Effect of Pacing-Induced Tachycardia and Myocardial Ischemia on Ventricular Pressure-Velocity Relationships in Man

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SUMMARY

Ventricular function was evaluated in 18 patients prior to left ventriculography and selective coronary arteriography. Simultaneous left ventricular pressure (catheter-tip manometer) and dP/dt were recorded at resting heart rates and during tachycardia induced by right atrial pacing. Pressure-velocity curves were constructed from which \( V_{\text{max}} \) and maximum measured contractile element velocity (max \( V_{\text{CE}} \)) were obtained. \( V_{\text{max}} \) and max \( V_{\text{CE}} \) initially increased with pacing-induced tachycardia in 17 of the 18 patients. Eight patients developed evidence of myocardial ischemia during atrial pacing. During the period of myocardial ischemia there was a decrease in \( V_{\text{max}} \) and max \( V_{\text{CE}} \) in all eight patients despite constant or increasing heart rate. In the 10 patients who did not develop evidence of myocardial ischemia with pacing-induced tachycardia, \( V_{\text{max}} \) and max \( V_{\text{CE}} \) continued to increase or remained constant with increasing rate. Peak left ventricular dP/dt increased coincident with the onset of myocardial ischemia in six of eight patients despite a fall in \( V_{\text{max}} \) and max \( V_{\text{CE}} \) in all eight patients during the ischemic period. A highly significant difference was demonstrated between \( V_{\text{max}} \) values of patients with normal ejection fractions and patients with low ejection fractions, both at rest and during pacing-induced tachycardia.

Additional Indexing Words:
- \( V_{\text{max}} \) Contractile element velocity
- Left ventricular dP/dt
- Ventricular function
- Coronary atherosclerosis

EVALUATION of ventricular hemodynamic events during periods of increased myocardial oxygen demand induced by atrial pacing provides a useful measure of cardiac performance, particularly in patients with coronary atherosclerosis. Many of the hemodynamic changes associated with pacing-induced tachycardia and myocardial ischemia are known, but the influence of tachycardia and ischemia on the contractile state of the human ventricle requires further documentation.

Indices reflecting the contractile state of the intact human heart have been developed by the extension of force-velocity principles derived from basic work in muscle mechanics. The maximum velocity of contractile element shortening (\( V_{\text{max}} \)), derived from the instantaneous relation between left ventricular pressure (P) and the rate of pressure rise during isovolumic systole (dP/dt), has been shown to be a good index of ventricular...
contractility in the intact heart, reflecting changes in the inotropic state of the ventricle independent of loading conditions. The effects of atrial pacing and pacing-induced myocardial ischemia on ventricular hemodynamics and pressure-velocity parameters have been evaluated in 18 patients undergoing left heart catheterization and coronary arteriography. Changes in ventricular contractility, as estimated in this way, are compared with other hemodynamic and angiographic findings, with particular reference to differences between patients who did and patients who did not show evidence of myocardial ischemia during atrial pacing.

**Materials and Methods**

Studies in the 18 patients were performed prior to diagnostic coronary arteriography and left ventriculography. As indicated in table 1, there were three female and 15 male patients with an average age of 49.5 years (range 42–67 years). All patients had a history suggesting ischemic chest pain, and all were candidates for diagnostic cardiac catheterization and coronary arteriography. None of the patients had valvular heart disease.

Hemodynamic and angiographic studies were performed in the Wellcome Research Laboratory of The Johns Hopkins University School of Medicine. The procedure was explained to all patients, and written consent was obtained. Patients were studied in the postabsorptive state after premedication with pentobarbital 100 mg, atropine 0.4 mg, diphenhydramine 50 mg, and morphine 5 mg. A bipolar electrode pacing catheter was positioned in the right atrium via an antecubital vein cutdown. A catheter-tip pressure transducer (Statham model SF1 transducer, catheter size no. 6.5 French) was introduced via percutaneous femoral artery puncture using a catheter introducer and sheath. This catheter was passed retrograde to the aortic root and left ventricle. The left ventricular pressure signal from the catheter-tip transducer was standardized against the signal from a Statham P23Db pressure transducer attached to the fluid-filled lumen of the Statham model SF1 transducer catheter.

The first derivative of left ventricular pressure (dP/dt) was continuously generated using a resistance-capacitance differentiating circuit* with a corner frequency of 160 Hz. In five cases the first derivative of pressure was also generated using a integrated-circuit high-fidelity differentiator with a corner frequency of 300 Hz. In these cases no differences were noted between pressure-velocity curves constructed using dP/dt generated by the two differentiating circuits.

**Procedure**

In the control state simultaneous measurements of high-fidelity left ventricular pressure and dP/dt were recorded on an Electronics for Medicine recorder at a paper speed of 200 mm/sec. Right atrial pacing was then begun at a rate of 90 beats/min and sequentially increased at increments of 20 beats/min. Each pacing rate was maintained for 1 min, at the end of which time left ventricular pressure and dP/dt were simultaneously recorded at that paced rate. Following this, the heart rate was increased to the next level, and the procedure was repeated.

Myocardial ischemia was defined as chest pain similar to the patients' previous complaints of typical angina pectoris, ischemic electrocardiographic changes, or both. If a patient developed evidence of myocardial ischemia during the course of pacing, left ventricular pressure and dP/dt were recorded after 1 min of pacing at that rate, and following this pacing was usually discontinued. In a few patients in whom there was no clinical contraindication, pacing at a higher rate was attempted. In those patients who did not develop evidence of myocardial ischemia during pacing, heart rate was increased to a maximum of 170 beats/min if technically possible. The entire pacing protocol lasted approximately 10–15 min. All patients tolerated pacing-induced tachycardia and/or pacing-induced ischemia well, and there were no complications attributable to atrial pacing. Following the pacing study, routine left ventriculography in the right anterior oblique projection and coronary arteriography were performed in all patients.

**Analysis of Results**

Contractile element velocity (VCE) was calculated from the expression (dP/dt)/32P during the period of isovolumic systole at 5-msec intervals as shown in figure 1. VCE values were then plotted against the corresponding instantaneous isovolumic left ventricular pressures. The descending limb of the resultant pressure-velocity curve was extrapolated to zero pressure allowing estimation of Vmax using the method of Mason et al.7 (fig. 2). Pressure-velocity curves yielding values of Vmax and maximum VCE (max VCE) were constructed in this way for each pacing rate. Measurements of LVEDP, peak left ventricular dP/dt, and aortic diastolic pressure were also recorded at each paced rate. The course of a

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*Electronics for Medicine, model DR8, White Plains, New York.
typical pacing study in a patient who developed angina pectoris during atrial pacing is outlined in figure 3. End-systolic volume and end-diastolic volume were calculated by the single-plane angiographic method of Greene et al.8 and ejection fraction was determined from these measurements.

Results

The hemodynamic and angiographic findings of the 18 patient studies are summarized in table 1. The patients were divided into two general groups, based on the presence (ischemic responders) or absence (nonischemic responders) of myocardial ischemia during the course of atrial pacing.

Nonischemic Responders

Ten of the 18 patients studied (patients 1-10 in table 1) did not develop evidence of myocardial ischemia during pacing-induced tachycardia despite heart rates of 130-170 beats/min. These patients are designated nonischemic responders to pacing-induced tachycardia. Significant coronary atherosclerotic lesions (greater than 70% narrowing of one of more major coronary vessels) were present in six of these 10 patients (patients 5-10), while the other four patients (patients 1-4) had normal coronary arteriograms. Ejection fractions were normal (0.50 or greater) in nine of the 10 patients, although two of these nine patients with normal ejection fractions had localized ventricular kinetic abnormalities. One of the 10 nonischemic responders had a markedly reduced ejection fraction of 0.27 and a diffusely hypokinetic ventricle secondary to severe coronary atherosclerosis.

Figure 4 summarizes the effect of increasing heart rate on \(V_{\text{max}}\) in the four nonischemic responders with normal coronary arteries (patients 1-4). All patients showed progressive increases in \(V_{\text{max}}\) with increasing heart rates. \(V_{\text{max}}\) increased significantly from a mean control value of 2.2 to a mean of 3.1 muscle lengths/sec at the highest paced rate achieved (\(P < 0.005\)). As noted in table 1, max \(V_{\text{OE}}\)

### Table 1

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Control rate (beats/min)</th>
<th>Highest paced rate (beats/min)</th>
<th>Evidence of ischemia</th>
<th>Angiographic findings</th>
<th>(V_{\text{max}}) (muscle lengths/sec)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Artgm</td>
<td>Ventgm</td>
</tr>
<tr>
<td>1</td>
<td>45</td>
<td>M</td>
<td>60</td>
<td>130</td>
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<td>Norm</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>M</td>
<td>95</td>
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<tr>
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<td>82</td>
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<tr>
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<td>Ap asyn</td>
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<td>Inf asyn</td>
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<tr>
<td>11</td>
<td>44</td>
<td>M</td>
<td>70</td>
<td>170</td>
<td>At 170</td>
<td>CAS</td>
<td>Gen hypokin</td>
</tr>
<tr>
<td>12</td>
<td>48</td>
<td>M</td>
<td>106</td>
<td>170</td>
<td>At 170</td>
<td>CAS</td>
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</tr>
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<td>CAS</td>
<td>Ap asyn</td>
</tr>
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<td>54</td>
<td>F</td>
<td>95</td>
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<td>At 170</td>
<td>CAS</td>
<td>Inf hypokin</td>
</tr>
<tr>
<td>15</td>
<td>42</td>
<td>M</td>
<td>53</td>
<td>90</td>
<td>At 90</td>
<td>CAS</td>
<td>Norm</td>
</tr>
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<td>16</td>
<td>55</td>
<td>M</td>
<td>73</td>
<td>110</td>
<td>At 110</td>
<td>CAS</td>
<td>Ant &amp; inf asyn</td>
</tr>
<tr>
<td>17</td>
<td>67</td>
<td>M</td>
<td>67</td>
<td>150</td>
<td>At 150</td>
<td>CAS</td>
<td>Gen hypokin</td>
</tr>
<tr>
<td>18</td>
<td>55</td>
<td>M</td>
<td>77</td>
<td>170</td>
<td>At 170</td>
<td>CAS</td>
<td>Gen hypokin</td>
</tr>
</tbody>
</table>

Abbreviations: CAS = coronary atherosclerosis; EF = ejection fraction; Artgm = coronary arteriogram; Ventgm = ventriculogram; Cont = control; Preisch = at peak rate prior to ischemia; Postisch = following ischemia; Ap asyn = apical asynergy; Inf asyn = inferior asynergy; hypokin = hypokinesis.
values also progressively increased with increasing heart rates in these individuals.

Figure 5 summarizes the effect of increasing heart rate on \( V_{\text{max}} \) in the six nonischemic responders who had evidence of significant coronary atherosclerosis (patients 5–10). Five of these six patients (patients 5–9) showed progressive increases in \( V_{\text{max}} \) with increasing heart rate. In these five patients there was a significant increase in \( V_{\text{max}} \) from a mean control value of 1.9 to a mean value of 3.7 muscle lengths/sec at the highest paced rates achieved \((P < 0.005)\). Parallel increases in \( V_{\text{CE}} \) were observed as indicated in table 1. One individual (patient 10) with a diffusely hypokinetic ventricle and low ejection fraction (0.27) had no significant change in \( V_{\text{max}} \) with increasing heart rate. In this patient, \( V_{\text{max}} \) values were the lowest of the entire group of 10 patients and remained stable in the range of 0.8–1.0 muscle lengths/sec despite increasing heart rate.

LVEDP did not change significantly with increasing heart rate in six of the 10 nonischemic responders. Of the four other patients, two showed increases and two decreases in LVEDP with pacing-induced tachycardia. Diastolic aortic pressure did not change significantly in eight of 10 nonischemic responders, increased in one, and decreased in another as heart rate increased. Left ventricular peak dP/dt rose with increasing heart rate in all 10 nonischemic responders.

Ischemic Responders

Eight patients (patients 11–18) developed evidence of myocardial ischemia during the course of atrial pacing. All of these patients had significant coronary atherosclerotic lesions. Ejection fractions were normal in six of the eight patients (patients 11–16), although four of these six patients had localized areas of ventricular akinesia. Two of the eight patients (patients 17 and 18) had generalized ventricular hypokinesis and ejection fractions of 0.22.

Figure 6 illustrates the effect of increasing heart rate and tachycardia-induced myocardial ischemia on \( V_{\text{max}} \) in these patients. All eight patients had a progressive increase in

<table>
<thead>
<tr>
<th>Max ( V_{\text{CE}} ) (muscle lengths/sec)</th>
<th>LVEDP (mm Hg)</th>
<th>Diastolic aortic pressure (mm Hg)</th>
<th>LV peak dP/dt (mm Hg/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cont</td>
<td>Preisch</td>
<td>Postisch</td>
<td>Cont</td>
</tr>
<tr>
<td>1.9</td>
<td>2.4</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>2.0</td>
<td>2.9</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>1.5</td>
<td>2.0</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>1.3</td>
<td>1.4</td>
<td>—</td>
<td>12</td>
</tr>
<tr>
<td>1.5</td>
<td>2.6</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>1.7</td>
<td>2.6</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>1.5</td>
<td>1.9</td>
<td>—</td>
<td>9</td>
</tr>
<tr>
<td>0.7</td>
<td>2.3</td>
<td>—</td>
<td>17</td>
</tr>
<tr>
<td>1.2</td>
<td>1.5</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>0.5</td>
<td>0.8</td>
<td>—</td>
<td>21</td>
</tr>
<tr>
<td>2.8</td>
<td>5.6</td>
<td>4.2</td>
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</tr>
<tr>
<td>2.9</td>
<td>5.1</td>
<td>2.0</td>
<td>2</td>
</tr>
</tbody>
</table>

| 1.0                                | 3.0           | 1.8                             | 14                       | 4             | 4              | 85                      | 85           | 93             | 1992                     | 2192            | 2368 |
| 1.3                                | 2.9           | 2.2                             | 10                       | 6             | 13             | 80                      | 80           | 90             | 1860                     | 2785            | 3126 |
| 0.9                                | 1.4           | 1.2                             | 14                       | 10            | 13             | 55                      | 65           | 75             | 1220                     | 1652            | 1759 |
| 1.1                                | 1.5           | 1.2                             | 7                        | 8             | 8              | 52                      | 68           | 55             | 813                      | 1070            | 836  |
| 0.5                                | 0.8           | 0.7                             | 26                       | 13            | 16             | 83                      | 72           | 92             | 1043                     | 1112            | 1280 |
| 0.5                                | 0.9           | 0.7                             | 34                       | 18            | 25             | 76                      | 50           | 58             | 980                      | 1117            | 1000 |
Vmax decreased in all eight patients despite constant or increasing heart rates. In these eight patients, Vmax increased significantly from a mean control value of 1.8 to a mean value of 3.5 muscle lengths/sec at the highest paced rate achieved prior to the onset of myocardial ischemia ($P < 0.005$). Following the onset of myocardial ischemia, Vmax decreased significantly to an average value of 2.6 muscle lengths/sec ($P < 0.01$) despite constant or increasing heart rate. Changes in max $V_{CE}$ paralleled Vmax changes in all cases (table 1). The two patients with low ejection fractions (patients 17 and 18) had the lowest Vmax values of the group throughout the entire range of heart rates. Changes in Vmax

Vmax with increasing heart rate up to the point at which evidence of myocardial ischemia was noted. During myocardial ischemia,

**Figure 1**

Calculation of $V_{CE}$ from simultaneous values of left ventricular pressure and dp/dt during isovolumic systole (area between dotted lines).

**Figure 2**

Estimation of $V_{max}$ from plot of $V_{CE}$ versus simultaneous left ventricular pressure during isovolumic systole.

**Figure 3**

A typical experiment in a patient who developed myocardial ischemia manifested as angina pectoris when paced to a rate of 170 beats/min. Changes in heart rate, LVEDP, aortic diastolic pressure, LV peak dp/dt, max $V_{CE}$, and $V_{max}$ are plotted against time.
PACING-INDUCED TACHYCARDIA

Effect of increasing heart rate on $V_{\text{max}}$ in nonischemic responders with normal coronary arteries.

Effect of increasing heart rate on $V_{\text{max}}$ in nonischemic responders with coronary atherosclerosis.

with onset of myocardial ischemia in these two patients were not as large as in other patients in the group. This response to pacing is similar to that of the individual (patient 10) with a low ejection fraction in the nonischemic group.

Prior to the onset of signs or symptoms of myocardial ischemia, LVEDP fell in six patients and remained stable in two with increasing heart rate induced by atrial pacing. Coincident with the onset of myocardial ischemia, LVEDP increased in six patients and remained stable in two. Diastolic aortic pressure showed a variable response to increasing heart rate in this group of eight patients prior to the onset of myocardial ischemia, but coincident with the onset of ischemia diastolic aortic pressure rose in seven of eight patients.

Peak left ventricular $dP/dt$ rose with increasing heart rate in all eight patients prior to the onset of ischemia. Coincident with the onset of ischemia, peak left ventricular $dP/dt$ continued to increase in six of eight patients, while decreasing in only two patients. The increase in peak left ventricular $dP/dt$ in these six patients occurred despite a fall in $V_{\text{max}}$ and $V_{\text{CE}}$ in all eight patients with the onset of ischemia. Changes in $V_{\text{max}}$, $V_{\text{CE}}$, LVEDP, aortic diastolic pressure, and peak left ventricular $dP/dt$ with onset of myocardial ischemia in this group of eight patients are summarized in table 2.

Relationship of $V_{\text{max}}$ to Ventricular Ejection Fraction and to Presence of Coronary Atherosclerosis

Control $V_{\text{max}}$ values of the three patients with abnormally low ejection fractions of less than 0.30 (patients 10, 17, and 18) were 0.8, 0.6, and 0.6 muscle lengths/sec, respectively. The mean control $V_{\text{max}}$ values of these three patients was 0.7 muscle lengths/sec. The mean control $V_{\text{max}}$ values of the remaining 15

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patients with normal ejection fractions was 2.1 muscle lengths/sec. The difference between these two means is significant (P < 0.0005).

Peak $V_{\text{max}}$ values of the three patients with low ejection fractions, calculated from data obtained during atrial pacing, were 1.0, 1.3, and 1.2, respectively, with a mean value of 1.2 muscle lengths/sec. The mean peak $V_{\text{max}}$ values of the 15 patients with normal ejection fractions was 3.8 muscle lengths/sec. The difference between these two means is significant (P < 0.0005).

A significant difference in $V_{\text{max}}$ values was therefore demonstrated between patients with normal ejection fractions, and patients with low ejection fractions, both at rest and during pacing-induced tachycardia. However, within the group of 15 patients with normal ejection fractions, there was a poor correlation between control $V_{\text{max}}$ and ejection fraction values ($r = 0.14$).

There was no significant difference, either at control heart rates or during pacing-induced tachycardia prior to the onset of myocardial ischemia, between the mean $V_{\text{max}}$ values of the following three groups of patients: (1) nonischemic responders with normal coronary arteries (patients 1–4); (2) nonischemic responders with coronary atherosclerosis (patients 5–10); and (3) ischemic responders with coronary atherosclerosis (patients 11–18).

**Discussion**

In recent years force-velocity principles derived from isolated muscle preparations have been applied to the intact human ventricle as a means of evaluating ventricular performance. The $V_{\text{max}}$, determined from isovolumic pressure and dP/dt, appears to be responsive to changes in the inotropic state of the ventricle induced by pharmacologic and other interventions. Relatively subtle changes in ventricular performance have been detected in this way.

The positive inotropic influence of increasing heart rate has been documented previously, and was originally described as the "treppe" phenomenon of Bowditch. Hence, the influence of heart rate on $V_{\text{max}}$, or other indices of inotropic state, must be considered, and heart rate must be controlled in studies utilizing $V_{\text{max}}$ to evaluate ventricular performance. Prior to the onset of myocardial ischemia, 17 of our 18 patients demonstrated a progressive increase in $V_{\text{max}}$ with increasing heart rate. This increase in $V_{\text{max}}$ probably reflects improvement in ventricular contractility associated with increased rate, particularly since preload response to atrial pacing was variable. Only one patient, a man with generalized ventricular hypokinesis secondary to severe diffuse coronary atherosclerosis, failed to increase $V_{\text{max}}$ significantly with pacing-induced tachycardia (patient 10).

All eight patients who developed myocardial ischemia during the course of atrial pacing demonstrated a fall in $V_{\text{max}}$ coincident with the onset of ischemia despite constant or increasing rate. Impaired ventricular contractility associated with ischemia is the most likely explanation for this decrease in $V_{\text{max}}$.

Changes in $V_{\text{max}}$ with atrial pacing and myocardial ischemia were closely paralleled by changes in maximum measured $V_{\text{CE}}$ in all 18 patients studied. Max $V_{\text{CE}}$ progressively increased with increasing heart rate and fell when myocardial ischemia was noted. Although theoretically less independent of loading conditions, max $V_{\text{CE}}$ appears to be a good index of ventricular contractility under the conditions of this study and has the advantage.

**Table 2**

*Summary of Hemodynamic Changes in Eight Ischemic Responders Coincident with the Onset of Myocardial Ischemia*

<table>
<thead>
<tr>
<th>Patients</th>
<th>$V_{\text{max}}$</th>
<th>Max Vce</th>
<th>LVEDP</th>
<th>Aortic diastolic pressure</th>
<th>Peak LV dP/dt</th>
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<tr>
<td>No. increased</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>No. decreased</td>
<td>8</td>
<td>8</td>
<td>2</td>
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</table>

*Circulation, Volume XLVI, July 1972*
of being a parameter measured directly from isovolumic pressure and $dP/dt$ without extrapolation. Other reports have likewise cited a good correlation between these two indices.\textsuperscript{10, 12}

The peak rate of rise of left ventricular pressure has been widely used as an index of left ventricular contractility.\textsuperscript{21} However, the influence of preload and afterload on peak $dP/dt$ limits its application in many circumstances. In this study changes in peak $dP/dt$ parallel changes in $V_{\text{max}}$ and max $V_{CE}$ in some instances but not in others. As outlined in table 2, following the onset of myocardial ischemia peak left ventricular $dP/dt$ increased in six of eight patients and decreased in only two. These changes in peak $dP/dt$ occurred in the face of decreasing ventricular contractility as reflected by decreases in $V_{\text{max}}$ and max $V_{CE}$ in all eight patients. This apparent discrepancy can be largely explained by changes in ventricular loading conditions coincident with the onset of myocardial ischemia. Six of eight patients had increases of LVEDP at the onset of myocardial ischemia, and seven of eight patients developed an increase in aortic diastolic pressure. These increases in preload and afterload could be responsible for an increase in left ventricular peak $dP/dt$ and overshadow a concomitant decrease in the contractile state of the left ventricle. The experimental data of the patient presented in figure 3 clearly illustrates these points.

Correlation between $V_{\text{max}}$ and other indices of ventricular performance has been reported previously.\textsuperscript{6-11} In this study a highly significant difference was noted between $V_{\text{max}}$ values of patients with normal ejection fractions and $V_{\text{max}}$ values of patients with low ejection fractions. The three patients with low ejection fractions had low $V_{\text{max}}$ values throughout the entire range of heart rates observed. $V_{\text{max}}$ varied only slightly with changes in heart rate or with the onset of myocardial ischemia in these patients.

Neither $V_{\text{max}}$ nor ejection fraction can be regarded as a specific and sensitive indicator of ventricular contractility under all conditions. This may explain the absence of a good correlation between these two parameters in the group of 15 patients with normal ejection fractions. Several of the patients in this series had localized abnormalities of left ventricular wall motion which may have influenced the shape of the pressure-velocity curves, although recent evidence suggests that $V_{\text{max}}$ is not influenced by such kinetic abnormalities.\textsuperscript{22, 23} Changes in a number of other factors, such as the series-elastic properties of the myocardium, or course of the active state, could theoretically influence pressure-velocity indices independent of changes in contractility.

We were unable to show a significant difference between mean $V_{\text{max}}$ values of patients with normal coronary arteriograms, nonischemic responders with coronary atherosclerosis, and ischemic responders with coronary atherosclerosis, either at control heart rates or during pacing-induced tachycardia prior to the onset of myocardial ischemia. However, changes in hemodynamic parameters and $V_{\text{max}}$ at the onset of myocardial ischemia in the group of ischemic responders clearly separated these patients from the other two groups. The majority of patients with coronary atherosclerosis had normal ejection fractions, and overall ventricular contractility in these patients was judged to be good by angiographic methods. It is therefore not entirely surprising that no significant difference in $V_{\text{max}}$ could be demonstrated between these three groups of patients prior to the onset of myocardial ischemia.

A number of assumptions are made in the application of force-velocity principles to cardiac muscle, either in the isolated muscle preparation or in the intact heart.\textsuperscript{6} Consequently, the use of this technic to evaluate contractility has been criticized, and the specificity of $V_{\text{max}}$ has been questioned.\textsuperscript{24, 25} $V_{\text{max}}$ derived from pressure-velocity curves constructed using total pressure, as in this study, has been shown to be independent of preload in some studies,\textsuperscript{7} while other investigators have found this index to be somewhat dependent on preload.\textsuperscript{26} The use of developed ventricular pressure, rather than total pressure, in the construction of pressure-velocity
curves has been reported to yield a more specific, though less sensitive index of contractility.\textsuperscript{26} It is possible that some of the changes in $V_{\text{max}}$ and $V_{\text{CE}}$ noted in this study were influenced by factors other than changes in contractility. However, the consistent increase in $V_{\text{max}}$ and max $V_{\text{CE}}$ with increasing heart rate despite the variable response of preload and afterload, the consistent decrease in these indices with the onset of myocardial ischemia, and the highly significant differences noted in $V_{\text{max}}$, between patients with normal ejection fractions and patients with low ejection fractions strongly suggests that contractility plays the major role in determining $V_{\text{max}}$ under conditions of this study. These findings support the use of this technic to provide a measure of ventricular contractility and to aid in the evaluation of overall ventricular performance.

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