Left Ventricular Function During Stable Sustained Ventricular Tachycardia

Hemodynamic and Echo-Doppler Analysis

Scott B. Baron, M.D.;* Shoei K. Stephen Huang, M.D.;† and Keith A. Comess, M.D.*

To assess the left ventricular function during sustained stable ventricular tachycardia (VT), ten patients, aged 58 to 74, underwent simultaneous echo-Doppler and hemodynamic studies during sinus rhythm and induced sustained stable monomorphic VT. The VT cycle length was 447 ± 92 ms (mean ± SD). During VT, cardiac index fell from 2.32 ± 0.54 to 1.62 ± 0.63 L/min/m² (p<0.001), and systemic systolic blood pressure fell from 129 ± 18 to 107 ± 18 mm Hg (p<0.001), while left ventricular end-diastolic pressure showed a rising trend from 9 ± 7 to 15 ± 12 mm Hg, and pulmonary artery wedge pressure rose from 10.2 ± 1.6 to 24.2 ± 2.3 mm Hg (p<0.005). By echo-Doppler the ejection fraction and the presence and degree of valvular regurgitation were not significantly changed during VT. The mean maximal left ventricular inflow tract velocities, mean time velocity integrals, and the mean time velocity integrals normalized for heart rate (measures of left ventricular diastolic filling) decreased from 0.59 ± 0.074 to 0.40 ± 0.053 m/s (p<0.05), from 0.12 ± 0.029 to 0.021 ± 0.012 m (p<0.001), and from 7.43 ± 1.20 to 3.21 ± 1.49 m x beats/min (p<0.001) during VT, respectively. We conclude that hemodynamic changes during stable sustained VT are neither associated with significant changes in systolic left ventricular function nor related to valvular regurgitation and are likely caused by impaired left ventricular diastolic filling. (Chest 1989; 96:275-80)

The cause of impaired cardiac performance during sustained ventricular tachycardia (VT) has not been well characterized. Hemodynamic compromise has been attributed to decreased systolic or diastolic ventricular function or to valvular incompetence.1-10 While echocardiographic evaluation during ventricular tachycardia has been previously reported,1,10 few thorough Doppler investigations have been described. This study attempts to elucidate the mechanisms of hemodynamic changes and left ventricular function during sustained stable VT with the use of combined two-dimensional (2D) and Doppler echocardiography in conjunction with the hemodynamic measurements.

**MATERIAL AND METHODS**

**Patient Selection**

Ten patients with recurrent, sustained, hemodynamically stable VT who had been referred for electrophysiologic study were prospectively enrolled. "Sustained" was defined as VT lasting more than 30 min to allow for an adequate hemodynamic and echocardiographic evaluation and "hemodynamically stable" as systolic blood pressure by phonemomanometry or by arterial line as greater than or equal to 90 mm Hg.

**Clinical Characteristics**

All patients were men whose mean age was 65 (range, 58 to 74) years. All had coronary artery disease and had had prior myocardial infarctions, none within the preceding six months. All had depressed left ventricular function with angiographic ejection fractions of 31 ± 16 percent (mean ± SD), range, 20 to 51 percent. The diagnosis of VT was made by electrocardiography with a right bundle branch block (RBBB) pattern in eight patients and left bundle branch block (LBBB) pattern in two patients. The rate of spontaneous VT ranged from 115 to 230 beats/min. Four patients were receiving antiarrhythmic therapy (three amiodarone, one propafenone) at the time of study. All others had antiarrhythmic agents discontinued at least five drug half-lives before the study began.

**Protocol**

Patients gave informed consent for the performance of both hemodynamic and electrophysiologic studies. In all but one case, patients underwent baseline hemodynamic and 2D and Doppler echocardiographic evaluation in sinus rhythm. Ventricular tachycardia of morphology similar to the patients' clinical VT was then induced by programmed electrical stimulation. Repeated hemodynamic and 2D and Doppler echocardiographic assessments were made after VT had been sustained for 10 min. One patient underwent echocardiographic evaluation during spontaneous sustained VT and during subsequent sinus rhythm.

**Hemodynamic Evaluation**

Patients were fasting and lightly sedated with diazepam. Percutaneous Swan-Ganz catheterization was performed from the right femoral vein and left heart catheterization from the right femoral artery with a 7F pigtail catheter. Pressure measurements from the right atrium, pulmonary artery, pulmonary capillary wedge, left ventricle, and aorta were processed through fluid-filled Statham transducers linked to a multichannel physiologic recorder (Gould model ES1000), with hard copy displayed on a multichannel strip chart recorder. Cardiac output determinations were made by thermodilution technique. Saturation determinations from the right atrium, pulmonary artery, and systemic circulation were quantified by oximetry.
Electrophysiologic Study

Two 6F quadripolar electrode catheters were placed percutaneously into the right atrium and into the right ventricular apex. Three surface ECG leads (I, aVF, V1) were recorded simultaneously with the intracardiac electrograms filtered at 30 to 300 Hz and displayed on the Gould multichannel recorder. Programmed electrical stimulation (model DTU-201, Bloom Associates) employed a rectangular stimulation pulse and a pulse width of 2 ms at twice the diastolic threshold. Pacing at multiple cycle lengths (600, 500, and 400 ms) using single, double, and triple extrastimuli was performed from the right ventricular apex and, if necessary, from the right ventricular outflow tract. The stimulation protocol was similar to that described previously.11 A 12-lead ECG was obtained during induced sustained VT to compare the QRS morphologies with those of the patients' spontaneous, clinical VT.

Two-Dimensional and Doppler Echocardiography

This study was performed on an ATL Mark 600 (Bothell) using a 3.0 MHz transducer and recorded on videotape and hard copy on heat-sensitive paper. Echocardiographic ejection fractions (Fig 1) were determined in five patients by Simpson's rule (1-mm slices through the left ventricle in the apical four-chamber view) using an off-line computation package.

Pulsed-wave Doppler assessed the valvular regurgitation by flow mapping techniques, which are semi-quantitative and have been validated previously by cardiac catheterization.12-14 Specifically, for the atriobiventricular valves, regurgitation was considered mild if, over a broad waveform in multiple views, high-velocity flow extending at least to mid-systole in duration was detected one-quarter wave into the respective atrium; moderate if detected one-half wave; moderately severe if detected three-quarters way, and severe if detected to the posterior wall. This is exemplified in Figure 2 for the mitral valve. Aortic regurgitation was considered mild if, in multiple views, high-velocity characteristic spectral waveforms accompanied by a harsh audio signal were detected just below the aortic valve; moderate if detected to the mitral leaflet tips; and severe if detected farther into the left ventricle. The mean of the maximal transvalvar velocities (three consecutive beat average during sinus rhythm, 10 beat average during VT) for the mitral and tricuspid valves was obtained from Doppler waveforms obtained with the sample volume placed just distal to the coaptation point of each valve, while the aortic velocities were obtained with the Doppler sample volume placed in the left ventricular outflow tract just proximal to the aortic valve. The time velocity integral at the left ventricular inflow tract was calculated off-line by digitalization using specially designed software and a dedicated microcomputer system. Each echocardiogram was reviewed independently by two observers and on two occasions by one observer, blinded to the previous readings. Where differences in interpretation were noted, a consensus was reached by reevaluation and mutual agreement.

Statistical Analysis

Data during sinus rhythm and VT were analyzed to test for statistical significance (p<0.05) with the Student's paired t test. Intraobserver and interobserver agreement were determined.

Results

Electrophysiologic Testing

All patients were inducible into stable sustained VT having the same morphology as their clinical VT. One patient complained of headache during VT; all others were asymptomatic. The mean VT cycle length was 447 ± 92 ms (range, 260 to 600 ms). The mean sinus

Left Ventricular Function during Stable Ventricular Tachycardia (Baron, Huang, Comess)
cycle length was 865 ± 146 ms (range, 706-1116). Ventriculoatrial conduction during VT was present in only one patient.

**Hemodynamic Studies** (Table 1)

During VT there was a significant fall in the cardiac index from 2.32 ± 0.54 to 1.62 ± 0.63 L/min/m² (p < 0.001) and in the stroke volume index from 33 ± 7.5 to 12 ± 5.3 mL/m² (p < 0.005). The systemic systolic arterial pressure fell during VT from 129 ± 18 to 107 ± 18 mm Hg (p < 0.001), while the mean arterial pressure did not change significantly, 90 mm Hg during sinus rhythm and 86 mm Hg during VT. The left ventricular end-diastolic pressure showed a rising trend from a mean of 9 ± 7 mm Hg in sinus rhythm to 15 ± 12 during VT; however, too few values were available to prove statistical significance. While there were increases in right atrial mean pressure from 4.5 ± 2.2 to 11.4 ± 4.6 mm Hg (p < 0.05), pulmonary artery systolic pressure from 29.6 ± 10.8 to 37.2 ± 10.4 mm Hg (p < 0.05), pulmonary artery diastolic pressure from 13.5 ± 6.8 to 24.7 ± 7.4 mm Hg (p < 0.05), pulmonary artery mean pressure from 19.3 ± 6.7 to 30.1 ± 9.2 mm Hg (p < 0.05), pulmonary artery wedge pressure from 10.2 ± 1.6 to 24.2 ± 2.3 mm Hg (p < 0.005). Saturations determined from the right

<table>
<thead>
<tr>
<th>Measurement</th>
<th>No. of Patients</th>
<th>Sinus Rhythm</th>
<th>Ventricular Tachycardia</th>
<th>p Value</th>
</tr>
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<tbody>
<tr>
<td>Cycle length, ms</td>
<td>10</td>
<td>856 ± 146</td>
<td>447 ± 92</td>
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<tr>
<td>Cardiac index, L/min/m²</td>
<td>9</td>
<td>2.32 ± 0.54</td>
<td>1.62 ± 0.63</td>
<td>&lt;0.001</td>
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<tr>
<td>Stroke volume index, mL/m²</td>
<td>9</td>
<td>33 ± 7.5</td>
<td>12 ± 5.3</td>
<td>&lt;0.005</td>
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<tr>
<td>Right atrial pressure, mm Hg</td>
<td>8</td>
<td>4.5 ± 2.2</td>
<td>11.4 ± 4.6</td>
<td>&lt;0.05</td>
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<tr>
<td>Pulmonary artery systolic pressure, mm Hg</td>
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<td>29.6 ± 10.2</td>
<td>37.2 ± 10.4</td>
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<tr>
<td>Pulmonary artery diastolic pressure, mm Hg</td>
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<td>13.5 ± 6.8</td>
<td>24.7 ± 7.4</td>
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<tr>
<td>Pulmonary artery mean pressure, mm Hg</td>
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<td>19.3 ± 6.7</td>
<td>30.1 ± 9.2</td>
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<td>Pulmonary artery wedge pressure, mm Hg</td>
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<td>10.2 ± 1.6</td>
<td>24.2 ± 2.3</td>
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<tr>
<td>Left ventricular end-diastolic pressure, mm Hg</td>
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<td>9 ± 7</td>
<td>15 ± 12</td>
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</tr>
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<td>Systemic artery systolic pressure, mm Hg</td>
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<td>129 ± 18</td>
<td>107 ± 18</td>
<td>&lt;0.001</td>
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<td>Systemic artery mean pressure, mm Hg</td>
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<td>90.2 ± 9.0</td>
<td>86.4 ± 13.0</td>
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<tr>
<td>Systemic vascular resistance dynes/cm²</td>
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<td>1,712 ± 376</td>
<td>2,415 ± 924</td>
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<tr>
<td>Total pulmonary resistance, dynes/cm²</td>
<td>8</td>
<td>392 ± 141</td>
<td>844 ± 336</td>
<td>&lt;0.01</td>
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</tbody>
</table>

**Figure 2.** Detection of mitral regurgitation. **Left panels:** Two-dimensional echocardiograms with pulsed Doppler sample volume in left atrium. Upper: sinus rhythm; lower: ventricular tachycardia. **Right panels:** Pulsed Doppler spectral tracings with high-velocity systolic flow of mitral regurgitation below baseline and diastolic mitral inflow above baseline (baseline = horizontal in middle of frame; each horizontal from baseline denotes 0.1 m/s). Upper: sinus rhythm; lower: ventricular tachycardia.
atrium, pulmonary artery, and systemic artery did not change significantly during sustained VT.

**2D Echocardiography (Table 2)**

Echocardiographically determined ejection fraction in seven patients who had adequate definition of endocardium in the apical four-chamber view did not show a significant change from sinus rhythm to VT, 34 ± 11 percent vs 30 ± 13 percent. The values in sinus rhythm compared favorably with those obtained during prior left ventriculography (31 ± 16 percent).

**Doppler Echocardiography (Tables 2 and 3).**

Of nine adequate studies, Doppler found five patients to have mild and one moderate mitral regurgitation during sinus rhythm, and seven patients to have mild mitral regurgitation during VT. Mild aortic regurgitation was found in the same three patients during both sinus rhythm and VT. Mild tricuspid regurgitation was found in six patients during sinus rhythm and in seven during VT. Mean maximal left ventricular inflow tract velocity in sinus rhythm was 0.59 ± 0.074 m/s and fell to 0.40 ± 0.053 m/s during VT (p<0.05). The mean time velocity integrals for the mitral valve filling wave forms were 0.12 ± 0.029 m during sinus rhythm and 0.021 ± 0.012 m during VT (p<0.001). Multiplying the mitral time velocity integral by the heart rate of each patient during sinus rhythm and VT, respectively, and averaging this value for all patients, yielded a measurement of mean left ventricular filling per minute comparable between sinus rhythm and VT. This value was greater for sinus rhythm at 7.43 ± 1.20 m x beats/min compared to VT, 3.21 ± 1.49 m x beats/min (p<0.001).

There were no statistically significant intraobserver (71 percent agreement) or interobserver (74 percent agreement) differences in assigning the presence or degree of regurgitation by flow mapping during sinus rhythm compared with VT.

**DISCUSSION**

Mechanisms which have been proposed to affect cardiac performance during sustained ventricular tachycardia include changes in left ventricular systolic function, the degree of valvular regurgitation, the resistance to outflow, and left ventricular diastolic function. We evaluated ten selected patients during sustained and stable ventricular tachycardia with hemodynamic measurements and 2D and Doppler echocardiography and suggest that disorders of diastolic function may be the most important cause of impaired ventricular performance during this dysrhythmia. The results of our study are not intended to explain the mechanisms of syncope in a different group of patients with a hemodynamically unstable VT.

**Systolic Function**

We did not find systolic function to be significantly changed comparing ventricular tachycardia with sinus rhythm in our patients. No significant difference was found in echocardiographically determined ejection fraction. While others have proposed that asynchrony

<table>
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<th>Patient No.</th>
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<th>Tricuspid</th>
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</thead>
<tbody>
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<td></td>
<td>SR</td>
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<td>SR</td>
</tr>
<tr>
<td>1</td>
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</tr>
<tr>
<td>2</td>
<td>None</td>
<td>None</td>
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</tr>
<tr>
<td>3</td>
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<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>4</td>
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</tr>
<tr>
<td>5</td>
<td>None</td>
<td>Mild</td>
<td>Mild</td>
</tr>
<tr>
<td>6</td>
<td>Inadequate Study</td>
<td>Inadequate Study</td>
<td>Inadequate Study</td>
</tr>
<tr>
<td>7</td>
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<td>Mild</td>
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</tr>
<tr>
<td>8</td>
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</tr>
<tr>
<td>9</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>Mild</td>
<td>Mild</td>
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</tr>
</tbody>
</table>

*SR = sinus rhythm; VT = ventricular tachycardia.
of contraction, owing to abnormal activation sequence or abnormal conduction pathways, or ischemic ventricular dysfunction may impair systolic function during VT.\textsuperscript{4,6,8,9,15,16} We found that in our group of patients these mechanisms did not make an important contribution overall. Lima et al\textsuperscript{4} found that patients with hypotensive VT (unlike our patients) and impaired ventricular function (mean left ventricular ejection fraction, 29 percent) had marked incoordinate contraction as determined by pressure-dimension loops.

The mean VT rate in their patients was 193 beats/min, significantly higher than that in our study. Another group of their patients with a mean left ventricular ejection fraction of 61 percent and hypotensive VT, however, had relatively insignificant systolic dysfunction by pressure-dimension loops. The impairment in stroke volume in this group was thought to be due to incomplete relaxation. A third group of five patients with nonhypotensive VT and a slower VT rate (mean of 148 beats/min) had relatively normal left ventricular function at rest and virtually unchanged pressure-dimension loops. Again, this suggests an insignificant change in systolic function in these patients. The disparate findings in their first patient group with poor ventricular function, thought to have incoordinate contraction, has not been well explained and has not been substantiated by investigators assessing similar patients,\textsuperscript{5} nor have others\textsuperscript{4} reached the same conclusion.

Valvular Regurgitation

Increased valvular regurgitation during VT would lead to decreased cardiac output secondary to decreased forward flow.\textsuperscript{4,10} We did not find a significant increase in the number of patients who developed regurgitant lesions during VT nor in the severity of the valvular regurgitation during stable VT. Therefore, we believe that valvular incompetence in our patients is unlikely to be a significant cause of a decreased cardiac output during sustained VT.

Peripheral Vascular Resistance

It is possible that changes in peripheral vascular tone and autonomic mechanism play a significant role in the maintenance of blood pressure during VT.\textsuperscript{17} Our study did demonstrate an increase in total systemic vascular resistance and diastolic pressure with a narrowed pulse pressure during VT. It has been suggested that there are no hemodynamically significant changes in serum catecholamine levels in patients with inducible VT.\textsuperscript{18} It seems unlikely, then, that the ability to maintain a stable blood pressure during VT is directly due to a serum catecholamine response, though it is possible that local increases in norepinephrine or other mediators affecting vasomotor tone may play a role.

Diastolic Function

The present study indicates that a decrease in diastolic left ventricular filling appears to be the most important factor influencing cardiac performance during stable sustained VT. We determined the mean maximal velocities and the time velocity integrals in the left ventricular inflow tract with Doppler echocardiography as a measure of filling of the left ventricle\textsuperscript{19} and have demonstrated that ventricular filling appears to be decreased during VT, even when normalized for the faster heart rate during VT. With a faster heart rate, the filling time decreases per beat, which may impair left ventricular filling.\textsuperscript{5,7} but this is unlikely to be the only important factor. Figure 3 suggests that the correlation between VT cycle length and cardiac output is not strong. It is also possible that ventricular compliance may decrease during VT, suggested by significant increases in pulmonary capillary wedge pressures without a concomitant increase in left ventricular diastolic filling.

Another important mechanism for decreased diastolic filling in VT may be the inappropriate timing of atrial systole.\textsuperscript{1} Goldreyer et al\textsuperscript{20} considered this to be a factor for a fall in cardiac output during paroxysmal supraventricular tachycardia in patients with normal ventricles. Fisher et al\textsuperscript{21} presented a patient in VT with hypotension and DeMaria et al\textsuperscript{6} a patient with an idioventricular rhythm whose blood pressures improved dramatically with the addition of timed atrial pacing. Hamer et al\textsuperscript{3} further demonstrated that atrial pacing during VT may lessen hemodynamic compromise of this arrhythmia. That improvement in ventricular diastolic filling yields an improved cardiac output with properly timed atrial pacing during VT suggests that a decrease in left ventricular diastolic filling, not incoordinate ventricular contraction, is likely to be the major factor compromising cardiac performance during VT.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3.png}
\caption{Cardiac output during ventricular tachycardia as a function of cycle length (N = 7 patients).}
\end{figure}
Limitations

The number of patients in this study is relatively small and limited by a complex nature of the study protocol. Our study group was highly select and represented only those patients with a stable, sustained VT.

We were not able to study patients with hemodynamically unstable VT, and the data obtained from our patients may not be applicable to them. We were able to determine ejection fractions in only seven patients, in which good agreement was demonstrated between ventriculography and echocardiographic measures of ejection fractions during sinus rhythm. Though we did not validate the ejection fraction measurements obtained during ventricular tachycardia by another technique, others previously used this methodology both during ventricular tachycardia and with rapid pacing. The use of Simpson's rule with 1-mm slices should have fewer problems in volume measurement with possible ventricular distortion during ventricular tachycardia.

Also, though pulsed-wave Doppler echocardiographic flow mapping is an accepted technique for estimating valvular regurgitation, limitations are apparent in attempting to precisely calculate valvular regurgitation by this technique. Similar objections may be made regarding precise measurements of left ventricular filling using Doppler. Studies have confirmed the comparability and reproducibility of these measurements. Doppler methods should allow for comparisons in the same patient between sinus rhythm and ventricular tachycardia.

Conclusions

The mechanism of impaired cardiac performance in patients during stable sustained ventricular tachycardia appears likely to be due to decreased ventricular diastolic filling and is unlikely related to an increase in systolic dysfunction or valvular regurgitation.

References