analyzer windows centered at 75 and 167 keV, with window widths of 20% each. Count density in the region of the myocardium was from 1,500 to 2,000 counts per square centimeter. Each study included an anterior view, a 45° left anterior oblique, and a left lateral view. The scanning time for each view ranged from 8 to 12 min (average = 10 min). In the postoperative study, a second scintigram was obtained 3 hr after injection of Tl-201. The camera was connected to a MDS-PAD computer which then processed the images with smoothing, contrast enhancement, and background subtraction. With the digitized data from the scintigram, the background counts were subtracted and the peak count ratio between the abnormal and the normal areas was determined. Figure 2 shows the preoperative and postoperative processed images, after background subtraction of 20% and with smoothing. The preoperative MPI displays a large perfusion defect in the anterior wall and the apex, best seen in the anterior view. In the postoperative scintigram the perfusion abnormalities were much less evident, particularly in the anterior lateral wall of the left ventricle in the anterior projection. Preoperatively, the regional perfusion, as determined by the peak count ratio between abnormal and normal areas, was 0.5 in the anterior view and 0.54 in the left lateral view. Postoperatively, the ratio improved to 0.98 in the anterior and to 0.73 in the left lateral view. Preliminary data in our laboratory suggest that ratios of Tl-201 concentrations in opposite walls of the normal left ventricle are not lower

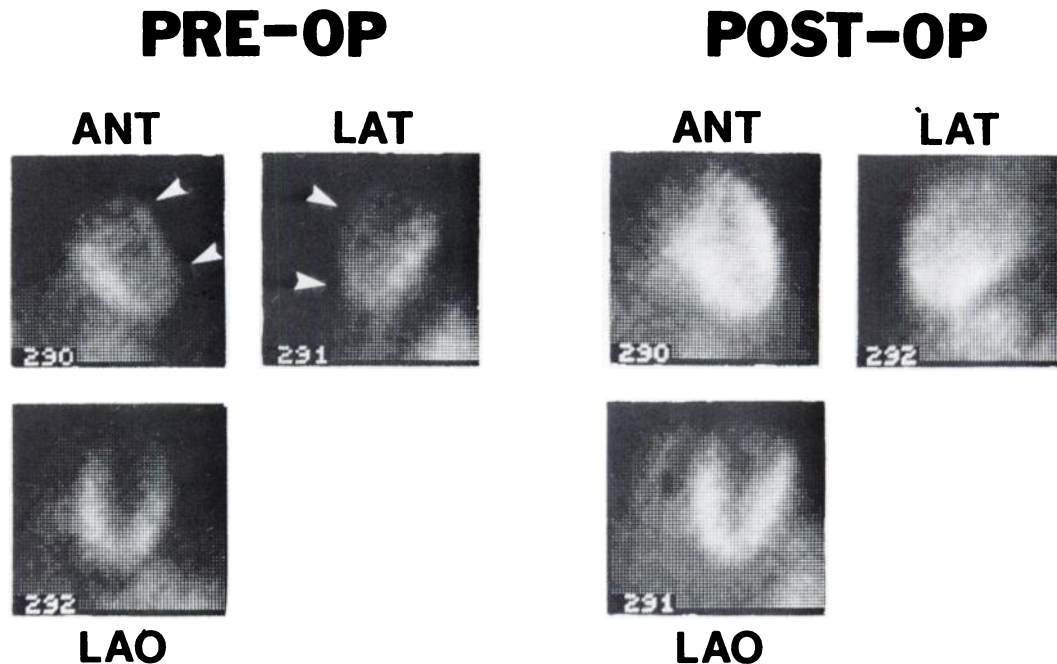


FIG. 2. Preoperative and postoperative thallium-201 scintigrams, computer-processed with background subtraction of 20% and smoothing. In preoperative scans, arrows indicate large perfusion defect in anterolateral wall of left ventricle. Obvious improvement is seen in postoperative images.

than 0.80. Thus, the 0.73 value obtained in the postoperative anterior Tl-201 scintigram is abnormal.

Postoperative isotope ventriculogram. The isotope ventriculograms were performed in the anterior and 45° left anterior oblique views. Imaging was started 10 min after the i.v. injection of 20 mCi of Tc-99m-labeled human serum albumin. The images were collected for a total of 200,000 counts per frame, using a multiple-gate acquisition system triggered by the R wave of the ECG. Left-ventricular ejection fraction was calculated in the left anterior oblique view.

The postoperative isotope ventriculogram revealed normal left and right ventricular size and a normal segmental contraction pattern. The calculated ejection fraction was 58%. In a series of 25 patients studied in our laboratory, the correlation between ejection fractions measured with contrast ventriculography and the isotope ventriculogram was 0.88. The ejection fractions in this population of patients varied from 12 to 73%.

DISCUSSION

With the Tl-201 scintigrams we have demonstrated improved regional myocardial perfusion in our patient after implantation of an anomalous left coronary artery into the aorta. Furthermore, we have also shown improved regional contraction in the re-perfused anterolateral wall of the left ventricle and an overall improvement of left-ventricular function (ejection fraction went from 45% in the preoperative contrast ventriculogram to 58% in the postoperative isotope ventriculogram).

The Tl-201 scintigram has been used in recent years to identify abnormal regional myocardial perfusion in coronary-artery disease (4-7). We have recently used Tl-201 scintigrams in the evaluation of the results of coronary-artery bypass graft surgery and found that 80% of patients have improved regional myocardial perfusion following the operation (8). Demonstration, however, of improved re-

gional myocardial perfusion after correction of anomalous left coronary artery had not been accomplished before.

In order to distinguish between old myocardial infarction with scarring and transient, reversible ischemia, Tl-201 scintigrams must be done at rest and during exercise. Perfusion defects present at rest represent scars, whereas defects appearing only during exercise represent transient, reversible ischemia (5). An alternative way to distinguish transient ischemia from scar is to obtain delayed scintigrams 3 or 4 hr after the exercise and to compare them with scintigrams obtained a few minutes after the exercise. Areas of ischemia will "fill in" in the delayed scintigrams, whereas areas that contain scar tissue will remain unchanged (6). In our patient, a 3-hr delayed scan postoperatively showed a small area of reduced perfusion, unchanged from the early post-exercise scintigram, suggesting that this perfusion defect indicated an area of scar.

It is assumed that the occasional survival to adulthood with an anomalous left coronary artery depends on the development of significant collateral circulation to the left coronary system (1,2,9,10). Since collaterals in this condition seem to function in part as arteriovenous communications, due to low resistance in the left coronary system, it is uncertain how much nutritional blood flow they deliver to the anterolateral wall of the left ventricle.

This study indicates that in our patient, substantial nutritional flow was provided to the anterolateral wall of the left ventricle preoperatively, since this area did contain significant amounts of thallium, taken up by viable, perfused myocardium. Whether this nutritional flow was provided by true collaterals from right to left coronary artery, or merely represented a more extensive distribution of the right coronary branches to the anterolateral wall of the left ventricle, cannot be ascertained from our data.

In summary, we have demonstrated substantial improvement in left-ventricular perfusion, accompanied by improved

regional myocardial contraction, following aortic implantation of an anomalous left coronary artery. In addition, this was accompanied by improved exercise tolerance and reversion of ischemic response on the exercise electrocardiogram.

ACKNOWLEDGMENT

The authors are grateful to Dr. Thomas Zimmerman, who performed the cardiac catheterization, and to Ruth Bonar for secretarial assistance.

Dr. Marcus is the recipient of a Research Career Development Award from the National Heart and Lung Institute, HL-00328.

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