The Deleterious Role of Tachycardia in Mitral Stenosis

By Djavad T. Arani, M.D., and Richard A. Carleton, M.D.

SUMMARY

Eleven patients with mitral stenosis and atrial fibrillation were studied during artificially induced tachycardia to determine the influence of rapid ventricular rates on hemodynamic variables. Significant elevation of pulmonary artery pressure occurred, but no increase in calculated pulmonary arterial resistance was found. Pulmonary arterial wedge pressures rose in each patient with an average increase of 7 mm Hg. Cardiac output fell at the fastest rate in each patient. These changes from rapid ventricular rates appear to account for the functional deterioration often encountered in patients with mitral stenosis with the advent of atrial fibrillation.

Additional Indexing Words:

Atrial fibrillation  Cardiac output  Pulmonary hypertension
Ventricular pacing

Patients with mitral stenosis commonly present a history of an abrupt increase in their symptoms coincident with the onset of atrial fibrillation. However, previous studies of the circulatory consequences of reversion from atrial fibrillation to normal sinus rhythm have suggested that only slight increases occur in cardiac output with reversion to normal sinus rhythm. These increases were statistically significant only when the ventricular rate had been rapid in atrial fibrillation. Two major factors seem likely to produce the clinical deterioration of patients with mitral stenosis with the advent of atrial fibrillation. First, coordinated atrial systole is lost and, second, the normal regulation of ventricular rate is lost. The result of removing effective atrial systole has previously been tested and found to produce remarkably little change in patients with mitral stenosis.

The present study was designed to test the importance of the second factor by determining the influence of different heart rates on the hemodynamic status of patients with pure mitral stenosis.

Material and Methods

Eleven patients with pure mitral stenosis underwent this study during diagnostic cardiac catheterization. No patient had disease of any other valve. Three patients had slight mitral regurgitation. Each patient had atrial fibrillation. Two patients (66-373 and 66-390) were asymptomatic; the other patients had experienced mild or moderate symptoms of pulmonary venous hypertension.

Each patient was studied while supine, at rest, and in the postabsorptive state. Mild sedation was achieved with meperidine, 50 to 75 mg, and sodium pentobarbital, 75 to 100 mg, administered intramuscularly 1 hour before study. Catheterization of the right side of the heart was performed in routine fashion; left ventricular catheterization was accomplished by retrograde catheter passage from the right brachial artery. All
in intracardiac pressures were recorded with bonded strain gauges and an optical galvanometer system which is critically damped at approximately 22 cycles/sec. Phasic pressures were measured directly. Mean pressures and mitral valvular gradients were measured by planimetric integration. Cardiac output was measured by the direct Fick method utilizing procedures previously described from this laboratory.*

The ventricular rate was controlled during each study by ventricular pacing. The tip of a bipolar pacing catheter* was placed in the apical portion of the right ventricle. The external terminals were connected to a battery-powered pacemaker which delivered 2-msec square wave impulses at 1.5 volts. Each patient was thought to be adequately digitalized with digoxin prior to the study and to have adequate digitalis effect. Regular ventricular pacing at a rate under 110/min without capture by the fibrillating atria was possible in nine of 11 patients. The existing frequency of impulses conducted through the A-V node prevented ventricular pacing at a rate of under 110 per minute in two patients. These two patients were studied at two ventricular rates; the other patients were studied at three ventricular rates. The sequence of paced rates was varied; four patients were studied initially at the fastest rate. The first measurements were made at the slowest rate in seven patients. Each rate was sustained for 5 to 8 minutes prior to the measurement of pressures for analysis and to the measurement of cardiac output.

Mitral valve areas were calculated by the standard Gorlin formula.4 Pulmonary arteriolar resistances were calculated by dividing the difference between mean pulmonary arterial and mean pulmonary arterial wedge pressures in dynes/cm² by cardiac output in cm³/sec.


Figure 1
Electrocardiogram, pulmonary arterial wedge pressure, and left ventricular pressure of patient 66-390. The broad QRS complexes initiated by ventricular pacing occur at rates of 93, 128, and 140/min. The mitral valve diastolic gradient and pulmonary arterial wedge pressure increased with increasing rate.
### Table 1

Data from Patients with Mitral Stenosis Studied at Different Ventricular Rates

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr), sex</th>
<th>BSA* (m²)</th>
<th>Heart rate (bts/min)</th>
<th>Oxygen consumption (L/min)</th>
<th>Arteriovenous difference (vol %)</th>
<th>Cardiac output (L/min/m²)</th>
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*BSA = body surface area.
†Groups I, II, and III. Group I: pacing rates, <110/min; group II: pacing rates, 110-139/min; group III, pacing rates, >139/min.
‡Left ventricular diastolic pressure obscured by artifact.

### Table 1 (continued§)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr), sex</th>
<th>Pulmonary artery pressure (mm Hg)</th>
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<th>Pulmonary arterial resistance (dyne·sec·cm⁻²)</th>
<th>Diastolic filling period</th>
<th>Mitral valve area (cm²)</th>
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§See first part of table 1 for footnotes as indicated.
Statistical analyses of differences in hemodynamic parameters were conducted using Student's *t* test.

**Results**

The results are presented in table 1. Representative pressure tracings from the left ventricle and pulmonary arterial wedge obtained in patient 66-390 are shown in figure 1.

**Heart Rate**

The studies have been divided into three groups. Group I included all studies conducted at a ventricular rate of less than 110 beats/min, group II included those between 111 and 139, and group III, those between 139 and 152 beats/min. The average rates within the three groups were 95, 124, and 146 beats/min.

**Cardiac Output**

Oxygen consumption showed minor changes which were not dependent on heart rate. The mean values were 230, 234, and 232 ml/min in the three groups, respectively. Seven of nine patients had a wider arteriovenous oxygen difference at the intermediate rate than at their slowest rate. Each patient had a significant widening of arteriovenous difference between their slowest and their fastest paced rates (*P* < 0.001). This increase reflected decreased pulmonary artery oxygen content in each instance. Cardiac output did not change significantly between the studies in group I and those in group II. However, each patient had a fall in cardiac output (*P* < 0.001) between the studies in group I and those in group III (fig. 2). The average cardiac output fell from 2.29 to 2.08 L/min/m². Correspondingly, there were striking decreases in stroke volume with increasing heart rate.

**Pulmonary Artery Pressure**

Pulmonary artery systolic pressure responded variably to increased ventricular rate. Nine of the 11 patients had a moderate increase in pulmonary artery systolic pressure from their slowest to their fastest paced rates. One individual (67-105) had his lowest pulmonary artery systolic pressure at the fastest rate. This was the first of three studies and suggests that pulmonary venous congestion occurred and progressed throughout the study in this patient.

Mean pulmonary artery pressure, however, uniformly increased between the slowest and the fastest paced rates regardless of the temporal sequence of paced rates used. The average increase in mean pressure of 7 mm Hg closely reflected the average change in pulmonary arterial wedge pressure.

**Pulmonary Arterial Wedge Mean Pressure**

The hemodynamic variable which most closely parallels the symptom of dyspnea in the presence of mitral stenosis is the left atrial pressure and its reflection, the pulmonary arterial wedge pressure. The trend of pulmonary arterial wedge pressure plotted against ventricular rate is shown in figure 3. There was an approximately linear increase

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**Figure 2**

Values for cardiac output in L/min/m² for each patient obtained at the slowest rate are compared with those obtained at the fastest rate. Cardiac output fell in each patient with increased ventricular rate (*P* < 0.001).
in this pressure with increasing heart rate. The mean values in the three groups of studies were 24, 27, and 31 mm Hg. It is noteworthy that pacing at a rate above 110 sufficed in the majority of patients to produce pulmonary arterial wedge pressures in excess of the usual plasma oncotic pressure of approximately 25 mm Hg. Frank pulmonary edema did not occur in any patient, but dyspnea appeared in four (66-373, 67-26, 67-67, and 67-125). Even patients with only moderate mitral stenosis, with mitral valve areas of more than 1.5 cm², developed striking left atrial and pulmonary venous hypertension at the fastest ventricular rates.

**Pulmonary Arteriolar Resistance**

No consistent changes occurred in pulmonary arteriolar resistance, reflecting the comparable increases in mean pulmonary artery and mean pulmonary arterial wedge pressures. The average value was 230 in group I, 310 in group II, and 300 dyne-sec cm⁻⁵ in group III. These data suggest that active pulmonary vasoconstriction did not occur as a result of increasing pulmonary venous hypertension.

**Mitral Orifice Parameters**

There were small increases in the pressure differential between the pulmonary arterial wedge and the left ventricular diastolic pressure in each patient between the slowest and the fastest ventricular rates. These increases in mitral valve gradient were approximately paralleled by linear decreases in the mitral diastolic filling period and by decreases in stroke volume. As a consequence of these balanced changes, calculated mitral valve areas remained essentially constant throughout each patient study.

**Left Ventricular Pressure**

There were only slight changes in left ventricular pressure throughout the course of this study. In general, faster rates were associated with lower left ventricular systolic pressure, reflecting the diminution in stroke volume. Left ventricular end-diastolic pressure showed no significant alterations as a function of increasing ventricular rate.

**Discussion**

The present data clearly demonstrate the deleterious effect of an uncontrolled ventricular rate in the presence of mitral stenosis. This effect is most clearly evident in two measures, both of which are important in the production of symptoms in patients with mitral stenosis. A significant diminution in cardiac output occurs when ventricular rate is accelerated from 95 to 146. There are concomitant widening of arteriovenous differences, diminution in pulmonary arterial oxygen saturations, and decreases in levels of tissue partial pressure of oxygen.

The approximately linear increase in pulmonary arterial wedge pressure which occurs with increased ventricular rate is paralleled most closely and appears to be causally related to the progressive decline in time available for mitral valve flow. The data in this regard are similar to the observations of Mitchell and co-workers⁵ who found in dogs that mean left atrial pressure increased more than left ventricular end-diastolic pressure with increasing mitral valve flow rates. Symptomatic pulmonary venous congestion can be confidently predicted when the ventricular rate accelerates as a result of atrial fibrillation.
in patients with only moderate mitral stenosis.

The present studies were conducted with the patients at rest. The tachycardia of exercise would be expected to have similar consequences. Moreover, the augmentation of demand for cardiac output with even mild exercise would accentuate these changes and produce even greater elevations of left atrial pressure.

Data previously reported have indicated that the hemodynamic role of the atria is measurable and hemodynamically significant in the absence of mitral valve disease. In the presence of mitral stenosis, however, the effectiveness of atrial systole was sharply attenuated; no significant change in cardiac output could be detected as a result of the removal of effective atrial systole.2

Thus, of the two major consequences of atrial fibrillation, only the increase in ventricular rate appears to be of major importance in the production of symptoms of pulmonary venous congestion in patients with mitral stenosis. Accordingly, the importance of controlling ventricular rate by reducing A-V nodal conduction with digitalis is reinforced.

Acknowledgment

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References

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