ness and cavity size have not been studied previously in athletes. In this investigation, RVAW was increased in all participants and RV cavity was increased in most. Whether this increase in RVAW was part of the general hypertrophy which occurs in the heart of if this was related to prolonged elevation of pulmonary artery pressure during in-water training for 2-4 hours per day is not known. Clearly this is not an altitude factor alone since the previous normals were from the same community. We cannot explain why the aortic and aortic cusp separation measurements exceeded the 95th percentile of normal in most swimmers.

In summary, the unique finding of this investigation is that children who participate extensively in training programs (swimming) may have echocardiograms which are quantitatively different from youngsters who do not participate in such strenuous activity. Thus, knowledge of athletic participation, at least in swimming, is necessary to interpret quantitative echocardiographic data.

Acknowledgment

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References


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Mid-diastolic Aortic Valve Opening in Severe Acute Aortic Regurgitation

WALT F. WEAVER, M.D., CHARLES S. WILSON, M.D., TERRY ROURKE, RDMS, AND CHRISTOPHER C. CAUDILL, M.D.

SUMMARY A case of severe acute aortic regurgitation is reported. Echocardiographic findings included mid-diastolic opening of the aortic valve, premature closure of the mitral valve, diastolic shuddering of the anterior mitral leaflet, probable demonstration of the flail aortic cusp in the left ventricular outflow tract, and increased left atrial and left ventricular dimensions. Correlation with hemodynamic, angiographic and surgical evidence is made.

HEMODYNAMIC AND ECHOCARDIOGRAPHIC FEATURES of acute aortic regurgitation have been described previously. The sudden volume and pressure overload of the left ventricle in acute aortic regurgitation has been shown to cause premature diastolic closure of the mitral valve in some cases. We recently evaluated a young man with acute severe aortic regurgitation who had not only early diastolic mitral valve preclosure but also mid-diastolic aortic valve opening.

Case Report

A 21-year-old male was in good health until early June 1975, when he had a febrile illness. He consulted his physician and was treated with oral antibiotics of unknown type. Fever and chills continued intermittently for several weeks but finally subsided. Approximately July 20, 1975, the patient noted the onset of exertional dyspnea which progressed over a period of two weeks until he could not carry on his usual activities. He saw his physician again and a cardiac murmur was noted for the first time. The patient was then referred to our institution for cardiac evaluation. Physical examination revealed temperature 98.8 degrees, pulse 70, respirations 20, blood pressure 100/54 mm Hg in both arms. No stigmata of bacterial endocarditis were noted and there was no evidence of Marfan's syndrome. Carotid pulsations had a brisk upstroke with systolic collapse. A heaving cardiac apical impulse was located at the midclavicular line in the sixth intercostal space. There was a palpable apical diastolic thrill. A grade II/VI systolic ejection murmur was loudest at the left sternal border in the second and third intercostal spaces and radiated into both carotid arteries. A grade IV/VI medium frequency early diastolic murmur, heard maximally at the left sternal border, had a crescendo-decrescendo configuration and ended in mid diastole (fig. 1). The liver and spleen were not palpable, and the remainder of the examination was unremarkable. Frequent rectal temperatures taken during his hospitalization were normal. Numerous aerobic, anaerobic, and fungal blood cultures revealed no growth. Standard 12-lead ECG revealed a 42 mm S wave deflection in V2 but

From the Cardiovascular Laboratory, Bryan Memorial Hospital, Lincoln, Nebraska. Address for reprints: WALT F. WEAVER, M.D., Bryan Memorial Hospital, 4848 Summer Street, Lincoln, Nebraska 68506. Received April 12, 1976; revision accepted August 27, 1976.
there was no increase in the lateral precordial R waves or in the limb lead voltage. The chest X-ray revealed moderate cardiomegaly.

Echocardiography

Echocardiography was performed using a Unirad Series C echoscope with an 1856A Honeywell stripchart recorder. A 2.25 MHz transducer was used. It was 13 mm in diameter with a 4–7 cm focal zone. A standard portal was obtained. Initial scans demonstrated an abnormal structure in the left ventricular outflow tract (fig. 2). The left ventricle measured 6.6 cm (left ventricular internal dimension index was 3.4 cm/m²). The mean rate of left ventricular circumferential fiber shortening (Vₚ) was 1.06 cm/sec. The mean posterior wall velocity was 4.33 cm/sec. Normalized posterior wall velocity was 0.66 sec⁻¹ and normalized interventricular septal velocity was 0.45 sec⁻¹. Well defined mitral valve shudder was not evident perhaps due to the mitral valve preclosure. The aortic valve and left ventricular outflow tract required special attention. Three things were remarkable: The abnormal structure in the left ventricular outflow tract appeared in diastole and disappeared in systole; an echo-free space was present in the area of the septum; and the aortic valve was somewhat difficult to visualize. The aortic valve cusps were visualized with unusual medial beam angulation and demonstrated diastolic opening approximately 20 msec following the onset of electrical atrial systole (figs. 2, 3). Transducer placement and beam angulation were manipulated so that, when scanning from the aortic root into the left ventricular cavity, the interventricular septum and the mitral anulus appeared simultaneously. When this was done, the structure in the left ventricular outflow tract in diastole persisted. Yeh et al.⁸ have suggested that in this circumstance, it is reasonable to conclude that the beam is parallel to the aortic anulus and that echoes seen in the left ventricular outflow tract may represent a flail aortic cusp. Further, the echo-free space was consistently demonstrable in the superior portion of the interventricular septum which may represent a cavity in the myocardium. However, dropout of septal echoes is an occasional technical occurrence and cannot be excluded as a possible cause of the echo finding. Phonocardiography (fig. 1) recorded at the apex during simultaneous echocardiographic examination at the lower left sternal border confirmed a systolic ejection murmur and a crescendo-decrescendo early diastolic murmur.

Cardiac Catheterization

Right and left heart hemodynamic and angiographic studies were performed in the usual manner after obtaining informed consent. Pressures (in mm Hg) were as follows: right atrium mean 6 (“a” wave), right ventricle 45/7, pulmonary artery 44/22 (mean 30), pulmonary capillary wedge mean 26 (equal amplitude “a” and “v” waves), left ventricle 95/54, aorta 92/54 (mean 67). Cardiac output by
the thermodilution technique was 5.5 L/min (cardiac index 2.82 L/min/m²). Calculated peripheral vascular resistance was 975 dynes-sec-cm⁻⁵ (normal values of 1130 ± 178). Hydrogen inhalation revealed a left-to-right shunt at the atrial level. Oxygen saturations were as follows: pulmonary artery 65.5%, mid right atrium 69.0%, superior vena cava 59.0%, inferior vena cava 67.0%, aorta 95.8%. Calculations using the Fick method (with assumed oxygen consumption) indicated a pulmonary to systemic flow ratio of 1.1:1.

Aortic root angiography demonstrated a 2 cm irregular outpouching in the region of the left coronary sinus, although it was not clear whether this represented a sinus of Valsalva aneurysm or whether the opening into the cavity was actually inferior to the sinus. Because of severe aortic regurgitation, contrast media appeared nearly simultaneously in the aortic root, the abnormal cavity, and the left ventricle. Selective coronary arteriography was normal.

As the catheter was advanced inferiorly from the aortic root, the tip entered the blind pouch in the region of the left coronary sinus. Selective angiography within this chamber (fig. 4) revealed an irregular cavity from which dye spilled into the left ventricular chamber. The left ventriculogram revealed moderate dilatation of this chamber with no evidence of mitral regurgitation in systole or diastole. Left ventriculography likewise opacified the abnormal cavity, and aortic valve opening was evident during mid diastole. Based on the aforementioned findings, surgical intervention was recommended. The preoperative differential diagnoses included sinus of Valsalva aneurysm with aortic regurgitation and right atrial communication, aorto-left ventricular tunnel, and ruptured aortic cusp secondary to bacterial endocarditis.

**Surgical Findings**

The aortic valve was exposed through an aortotomy. There was no evidence of aorto-left ventricular tunnel or sinus of Valsalva aneurysm. There were friable vegetations on the left coronary cusp which was torn and prolapsed into the left ventricular outflow tract. An opening just beneath the inferior attachment of the left coronary cusp led to a tortuous cavity in the myocardium which was 7.5 mm in diameter and 15 mm long, involving a portion of the interventricular septum and the anterior free wall. Friable necrotic debris was removed from this cavity but no attempt was made to obliterate it or to close its opening. The aortic valve was removed and replaced with a 14-A Lillehei-Kaster tilting disc valve. Because of the evidence of recent, possibly active, infective endocarditis, it was elected not to explore for the small atrial communication. Treatment for bacterial endocarditis with parenteral penicillin and streptomycin had been started one day prior to surgery, and because of the surgical findings, was continued for three weeks postoperatively. The patient’s subsequent course was uneventful. Aerobic, anaerobic, and fungal cultures of the resected valve and the debris from the cavity yielded no growth.

**Postoperative Echocardiography**

Mitral valve motion was normal. The aortic prosthetic valve was not well visualized. The echo-free space in the superior portion of the interventricular septum was not visualized. Paradoxic septal motion was present which has been reported to occur following aortic valve replacement.⁹ Left ventricular internal dimension was 5.5 cm (left ventricular internal dimension index of 2.8 cm/m²), compared to the preoperative measurements of 6.6 cm and 3.4 cm/m², respectively. The structure visualized echocardiographically in the left ventricular outflow tract preoperatively was no longer evident.

**Discussion**

There are at least three mechanisms which could explain diastolic aortic valve opening in severe acute aortic regurgitation. First, as the rapidly rising left ventricular diastolic pressure reaches the falling aortic root pressure, the aortic cusps may simply float into the open position. Secondly, late diastolic elastic recoil of the acutely overdistended left ventricular myocardium might contribute to reopening of the aortic valve. Thirdly, the force of atrial contraction, increasing the end-diastolic left ventricular pressure, might cause the aortic valve to open. This could occur even if the mitral valve had closed prematurely, by forceful downward displacement of the upward-tensed (but still closed) mitral leaflets. The presence of a markedly compliant precapillary arteriolar bed, manifest by a very low peripheral vascular resistance as documented in this case, would obviously complement any of these hypotheses by providing unimpeded diastolic run-off into a very low pressure distal compartment.

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Recollection of Residual Postoperative Shunts by Contrast Echocardiographic Techniques

LILLIAM M. VALDES-CRUZ, M.D., DANIEL R. Pieroni, M.D., JEAN-MICHEL A. ROLAND, M.D., AND JON P. SHEMATEK, M.D.

SUMMARY A bedside echocardiographic technique was used to detect and localize residual intracardiac shunts in 26 patients who had surgical repair of septal defects. Contrast echocardiography was performed through central venous and left atrial monitoring catheters at the same time as cardiogreen dye curves. Indicator dilution confirmed residual atrial defects in ten patients and ventricular defects in five. Contrast echocardiography indicated the presence and level of shunting in all 15 patients. Temporary flow through newly implanted septal patches was detected and differentiated from shunting across a true residual defect. The contrast echocardiographic technique using injections through the central venous and left atrial catheters as described detects and localizes right-to-left and left-to-right shunting. It is a safe and reliable method to evaluate residual intracardiac defects postoperatively.

THE IMMEDIATE POSTOPERATIVE PERIOD for children undergoing surgical repair of congenital heart defects may be complicated by desaturation, persistent congestive heart failure, and/or significant murmurs. It is at this time that the possibility of a residual intracardiac defect must be considered. The purpose of this paper is to report a bedside contrast echocardiographic technique to diagnose intracardiac shunting in the postoperative period.

Materials and Methods

The study included 26 patients, ages three days to 16 years (median age five years), who were selected because their postoperative clinical course suggested a residual defect. The preoperative diagnoses ranged from simpler deformities such as isolated ventricular septal defects to more complicated anomalies such as transposition of the great arteries and atrioventricular canal (table 1). The common denominator of the patient population was that all had septal defects preoperatively, the level and size having been established at cardiac catheterization and at surgery. Indicator dilution curves were performed in the first postoperative day using indocyanine green dye injected into the right atrium and sampled from the radial artery. These were carried out at the bedside in the Intensive Care Unit following the flush technique described by Bloomfield.1 The magnitude and duration of the shunt were determined according to established methods.2

Dye curves were immediately followed by contrast echocardiographic studies performed by two of the authors and interpreted, on a double blind basis, by the other two. These were repeated for 3–5 consecutive days in 13/19 patients who had evidence of a residual defect on initial evaluation. Injections were performed through the central venous and/or left atrial (LA) monitoring catheters placed at the time of surgery; 1–2 cc of the patient's own blood was withdrawn and rapidly reinjected by hand through each catheter. A strip chart echocardiogram was recorded simultaneously with each injection using an EKoline 20A with a Honeywell 1856 recorder. An electrocardiogram and an injection marker were used for timing purposes. The ultrasonic scope was set at the lowest reject and the highest near and coarse gain settings that permitted adequate outline of cardiac structures and recording of the contrast echoes.

A 2.25 MHz, 5 cm focused transducer was used for all examinations which were conducted with the patients in the supine position. Four areas were used for analysis: 1) the aorta, traversing the right ventricular outflow tract, aortic root and left atrium; 2) the mitral, including the right ventricle, interventricular septum and mitral valve at the atrioventricular level; 3) the ventricular, viewing the right ventricle, interventricular septum and left ventricle; and 4) the tricuspid, recording the tricuspid valve within the right ventricular inflow tract. Three to five injections were performed through each of the central lines in all four positions. An injection was acceptable for examination when the side into which it was made became completely opacified with contrast echoes.

A study was considered negative when ultrasonic reflections produced by central venous injections appeared confined to the right heart structures and those created by the
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W F Weaver, C S Wilson, T Rourke and C C Caudill

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