Echocardiography of the Tricuspid Valve in Congenital Left Ventricular-Right Atrial Communication

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SUMMARY
Two patients, ages 14 and 18, with congenital left ventricular-right atrial communication through a defect in the tricuspid valve were studied by echocardiography. Proof was obtained by angiocardiography and surgery in one and ultrasonic contrast injection and angiocardiography in the other. Both presented clinically as uncomplicated ventricular septal defects. Echocardiography consistently demonstrated a high frequency, low amplitude flutter of the tricuspid valve in systole and none in diastole. Following surgical correction of the defect in one patient, there was complete disappearance of the systolic flutter. Systolic flutter has not been observed with tricuspid incompetence or with other forms of ventricular septal defects. Fistulous communication from the aorta to the right atrium just above the tricuspid valve did not demonstrate systolic flutter in one patient studied. Tricuspid valve systolic flutter appears to be caused by the passage of the left ventricular jet of blood into the right atrium through a defect in the tricuspid valve. Echocardiographic study of the tricuspid valve is of value in the recognition of the congenital left ventricular-right atrial communication.

Additional Indexing Words:
Tricuspid valve systolic flutter
Aorto-right atrial fistula
Tricuspid incompetence
Mitral valve systolic flutter

VENTRICULAR SEPTAL DEFECTS which permit communication between the left ventricle and the right atrium are unusual but have recently been recognized with increasing frequency. The diagnosis is rarely made clinically and the true nature of the defect is usually not established until catheterization or surgery. From the surgical point of view, some of these defects can be repaired through the right atrium and ventricu- lotomy can thus be avoided. It would be useful if a noninvasive, nontraumatic technique such as echocardiography could contribute to the diagnosis of this condition. The purpose of this report is to describe characteristic echocardiographic findings in two patients who presented with this entity.

Methods
All echocardiographic examinations were performed using a commercially available echograph (Picker) and a 2 MHz transducer. Continuous records were made on 35 mm film by means of an oscilloscope record camera and a dual-beam oscilloscope operating as a slave. Routine echocardiographic studies of the mitral, aortic and pulmonary valves were obtained. The tricuspid valve was recorded by directing the ultrasonic beam medially and inferiorly from the aortic valve recording position as well as by angling the beam medially and slightly inferiorly from the mitral valve.

Case Reports
Case 1
This 14-year-old asymptomatic white female had been followed since childhood with a murmur suggestive of ventricular septal defect. Physical examination showed a well developed and well nourished child. A systolic thrill was palpable over the precordium in the 5th intercostal space along the left sternal border. A grade III-IV/VI pansystolic murmur was audible over the entire precordium, loudest at the left sternal border. The pulmonary component of the second heart sound was slightly accentuated. The electrocardiogram was consistent with left ventricular hypertrophy. Fluoroscopy showed a moderately enlarged heart with some increase in the pulmonary vascularity.

Echocardiographic recordings of the mitral, aortic and pulmonic valves did not show any abnormalities. The ventricular septum exhibited a normal motion pattern. The tricuspid valve showed high frequency, low amplitude oscillations of the systolic segment, present throughout systole (fig. 1). This was a constant finding over many cardiac cycles. The tricuspid systolic segment did not show any other abnormality. In diastole, no flutter was visible but a coarse undulating movement was occasionally observed. Cardiac catheterization was performed in October 1973. The pulmonary artery pressure was somewhat elevated.

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Tricuspid systolic flutter (case 1). The tricuspid valve echocardiogram shows a low amplitude, high frequency systolic flutter and an undulating movement in diastole. (Average frequency of flutter 65 cycles/sec, average amplitude 3.5 mm.) The structure posterior to the tricuspid valve is probably the interatrial septum. TV = tricuspid valve; PHO = phonocardiogram; ECG = electrocardiogram.

(36/12, mean 20 mmHg). Oximetry showed a step up in the oxygen saturation in the right atrium (82%) with a further step up in the right ventricle (87%) and pulmonary artery (87%). The systemic mixed venous value was 76%. The pulmonic-to-systemic blood flow ratio was 5:1. Left ventricular angiography showed immediate shunting of the contrast material from the left ventricle into the right ventricle and right atrium. Both chambers seemed to fill simultaneously. The outflow tract below the crista appeared to fill first and then the body of the right ventricle, suggesting a membranous septal defect.

Surgery using cardiopulmonary bypass was performed on March 12, 1974. At operation, a systolic thrill could be palpated over the right atrium. There was enlargement of the right ventricle with a coarse systolic thrill over it. The left atrium and the left ventricle were also slightly enlarged. The septal leaflet of the tricuspid valve was fused to some extent to the underlying ventricular septum. The anterior leaflet was free but its distal two thirds was thickened and scarred. The posterior leaflet was essentially normal. The ventricular septal defect was approximately 8 mm at its greatest diameter. There was thick scar tissue about it, probably caused by trauma produced by the jet of blood. The defect was closed with horizontal mattress sutures on teflon pledgets through the right atrium, and ventriculotomy was not performed. The postoperative course of the patient was uneventful except for postpericardiotomy syndrome characterized by a moderately large pericardial effusion.

Postoperative tricuspid valve echograms showed complete absence of the systolic flutter (fig. 2).

Case 2

This 18-year-old asymptomatic white male had been followed since the age of five when a clinical diagnosis of a small ventricular septal defect was made. Physical examination during the present visit to the clinic revealed a well developed young man in no obvious distress. The apex beat was in the mid clavicular line in the 5th intercostal space and was somewhat sustained. The second heart sound showed physiological splitting. A grade III/VI pansystolic murmur was heard best along the left sternal edge in the 3rd and 4th intercostal spaces and it radiated to the apex as well as, to some extent, to the base. No diastolic murmur and no gallops were heard. The echocardiogram, which had been normal so far, showed inverted T waves in leads III and aVF as well as biphasic T waves in the left precordial leads suggestive of left ventricular hypertrophy. Fluoroscopy revealed the heart size to be within normal limits. The pulmonary vascularity was minimally increased and the left ventricle was slightly enlarged.

Echocardiographic studies showed normal mitral and aortic valve records. No abnormality of the ventricular septum was detected. The anterior leaflet of the tricuspid valve was easily recorded. Fragments from the septal leaflet were also observed. The most prominent feature was the high frequency, low amplitude vibrations of the septal segment of the tricuspid valve (fig. 3). There was no diastolic flutter.

At cardiac catheterization, all pressures were found to be normal. Oximetry demonstrated increased oxygen saturation in the right atrium (83%) as compared to the mixed venous blood (80%). There was a further step up in the oxygen saturation in the right ventricle (86%) and pulmonary artery (86%). The pulmonic-to-systemic blood flow ratio was calculated to be 1.9:1. Echocardiographic studies were also performed during cardiac catheterization (fig. 4). Mitral valve recordings obtained during injection of indocyanine green into the left ventricle showed dense echoes in the right ventricle anterior to the ventricular septum confirming the presence of a ventricular septal defect. Indocyanine green was also injected into the left atrium during recording of the atrial septum which was detected by placing the transducer to the right of the sternum in the 4th intercostal space, and directing the ultrasonic beam medially and cephalad. A cloud of echoes appeared in the left atrium in diastole and in the right atrium during the next ventricular systole consistent with a communication between the left ventricle and the right atrium. Absence of initial diastolic
echos in the right atrium suggested an intact atrial septum. Selective left ventricular angiography, in steep LAO, revealed immediate shunting of the contrast material into the right ventricle with some opacification of the right atrium as well. A repeat injection in shallow RAO showed an immediate puff of regurgitant contrast into the right atrium from the left ventricle. It was felt that the patient had a membranous defect below the crista communicating with the right ventricle and with the right atrium, presumably through the septal leaflet of the tricuspid valve.

Discussion

The usefulness of echocardiographic studies of the mitral and aortic valves in the evaluation of various cardiovascular entities is well established. The tricuspid valve, on the other hand, has not been well studied and has had limited clinical usefulness. This is partly because of technical difficulties involved in its recording. It requires steep transducer angulation and hence high sensitivity settings for its detection. Also, considerable time varied gain is needed to minimize the nonstructural signals which clutter the anterior region. Complete recordings of the tricuspid valve are more easily obtained in young subjects and in patients with right heart enlargement. Tricuspid valve studies have been used in the diagnosis of tricuspid stenosis and Ebstein’s disease. Diastolic flutter of the tricuspid valve has been observed in pulmonary incompetence and is probably due to the regurgitant jet abutting against the open leaflet. We have also noted it in large atrial septal defects as well as in transposition of the great vessels especially following balloon septostomy or Blalock-Hanlon procedure, probably related to the increased flow across the valve. Systolic abnormalities have also been recorded in our laboratory (fig. 5). Sagging as well as straight downhill slopes of the tricuspid systolic segments have been demonstrated in patients with right heart dilatation and tricuspid incompetence. A sharp, mid to late systolic posterior displacement of the valve similar to that observed in the mitral valve echograms in patients with prolapsing mitral valves has also been observed recently by us and may represent tricuspid valve prolapse (fig. 5).

The mechanism for the production of the tricuspid valve systolic flutter noted in the two patients in the present study is not clear. It has not been reported previously and we have not observed it in any other condition. It may be caused by the passage of the left ventricular jet of blood into the right atrium through a
deficiency in the tricuspid valve, the margins of which are deformed by fibrosis and fused to some extent to the membranous septal defect. Following surgical correction of the defect in one patient (case 1), there was complete disappearance of the systolic flutter. We specifically studied the tricuspid valve in patients with atrial septal defect (20 cases), ventricular septal defect (14 cases, 4 with proven membranous defects not associated with shunting into the right atrium), pulmonary hypertension and tricuspid incompetence of various etiologies (21 cases), and in 12 normal children, but failed to find evidence of systolic flutter. Fistulous communication from the aorta to the right atrium just above the tricuspid valve (confirmed at surgery) did not demonstrate tricuspid valve systolic flutter in one patient studied by us. Systolic flutter would thus not be expected to occur in the supravalvar type of left ventricular-right atrial communication in which the shunt opens into the right atrium superior to the tricuspid valve.9 We have so far not studied patients with cleft tricuspid valves by ultrasound. Four patients having partial atrioventricular canal defects and cleft but not obviously deformed mitral valves at surgery did not show echocardiographic evidence of mitral systolic flutter. It would, therefore, appear unlikely that the presence of a simple cleft would result in systolic oscillations of an atrioventricular valve. Thus although it is conceivable that patients with endocardial cushion defects and cleft tricuspid valves may exhibit the same echocardiographic findings, absence of mitral valve systolic flutter in patients with cleft mitral valves makes this unlikely. We have to date observed mitral systolic flutter in only three patients, all of whom had echocardiographic findings of mitral valve prolapse and previous subacute bacterial endocarditis (fig. 6). Thus it would appear reasonable to suggest that the requirements for systolic flutter of the tricuspid or the mitral valve include flow through an irregular defect and sufficient local flexibility to produce fluttering.

Echocardiographic studies during cardiac catheterization utilizing indocyanine green as a contrast agent have been used to validate the anatomy of various structures observed and to verify abnormal flows occurring with shunts or valvar regurgitation.4 In case 2, ultrasonic recordings obtained during injection of the green dye into the left side of the heart demonstrated evidence of shunting into the right ventricle and into the right atrium together with an intact atrial septum.

**Figure 5**

Other systolic abnormalities of the tricuspid valve. Upper panel: Tricuspid valve echocardiogram in a patient with tricuspid incompetence due to right heart dilatation. The systolic segments show straight downhill slopes without obvious flutter. Lower panel: Tricuspid valve echocardiogram from a patient with typical echocardiographic features of mitral valve prolapse. The tricuspid valve executes a sharp posterior movement in midsystole raising the possibility of associated tricuspid valve prolapse. TV = tricuspid valve; PHO = phonocardiogram; ECG = electrocardiogram.

**Figure 6**

Mitral valve systolic flutter. A magnified, high speed (375 mm/sec) recording of the mitral valve in a patient with mitral valve prolapse who has had subacute bacterial endocarditis. The appearance of the flutter coincides with the onset of the valve prolapse. (Average frequency of flutter 90 cycles/sec, average amplitude 4.5 mm.) MV = mitral valve; PHO = phonocardiogram; S1 = first heart sound; ECG = electrocardiogram.

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Although our experience is limited, it appears that systolic flutter of the tricuspid valve is a rare echocardiographic finding which is of diagnostic value in cases of congenital left ventricular-right atrial communication associated with a defect involving the tricuspid valve. Systolic segments of the tricuspid valve should be carefully studied for the presence of tricuspid valve flutter in all patients with clinical evidence of ventricular septal defect to exclude this anomaly.

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References


Figure 7

Schematic representation of the type of left ventricular-right atrial communication observed in the present study.

The left ventricle is directly related to the right atrium over an area of the membranous ventricular septum which extends superior to the septal attachment of the tricuspid valve. Two major types of communications have been demonstrated between the left ventricle and the right atrium. In the first, the defect in the membranous portion of the ventricular septum opens directly into the right atrium above the tricuspid valve. In the second, the defect which lies lower in the membranous part of the septum opens first into the right ventricle behind the septal leaflet of the tricuspid valve and then into the right atrium through a second deficiency in the tricuspid valve, usually the septal leaflet, which has, in turn, become partially adherent to the septum (fig. 7). The shunt may thus be either solely from the left ventricle to the right atrium or at both ventricular and atrial levels. Both the cases studied by us appear to belong to the latter group.

The incidence of left ventricular-right atrial communications has been estimated to be about 0.08% of all congenital lesions. With increasing awareness of this entity and with more refined and precise methods of cardiac investigation by means of cardiac catheterization and angiocardiography, further cases have been diagnosed preoperatively and have been successfully operated. Also, acquired defects have been reported with increasing frequency and are due to a multitude of etiological factors including bacterial endocarditis, chest trauma and following replacement of a heavily calcified mitral valve.
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