Effect of Atrial and Ventricular Tachycardia on Cardiac Oxygen Consumption

By Henry S. Badeer, M.D., and Khalil A. Feisal, M.D.

Clinical experience has shown that ventricular tachycardia is a more serious arrhythmia than supraventricular tachycardia. A few observations in man made during cardiac catheterization suggest that this difference is partly related to the marked decrease in cardiac output and arterial pressure that occur in ventricular tachycardia. Studies on experimentally induced atrial and ventricular tachycardia in dogs have confirmed these findings.

In 1925, Wiggers presented evidence that ventricular contraction is less synchronous when the excitation originates from the surface of the ventricles than when the contraction follows stimulation over the normal conduction system, and more recently Gilmore and associates and Nakano have extended these observations in an attempt to explain the different effects of the two arrhythmias. As a further step in the elucidation of this problem, it seemed to us that a comparison of the myocardial energy expenditure in the two types of tachycardia would be of importance. The present communication describes a study of the effect of electrically induced moderate ventricular tachycardia and atrial tachycardia of equal rate on myocardial oxygen consumption.

Methods

Experiments were done on the dog heart-lung preparation, as modified in our laboratory for the purpose of determining cardiac oxygen uptake by the direct Fick method. Anesthesia was induced with sodium pentobarbital (30 mg/kg intraperitoneally) in mongrel dogs of both sexes, weighing 10 to 20 kg. The lungs were ventilated with 100% oxygen at a tidal volume of 150 to 200 ml and a frequency of 14 per minute. Total coronary flow (with the exception of the small Thebesian drainage into the left heart) was monitored with a Shipley-Wilson rotameter in the left pulmonary artery. A damping air chamber in the circuit reduced the flow pulsations. Arterial pressure was measured with a Sanborn transducer (model 267A) and both coronary flow and arterial pressure were recorded on a Sanborn direct-writing oscillograph. Blood pressure was maintained constant at 100 mm Hg and left ventricular output at about 900 ml/min throughout the entire experiment. Since in this preparation all the coronary venous blood flowed through one lung, changes in pulmonary vascular resistance with the respiratory cycle tended to affect the record of coronary flow. To avoid this, the respiration pump was stopped for short periods to read the mean coronary flow and to obtain arterial and coronary venous blood samples.

After measuring systemic output, recording coronary flow, and obtaining blood samples, atrial tachycardia was induced by electrodes previously applied to the right auricular appendage. Monophasic pulses of 2 msec duration and 4 volts intensity were delivered from a Grass stimulator (model S4). Tachycardias ranged from 142 to 186 beats per minute depending upon the prevailing sinus rhythm. Arterial pressure and cardiac output were kept at control levels. Under steady state conditions, coronary flow measurement and blood samples were obtained as described above. Subsequently, the stimulation was discontinued. After a recovery period of about five minutes, data were again secured to determine cardiac oxygen consumption during sinus rhythm.

Finally, the heart was driven by electrodes previously applied to the anterolateral surface of the left ventricle near the apex, using the same stimulus voltage, duration and frequency as in atrial pacing. There was a tendency for cardiac output to drop by about 50 to 100 ml, and blood pressure to fall by about 5 mm Hg, and both were restored to control values. Under steady state conditions, coronary flow and blood samples were again secured.

At the end of each experiment, the right heart was filled with saline under high pressure to check for possible septal defects which would invalidate the measurement of total coronary flow in the left pulmonary artery. The sequence of atrial and ventricular tachycardia was reversed in successive experiments.
CARDIAC OXYGEN UPTAKE IN TACHYCARDIAS

FIGURE 1
Coronary flow in experiment no. 7 during sinus rhythm and during electrical stimulation of the right auricle and the left ventricle (arterial pressure held constant at 100 mm Hg).

Results

Table 1 summarizes the results of seven experiments. The average sinus frequency before atrial and ventricular tachycardia was 130 beats per minute and was increased by 27 and 28 beats per minute during atrial and ventricular pacing, respectively. In all experiments, ventricular tachycardia increased myocardial oxygen uptake to a greater extent than did atrial tachycardia (output and arterial pressure being the same). In experiment number 3 the frequency of the heart during atrial and ventricular tachycardia was only slightly more than that of the sinus rhythm because it was impossible to drive the heart at faster rates. Nevertheless, ventricular pacing caused an increase in oxygen uptake whereas atrial pacing did not. The average increase in oxygen uptake in ventricular tachycardia was 50% as compared with an increase of 11.5% in atrial tachycardia (table 2).
<table>
<thead>
<tr>
<th>Exp. no.</th>
<th>DOE Weight, sex*</th>
<th>Heart weight, g</th>
<th>Cardiac pacemaker, S-A</th>
<th>Heart rate, beats/min</th>
<th>Left vent. output, ml/min</th>
<th>Total coronary flow, ml/min/100 g</th>
<th>Arterial oxygen content, vol %</th>
<th>A-Vt oxygen difference, vol %</th>
<th>Myocardial oxygen uptake, ml/min/100 g</th>
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<tbody>
<tr>
<td>2</td>
<td>14 F</td>
<td>110.5</td>
<td>S-A</td>
<td>10.3</td>
<td>28.48</td>
<td>17.03</td>
<td>22.92</td>
<td>0.15</td>
<td>4.15</td>
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<td>3</td>
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<td>107.5</td>
<td>S-A</td>
<td>9.0</td>
<td>28.29</td>
<td>15.01</td>
<td>23.47</td>
<td>0.50</td>
<td>5.60</td>
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<tr>
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<td>10 M</td>
<td>108.5</td>
<td>S-A</td>
<td>9.0</td>
<td>28.29</td>
<td>12.30</td>
<td>20.84</td>
<td>1.00</td>
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<td>S-A</td>
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<td>7</td>
<td>15 M</td>
<td>120</td>
<td>S-A</td>
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<td>28.48</td>
<td>12.30</td>
<td>23.58</td>
<td>0.77</td>
<td>5.60</td>
</tr>
</tbody>
</table>

TABLE 2
Changes in Heart Rate, Coronary Flow and Myocardial Oxygen Consumption in Atrial and Ventricular Tachycardia (aortic pressure held constant at 100 mm Hg)

<table>
<thead>
<tr>
<th>Expt. no.</th>
<th>Control heart rate beats/min</th>
<th>Atrial tachycardia beats/min</th>
<th>Control heart rate beats/min</th>
<th>Ventricular tachycardia beats/min</th>
<th>Increase in coronary blood flow during</th>
<th>Increase in cardiac oxygen uptake during</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Atrial tachycardia %</td>
<td>Ventricular tachycardia %</td>
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<tr>
<td>2</td>
<td>126</td>
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<td>21.5</td>
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<tr>
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<td>142</td>
<td>112</td>
<td>142</td>
<td>22.0</td>
<td>55.9</td>
</tr>
<tr>
<td>8</td>
<td>122</td>
<td>152</td>
<td>120</td>
<td>155</td>
<td>14.3</td>
<td>53.0</td>
</tr>
<tr>
<td>Means</td>
<td>130</td>
<td>157</td>
<td>130</td>
<td>158</td>
<td>17.5</td>
<td>52.0</td>
</tr>
</tbody>
</table>

In every experiment, the coronary flow increased during tachycardia. Figure 1 shows the records obtained in one experiment. The average increase of coronary flow was 52% during ventricular tachycardia as compared with 17.5% during atrial tachycardia (table 2). On the other hand, the coronary arteriovenous oxygen difference did not change markedly nor show a definite pattern during either atrial or ventricular pacing.

Discussion
Available evidence shows that direct electrical stimulation of the surface of the left ventricle produces a less synchronous ventricular contraction than that resulting from the normally propagated excitation from the atria. This is presumably a consequence of the distinctly slower velocity of conduction in typical ventricular muscle tissue as compared with the velocity in the specialized conducting system of the mammalian ventricles. The cardiodynamic effects of ventricular tachycardia, as opposed to atrial tachycardia, have been analyzed recently by Gilmore and associates and by Nakano in open-chest dogs. These workers have demonstrated that in ventricular tachycardia the disturbed dynamics may differ from atrial tachycardia in the following respects: (1) Atrial systole may or may not contribute to ventricular filling depending upon the existing atrioventricular sequence. (2) There is a tendency toward some degree of mitral and tricuspid regurgitation. (3) Ventricular fractionate contractions are retarded and less synchronous. All these factors tend to reduce the volume of blood ejected into the arteries.

A comparison between the effect of ventricular and atrial tachycardia on myocardial oxygen consumption requires that variations in cardiac output and arterial pressure, which might occur to different degrees as a result of the two arrhythmias, be eliminated. The heart-lung preparation is advantageous for this purpose since it permits ready control of these variables, as shown in figure 1. Although the coronary blood flow and myocardial oxygen uptake are known to increase progressively during the course of an experiment in such a preparation, it was possible by working rapidly and by alternating the sequence of ventricular and atrial tachycardia in different experiments to minimize the influence of the time factor.

In our experiments, left ventricular stroke volume in ventricular tachycardia was kept equal to that in atrial tachycardia by proper adjustment of filling pressure. Under these experimental conditions, the increase in stroke oxygen uptake in ventricular tachycardia was nevertheless distinctly greater than in atrial tachycardia. A plausible explanation for this finding seems to be the prolongation of the period of systole due to the slowing of the phasic entry of myocardial fractions when the surface of the ventricle is stimulated. The increase in the duration of systole would increase the "tension-time index" which was
shown by Sarnoff et al. and others to be one of the major determinants of cardiac oxygen consumption. Another factor that might have affected the tension-time index is a possible difference in ventricular blood volume in the two types of tachycardia. A larger end diastolic volume in ventricular tachycardia would have resulted in increased myocardial tension throughout systole in accordance with the Laplace equation for a sphere, \( T = P \cdot r/2 \), and a consequent increase in oxygen consumption. However, we do not have data that would permit us to evaluate the possible role of this factor in our experiments.

Total coronary blood flow increased during atrial and ventricular tachycardia, but more so in the latter case. The mechanism underlying the increase in coronary flow in tachycardia has been debated and many factors play a role. The fact that the increase in coronary flow and in oxygen consumption was greater during ventricular tachycardia lends support to the view of several investigators that one of the most important factors determining coronary flow is the metabolic rate of the myocardium. Whether or not an increased production of vasodilator metabolites is involved in decreasing coronary vascular resistance when metabolic rate increases remains conjectural. It is interesting to note that Węgria and co-workers reported, in open-chest dogs, an increase of coronary flow in most experiments during both atrial and ventricular tachycardia despite a reduction of cardiac output and arterial pressure. This observation points to the potency of the coronary dilator mechanism(s) in tachycardia.

The implications of these findings in clinical tachycardias remain to be determined. Ventricular tachycardias are reported to cause more severe symptoms and hemodynamic disturbances than atrial tachycardias. This may, in part, be due to the fact that ventricular tachycardia is more often associated with severe organic heart disease. In addition, our findings suggest that heart failure is more likely to supervene in ventricular tachycardia because of the greater oxygen requirement of the heart in doing a given amount of pressure and volume work. This likelihood is further increased by the presence of disease of the coronary arteries.

**Summary**

Atrial tachycardia and ventricular tachycardia were induced by electric stimuli applied to the heart in the modified dog heart-lung preparation. Cardiac oxygen consumption was measured by the direct Fick method. Left ventricular output was kept constant at about 900 ml/min and arterial pressure at 100 mm Hg during both atrial and ventricular pacing. An increase of heart rate from 130 to 157 beats per minute in atrial tachycardia, and from 130 to 158 beats per minute in ventricular tachycardia, increased cardiac oxygen uptake by 11.5% and 50% respectively (mean of seven experiments). The greater energy expenditure of the heart in ventricular tachycardia, when compared with atrial tachycardia at equivalent heart rates, was attributed to the asynchrony of fractionate contractions of the ventricular muscle when stimuli are applied to the ventricular surface. Total coronary flow increased in both types of tachycardia, but more in ventricular tachycardia.

**Acknowledgment**

The authors thank Dr. George Fawaz, Department of Pharmacology, for proposing this study. Fawaz and Tutunji have reported that ouabain-induced ventricular tachycardia increased cardiac oxygen uptake by 76%, whereas an equal increase in heart rate due to auricular stimulation increased oxygen uptake by only 23%. It was uncertain, however, whether or not electrically induced tachycardia of ventricular origin would have an effect different from tachycardia of atrial origin.

**References**


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