

ruptured chordae tendineae. Pathologic examination revealed scarring and myxomatous degeneration of the mitral valve.

The origin of the systolic murmur, mimicking that of aortic stenosis, is well documented to be due to a regurgitant jet striking the anterosuperior wall of the left atrium adjacent to the aorta, thus producing radiation toward the carotid arteries.³⁻⁵ However, the cause of the diastolic murmur, mimicking aortic regurgitation, is not so well defined. Pulmonic insufficiency is made unlikely by the location of the murmur in the aortic region and by its timing, beginning before the pulmonic closure sound. Mitral flow murmurs tend to be rumbling and mid-diastolic, while the murmur in this case was high-pitched and early diastolic. Given the timing of the diastolic murmur, coincident with the continued early diastolic mitral prolapse, we believe the murmur was due to turbulence created by the flail leaflet describing a rapid movement from its prolapsed position in the left atrium to the open position in the left ventricle, in the face of the onrush of blood from a volume-overloaded left atrium in early diastole.

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Transposition of the great arteries, right aortic arch, coarctation, and isolation of the left subclavian artery: Report of surgical therapy

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To date, only six cases of coarctation with right aortic arch have been reported, all in patients with normally related great vessels. The purpose of this communication

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is to report an infant who had this arch malformation in association with D-transposition of the great arteries who underwent successful surgical treatment utilizing a unique approach.

A newborn male infant developed cyanosis and tachypnea and was transferred to the C. S. Mott Children's Hospital at 6 days of age. Remarkable physical findings included absence of not only the femoral pulses but also the left arm pulse, with a right arm blood pressure of 120 mm Hg systolic. An electrocardiogram revealed right axis deviation, left ventricular hypertrophy, right-sided forces normal for age, and diffuse ST-T changes. Chest x-ray examination disclosed moderate cardiomegaly and pulmonary plethora with a suggestive right aortic arch. Cardiac catheterization with angiography proceeded uneventfully and confirmed the sector scan diagnosis of transposed great vessels with anterior aorta, large muscular ventricular septal defect, hypoplastic right ventricle with overriding tricuspid valve, patent foramen ovale, and discrete coarctation. An ascending aortic angiogram showed a right arch with right descending aorta and a discrete coarctation (Fig. 1, A). The arch gave rise in order to the left common carotid, right common carotid, and right subclavian arteries, followed by the coarctation. Late filling of an isolated left subclavian artery via retrograde left vertebral artery flow was easily appreciated.

The surgical approach to the lesion was a right thoracotomy and repair was accomplished without difficulty by means of a Gore-Tex patch angioplasty rather than a right subclavian patch angioplasty, so as to preserve the right arm blood pressure for postoperative assessment of the surgical result. The pulmonary artery was also banded. Outpatient follow-up has been very satisfactory, with equal right arm and leg blood pressures and a continued absence of the left upper extremity pulse.

The association of coarctation and right aortic arch is extremely rare, with only six reported cases.¹ Our case constitutes the first report of repair of coarctation with right arch from the right chest. The finding of coarctation in a patient with transposition and right aortic arch is also unique.^{2, 3} According to a practical classification of right aortic arch,⁴ which divides them into three types, the added finding of isolation of left subclavian artery constitutes the least common type 3 right arch. While type 2 right arch with aberrant left subclavian artery is not usually associated with congenital heart disease, type 1 with mirror image branching of the brachiocephalic vessels and type 3, as in our patient, are almost always associated with major cardiac malformation. Furthermore, a third feature of the case without precedent is the finding of transposition associated with type 3 right arch.⁵

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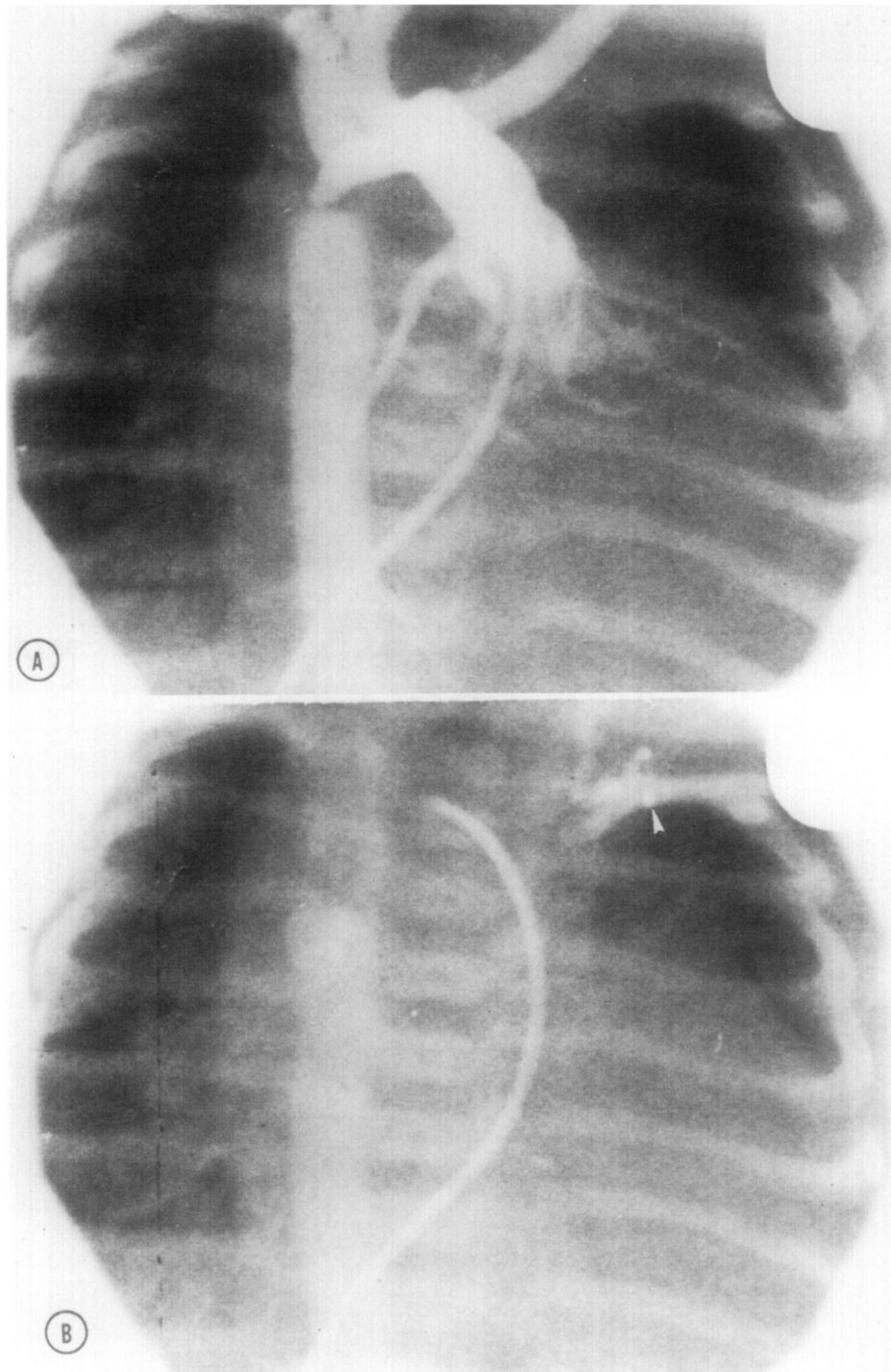


Fig. 1. A, Ascending aortic angiogram shows right aortic arch with right descending aorta and a discrete severe coarctation. **B,** A later frame shows the left subclavian artery (*white arrow*) filling via retrograde flow from the left vertebral artery.

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Exertional ventricular tachycardia

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Only rarely do ventricular tachyarrhythmias requiring electrical defibrillation or cardiopulmonary resuscitation complicate diagnostic stress testing¹⁻⁵ and supervised exercises for physical conditioning.^{5, 6} In our laboratory this complication has occurred once in over 4000 stress tests. The patient was a 56-year-old male with three previous episodes of exertional syncope. The results of a routine work-up, Holter monitor, echocardiogram, EEG, and computerized atrial tomography (CAT) scan of the brain were normal. While undergoing an exercise test the patient developed ventricular tachycardia (VT) (Fig. 1) 30 seconds

into stage II of the Bruce protocol, corresponding to 12% grade at 2.5 MPH. Four countershocks were required to restore normal sinus rhythm (Fig. 2). Subsequently, coronary angiography revealed severe triple-vessel disease and normal left ventriculogram. On electrophysiologic testing, a sustained ventricular arrhythmia could not be induced.

Review of the literature revealed 16 patients who had developed this complication during exercise testing and 22 who developed it during supervised exercises for physical conditioning. All were males and there was a high frequency of coronary disease clinically with two-thirds (27 of 39) having had a myocardial infarction. Previous syncope had occurred in only four patients. Most of the patients who developed ventricular fibrillation during supervised exercises for physical conditioning had ischemic ST segment depression and ventricular ectopic beats in their most recent exercise tests, and may well have been exceeding their prescribed activity when the ventricular arrhythmia occurred. On the other hand, the arrhythmia in patients undergoing stress testing occurred suddenly and unexpectedly. Premature ventricular contractions were absent on the baseline ECG in all but two patients, and the onset was not preceded by ischemic ST segment changes or premonitory arrhythmias. The arrhythmias occurred at relatively low levels of exertion. Of the 14 patients who performed on the treadmill, eight developed the arrhythmia during stage I or less, five during stage II, and one at 45 seconds into stage III. The patient who had a Master's test finished 19 trips. Two other patients tested on the bicycle also developed the arrhythmia at fairly low levels of exercise. The arrhythmias occurred during exercise in about half of the patients (9 of 17) and post-exercise in the other half. In six of eight patients who developed the

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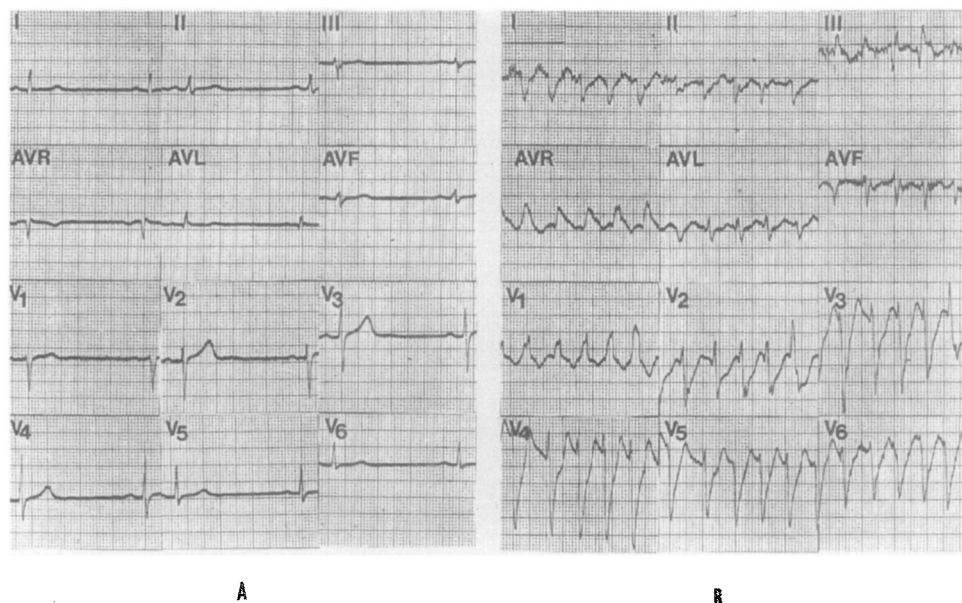


Fig. 1. Electrocardiogram at rest preexercise (A) and at the onset of ventricular tachycardia during stage II of the Bruce protocol (B).