CASE REPORTS

Myocardial Dysfunction During Paroxysmal Supraventricular Tachycardia

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SUMMARY Paroxysmal supraventricular tachycardia (PSVT) is associated with altered hemodynamics. We describe the echocardiographic features of myocardial dysfunction during a prolonged episode of PSVT in an 11-year-old male. The abnormality of the phases of cardiac activity presented as a markedly prolonged left ventricular systole (320 msec) and isovolumic relaxation phase (220 msec) and a shortened and delayed diastolic filling period (140 msec). These abnormalities reverted to normal immediately after spontaneous conversion to sinus rhythm. Propranolol, which was used to prevent PSVT in this child, may have been involved in the mechanism of the altered mechanical events.

ALTERED HEMODYNAMICS during paroxysmal supraventricular tachycardia (PSVT) have been described.1-6 The proposed mechanisms for these hemodynamic alterations include a decreased diastolic filling period, atrioventricular (AV) valve regurgitation and changes in the timing of AV contraction relationships.6

We describe a new mechanism for altered hemodynamics in a patient with AV reentrant PSVT. An alteration of the normal cardiac mechanical events caused the ventricular dysfunction.

Case Report

An 11-year-old black male was admitted to the emergency room with a complaint of fatigue and dull, aching chest pain of 36 hours duration. He had a history of recurrent episodes of PSVT. Four years before admission he underwent cardiac catheterization and electrophysiologic studies. The tachycardia was found to be AV reentrant PSVT, with retrograde conduction via a concealed left lateral Kent bundle.6 Left- and right-heart pressures and oxygen saturations were normal.

Current medications included propranolol, 200 mg/day, and digoxin, 0.25 mg/day. The blood pressure was 130/80 mm Hg and the pulse rate was 90 beats/min. On cardiac examination the only abnormal finding was an intermittent third heart sound. The apical rate was also 90 beats/min. A 12-lead ECG showed a supraventricular tachycardia at a rate of 180 beats/min and minor nonspecific ST-T-wave changes (fig. 1).

The patient was transferred to the noninvasive laboratory for further evaluation. Echocardiography was performed using an Advanced Technology Laboratories Mark III Echocardiograph system. Phonoangiography and carotid pulse recordings were performed with an Irex Continutrace 100 recorder at a high-frequency setting (100 Hz). The phonocardiogram confirmed that for every two electrical events (QRS complex), a single effective mechanical event (ejection) occurred (fig. 2).

Left Ventricular Events

The motion of the posterior wall of the left ventricle was abnormal (fig. 3). Alternating QRS complexes were labeled A, B, A, B. There was a periodicity of anterior motion of approximately 90 beats/min. This anterior motion of the posterior wall began shortly after QRS “A” with a slow rate of rise (1.5 cm/sec), reaching a peak at QRS “B.” The total amplitude of left ventricular (LV) motion was reduced. The anterior position of the LV posterior wall was maintained until relaxation occurred, when the posterior displacement of this wall was observed. Thus, the LV posterior wall had a single atypical motion for each two QRS complexes (“A” and “B”) and there was no apparent relaxation for the expected diastole of QRS cycle “A.” As indicated from the positions of the first and second heart sounds recorded on the phonocardiogram, ejection occurred between QRS complexes “A” and “B,” with aortic valve closure just before QRS “B” (fig. 2).

The rate of motion of the interventricular septum (IVS) was 180 beats/min and entirely synchronous with the expected electrical systole and diastole, although alternation in the diastolic positions of the IVS was apparent (fig. 3A).

Echocardiography of the mitral valve revealed that it opened once for every two QRS complexes, with an abortive anterior motion visible between the two actual openings (fig. 3A). The duration of the diastolic filling period was 140 msec, which is the expected duration for a heart rate of 180 beats/min, but...
markedly shortened for a rate of 90 beats/min (400 msec).

An M-mode echocardiographic recording of the aortic valve could not be obtained. Two-dimensional imaging demonstrated the rate of aortic valve opening to be 90 beats/min. The valve opening occurred only with alternate QRS complexes.

Systolic time intervals were calculated using the ECG, phono- and echocardiographic recordings. The carotid pulse tracing was of poor quality and only approximate measurements could be obtained. LV systole (QS₂) was 320 msec, corrected for a rate of 180 beats/min to 698 msec (QS₂I), which is markedly prolonged (546 ± 28 msec). The pre-ejection period (PEP) and LV ejection time (LVET) were approximately 130 and 190 msec, both markedly prolonged for a heart rate of 180 beats/min.

Right Ventricular Events

The right ventricular (RV) wall motion differed from that of the left ventricle. There was an apparent rate of motion of the RV free wall (RVFW) at 180 beats/min (fig. 4). However, the amplitudes and rate of motion of the RVFW were alternating with each QRS. At each QRS "A," the amplitude and rate of motion of the RVFW was greater than that at each QRS "B."

The motion of the tricuspid valve (TV) differed significantly from that of the mitral valve. The TV showed diastolic openings at a rate of 180 beats/min (fig. 5). Again, however, there was an alternating pattern of valve motion. The TV opening occurring before each QRS "B" displayed varying amplitude and duration and showed delayed closure in relation to QRS "B." A phonocardiogram performed at the fourth left interspace recorded a sound of varying intensity that correlated in time with the "extra" tricuspid closure at QRS "B" (figs. 5 and 6). There was no mitral or aortic event occurring at this time, so it appears that this was a first heart sound emanating from tricuspid closure. This sound was apparently what was heard at the apex on the initial physical examination and erroneously called third heart sound.

The pulmonic valve was not recorded on the echocardiogram, so we do not know if a right-sided ejection followed this "extra" diastole. However, no

Figure 1. Twelve-lead ECG taken during paroxysmal supraventricular tachycardia.

Figure 2. Phonocardiogram at the second right intercostal space (2 RICS) and the third left intercostal space (3 LICS) and electrocardiographic lead II. S₁ = first heart sound; S₂ = second heart sound. The alternate QRS complexes are labeled A, B, A, B. The S₁ and S₂ are present for only alternate QRS complexes. The S₅ occurs late, coinciding with QRS "B," and is a manifestation of the prolonged left ventricular ejection time.
MYOCARDIAL DYSFUNCTION DURING PSVT/Warnowicz et al.

**Figure 3.** Echocardiograms of the right ventricular free wall (RVFW), interventricular septum (IVS), mitral valve (MV), left ventricle (LV) and left ventricular posterior wall (LVPW). Electrocardiographic lead II is displayed at the bottom. (A) Echocardiographic findings during paroxysmal supraventricular tachycardia (PSVT). Alternate QRS complexes are labeled A, B, A, B. The RVFW has alternating positions of diastolic stretch. Each large arrow demonstrates the cycle with a greater degree of motion. This also corresponds to greater degrees of diastolic motion in the IVS and MV. The small arrows indicate the coinciding “weak” diastole of the right ventricle, IVS and MV. The MV displays only an abortive opening motion at the small arrows. The LVPW shows a slow rate of systolic rise at each QRS “A” and maintains an anterior position until posterior motion occurs at the subsequent QRS “A.” (B) Echocardiographic findings during sinus rhythm. Note normalization of septal, posterior wall and mitral valve motion immediately after conversion of PSVT to sinus rhythm.

sound consistent with pulmonary closure was recorded for this cycle.

The rhythm converted spontaneously to normal sinus rhythm at a rate of 83 beats/min during the insertion of an i.v. catheter. The blood pressure remained unchanged. The echocardiographic study was repeated. Normal relationship of electrical to mechanical events were demonstrated. The movement of the LV posterior wall and the mitral valve echocardiogram became normal (fig. 3B). Systolic time intervals were calculated from the aortic valve recorded on the M-mode echocardiogram. The corrected PEP was 164 msec (prolonged) and the corrected LVET was 391 msec (normal). The duration of isovolumic relaxation phase was 40 msec and the filling period was 390 msec (normal). RV and TV motion were normal.

**Discussion**

These events describe an unusual response of an apparently normal myocardium to prolonged AV reentrant tachycardia in a child who received large doses of propranolol. The LV mechanical events during the tachycardia are illustrated in figure 7.

At each QRS “A” the total duration of systole (PEP and LVET) was markedly prolonged, occupying the entire RR interval. Ventricular contraction was preceded by a prolonged PEP. This prolongation
of PEP may be explained by a depression of myocardial function by the prolonged episode of tachycardia or it may be the result of β blockade. The latter explanation is more likely because the PEP and LVET remained prolonged after conversion to sinus rhythm. The aortic valve closed just before QRS "B." Diastolic filling should have occurred at this point; however, only an abortive anterior motion of the mitral valve was seen on the M-mode echocardiogram and no actual opening of the valve was seen during two-dimensional real-time imaging. This phase of the cardiac cycle is labeled "isovolumic relaxation" because it followed the LV ejection period and preceded ventricular filling, i.e., during this phase of the cardiac cycle both aortic and mitral valves remained closed. However, the motions of the posterior and septal LV walls were discordant during this period. The IVS displayed an initial posterior motion but subsequently was displaced in an anterior direction while the posterior wall remained in an anterior position (fig. 2). The total duration of the isovolumic relaxation period was 240 msec, which is markedly prolonged. After this prolonged isovolumic relaxation phase, ventricular filling was observed. The duration of ventricular filling was markedly shortened, lasting for 140 msec.

The RV systolic time intervals could not be accurately determined because the pulmonary valve motion could not be recorded. However, based on the phonocardiographic findings, it was apparent that the right ventricle was also ejecting blood at a rate of 90 beats/min (fig. 2). In addition, the pulmonary artery posterior wall showed a gross periodicity of 90 beats/min (fig. 5). The posterior RV wall motion at QRS "A" had a slow rate of rise and peaked late. Qualitatively...

**Figure 4.** Echocardiogram of the right ventricular free wall (RVFW) demonstrating the alternating diastolic positions. Abbreviations are as in figure 2. Large arrows show the greatest anterior excursion (relaxation). Small arrows show the "weak" and delayed relaxation.

**Figure 5.** Echocardiogram of the tricuspid valve (TV) showing the "extra" tricuspid valve opening at each QRS "B" and the delayed closure. Arrows demonstrate the varying amplitudes of excursion of the TV. The pulmonary artery posterior wall (PAPW) is seen moving posteriorly with a periodicity of motion at 90 beats/min.
MYOCARDIAL DYSFUNCTION DURING PSVT/Warnowicz et al. 425

Figure 6. Phonocardiogram at the fourth left intercostal space (4 LICS) and second left intercostal space (2 LICS). Abbreviations are as in Figure 1. An extra S₁ is recorded consistently at 4 LICS with varying intensity. It is recorded only once at 2 LICS (arrow). The timing of this "extra" S₁ corresponds to the extra tricuspid closure.

Figure 7. Cardiac mechanical events during paroxysmal supraventricular tachycardia at a cycle length of 320 msec. (A) Normal response. Systolic time intervals were calculated based upon published data.7 (B) The cardiac mechanical events in our patient. Systolic time intervals were calculated based upon the ECG, phonocardiographic, and echocardiographic data shown in figures 2 and 3.
Altered hemodynamic findings during PSVT have been described. The cardiac output is lowered significantly compared with sinus tachycardia. Goldreyer et al. suggested that there must be specific changes other than rate to account for the alteration in hemodynamics. Among those considered were decreased ventricular filling due to shortening of diastole, AV valve regurgitation, and changes in AV contraction relationships. Their studies suggested that the abnormal AV contraction relationships in AV reentrant tachycardia was the most significant difference from sinus tachycardia leading to impaired ventricular filling.

In the case presented, a different etiology for impaired ventricular function was defined. An alteration of cardiac mechanical events, manifested by an asynchronous myocardial muscle response, appeared to be responsible for the ventricular dysfunction. The abnormally prolonged LV systole, following QRS "A", encroached upon the ventricular filling period. No blood had entered the ventricle, so the next contraction, after QRS "B", failed to open the aortic valve and no ejection occurred. This beat was mechanically ineffectual and thus of short duration, allowing for subsequent relaxation and adequate ventricular filling.

The abnormality in this case bears some relationship to pulsus alternans. In pulsus alternans, there is a strong and a weak beat, analogous to the alternating present and absent beats of our patient. The exact mechanism for pulsus alternans is not certain. Some investigators believe it involves either a primary muscle abnormality or to be secondary to impaired degrees of diastolic stretch (Starling mechanism). In pulsus alternans, the LVET for the strong beat is prolonged; however, the total time for systole (PEP and LVET) remains constant for both the strong and weak beats. Therefore, variations in diastolic filling seen on the mitral echocardiogram cannot be attributed to the delayed onset of diastole. These variations are probably secondary to alteration in ventricular relaxation within alternate cycles.

While the findings in patients with pulsus alternans and in our patient may be qualitatively similar, there are important quantitative differences. Our case demonstrates a prolonged systole, a slow relaxation manifested by a markedly prolonged "isovolumetric relaxation" phase and delayed and shortened period of filling. The basic mechanism for these changes is not known.

The abnormality responsible for the altered myocardial contractility might be located at the level of the contractile proteins, the availability of chemical energy or abnormality of the regulation of intracellular calcium flux. The normal hemodynamics observed during cardiac catheterization, the maintenance of normal blood pressure during tachycardia, the abrupt and complete normalization of both ventricular ejection (contraction) and filling (relaxation) as well as normal ventricular function observed immediately after conversion to sinus rhythm would argue against the first two possibilities. More likely, the abnormality is localized at the level of the sarcoplasmic reticulum, which failed to release and take up calcium at the rapid heart rates. The calcium uptake by the sarcoplasmic reticulum is enhanced by cyclic AMP-dependent phosphorylation. Propranolol, a β-blocking agent, may have interfered with a calcium uptake by lowering intracellular cyclic AMP levels and by further slowing relaxation.

The prolonged contraction and relaxation without major alteration of cardiac output may be an "energy-saving" mechanism that the heart uses when exposed to rapid heart rates for prolonged periods in the presence of β-blocking agents. Experimental animal work is needed to elucidate these rate-dependent variations of the contractile response of cardiac muscle.

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References

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