The Effects of Mitral Regurgitation on the Pattern of Instantaneous Aortic Blood Flow

Clinical and Experimental Observations

By Ronald C. Elkins, M.D., Andrew G. Morrow, M.D., John S. Vasko, M.D., and Eugene Braunwald, M.D.

SUMMARY

Instantaneous ascending aortic blood flow was recorded at operation in five patients with severe, pure mitral regurgitation, and in nine dogs in which mitral regurgitation was produced experimentally under controlled conditions. In both the patients and the experimental animals, the pattern of aortic flow was abnormal during mitral regurgitation; peak flow occurred early, the percentage of total forward flow was abnormally high during the first half of the ejection period and abnormally low during the last quarter. When stroke volume, heart rate, and aortic pressure were maintained constant, mitral regurgitation also resulted in increases in peak flow, mean ejection rate, and maximum acceleration of flow. These abnormalities of the aortic flow patterns are attributable to inability of the ventricle to sustain forward ejection during late systole in the presence of severe regurgitant flow through the mitral valve.

Additional Indexing Words:
Left ventricular pressure Peak flow

Total forward flow Ejection period

Methods

Experimental Observations

Adult mongrel dogs, weighing 18 to 25 kg, were anesthetized with intravenous chloralose (60 mg/kg), and ventilation was maintained through auffed endotracheal tube by an intermittent positive pressure respirator supplying room air. A bilateral transverse thoracotomy was made through the fourth intercostal spaces, and heparin (3 mg/kg) was administered intravenously. The sino-atrial node was crushed, and the heart rate was fixed and controlled by electrical stimulation of the right atrium at a constant rate between 113 and 194 beats/min. Body temperature, measured from an esophageal thermistor, was maintained between 36.5 and 37.5°C by means of a heat exchanger in the extracorporeal circuit. Left atrial, left ventricular, and central aortic pressures were measured through semirigid catheters attached to Statham P23Db pressure transducers, and the transducer of an electromagnetic flowmeter (Biotronix BL-410) was placed around the ascending aorta.

The right heart bypass system, shown diagrammatically in figure 1, was utilized to permit independent control of systemic flow and aortic pressure. The extracorporeal circuits were

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Diagrammatic representation of the right heart bypass preparation utilized for determining the volume-temporal relationships of ascending aortic blood flow during mitral regurgitation. Heart rate was maintained constant by electrical stimulation of the right atrium (RA). Left ventricular (LV) output was maintained constant by the extracorporeal circuit shown, and aortic pressure was controlled by use of the variable volume reservoir attached to the circulation through both femoral arteries.

Primed with fresh, heparinized, homologous blood. Following the institution of right heart bypass and an appropriate period of stabilization, control measurements of instantaneous aortic blood flow and ascending aortic, left atrial, and left ventricular pressures were made. Mitral regurgitation was then created by passing a cannula and obturator through the mitral valve and into the left ventricle (fig. 2); the instrument was similar to that described by Wiggers and Feil. Aortic flow, left atrial, and left ventricular pressures were again recorded during mitral regurgitation, after a steady hemodynamic state was achieved. Heart rate, stroke volume, and aortic pressure were maintained constant during the control period and the period of valvular regurgitation. In four animals, the aortic flow and left ventricular pressure signals were fed into a Donner 3400 computer, and stroke volume, acceleration of flow, stroke power, and stroke work...
Clinical Observations

Five patients with mitral regurgitation were studied in the course of corrective operations. In all, severe symptoms indicated the necessity of mitral valve replacement. The diagnosis of severe, pure mitral regurgitation had been established at preoperative hemodynamic and angiographic study and was confirmed by the operative appearance of the mitral valve. All patients had normal aortic valve function.

The general operative methods utilized for mitral valve replacement have been previously described. After exposure of the ascending aorta, the hinged or keyed transducer of a sine-wave electromagnetic flowmeter (Medicon-K2004) was

were instantaneously computed. At the conclusion of each study the flowmeter was calibrated with measured flows of normal saline, as previously described.2,3

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placemed around the aorta immediately below the origin of the innominate artery. A transducer with a lumen several millimeters less than the measured external diameter of the aorta was utilized, and the transducer was left in place throughout the operative procedure. Left ventricular and left atrial pressures were measured with 20-gauge needles and rigid (150 cm) saline-filled nylon catheters (1.7 mm I.D.), attached to equisensitive Statham P23Db pressure transducers. The pressure pulses and the instantaneous aortic flow pattern were recorded simultaneously on magnetic tape, and subsequently reproduced with a photographic recorder operated at appropriate paper speeds. Observations were made before the institution of cardiopulmonary bypass and after a period of circulatory stabilization following mitral valve replacement. The flowmeter was calibrated as described above.

**Calculations**

The methods by which the measured and calculated components of the flow pattern were determined were similar in the clinical and experimental studies. Zero flow was determined from the flow record during diastole. The systolic ejection period was the time (sec) from the onset of forward flow to that of zero flow. Systolic seconds per minute was the product of the systolic ejection period and the heart rate. Peak flow was measured from the flow record, and expressed as ml/sec. The time-to-peak flow (sec) was the interval between onset of forward flow and peak flow. Time-to-peak flow was also expressed as a percentage of the total duration of the systolic ejection period. The mean ejection rate (ml/systolic second) was the stroke volume divided by the systolic ejection time. The first derivative of instantaneous velocity of blood flow (acceleration) was determined by use of a resistance-capacitance (RC) differentiating circuit. The stroke volume was determined by planimetric or electronic integration of the area of the flow curve above the line of zero flow. The total systolic ejection period was divided into four equal periods, and the flow during each period was determined by planimetric or electronic integration and expressed as the percentage of total stroke volume per one fourth of the systolic ejection period. All measurements of pressure and flow presented are averages of five or more consecutive cardiac cycles.

**Hemodynamic Observations in Nine Dogs under Control Conditions and after the Production of Mitral**

<table>
<thead>
<tr>
<th>Animal</th>
<th>Condition</th>
<th>Heart rate (beats/min)</th>
<th>Forward stroke vol. (ml)</th>
<th>Mean aortic pressure (mm Hg)</th>
<th>Syst. eject. period (sec)</th>
<th>Peak eject. time (sec)</th>
<th>Peak eject. time/total eject. time (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control</td>
<td>146</td>
<td>12.3</td>
<td>105</td>
<td>0.178</td>
<td>0.101</td>
<td>56.1</td>
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<td></td>
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<td>146</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.168</td>
<td>0.081</td>
<td>48.2</td>
</tr>
<tr>
<td>2</td>
<td>Control</td>
<td>123</td>
<td>12.8</td>
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<td>0.188</td>
<td>0.104</td>
<td>55.4</td>
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<tr>
<td></td>
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<td>123</td>
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<td>&quot;</td>
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<td>3</td>
<td>Control</td>
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<td>13.8</td>
<td>110</td>
<td>0.140</td>
<td>0.075</td>
<td>53.6</td>
</tr>
<tr>
<td></td>
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<td>140</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.140</td>
<td>0.058</td>
<td>41.4</td>
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<tr>
<td>4</td>
<td>Control</td>
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<td>7.4</td>
<td>105</td>
<td>0.110</td>
<td>0.055</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
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<td>194</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.105</td>
<td>0.055</td>
<td>39.3</td>
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<td>5</td>
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<td>11.0</td>
<td>100</td>
<td>0.151</td>
<td>0.078</td>
<td>51.6</td>
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<td>136</td>
<td>&quot;</td>
<td>&quot;</td>
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<td>0.071</td>
<td>48.2</td>
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<tr>
<td>6</td>
<td>Control</td>
<td>176</td>
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<td>105</td>
<td>0.120</td>
<td>0.045</td>
<td>37.5</td>
</tr>
<tr>
<td></td>
<td>MR</td>
<td>176</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.110</td>
<td>0.045</td>
<td>40.9</td>
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<tr>
<td>7</td>
<td>Control</td>
<td>113</td>
<td>17.1</td>
<td>90</td>
<td>0.150</td>
<td>0.060</td>
<td>40.0</td>
</tr>
<tr>
<td></td>
<td>MR</td>
<td>113</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.135</td>
<td>0.045</td>
<td>33.3</td>
</tr>
<tr>
<td>8</td>
<td>Control</td>
<td>129</td>
<td>11.6</td>
<td>95</td>
<td>0.145</td>
<td>0.066</td>
<td>45.5</td>
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<tr>
<td></td>
<td>MR</td>
<td>129</td>
<td>&quot;</td>
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<tr>
<td>9</td>
<td>Control</td>
<td>130</td>
<td>9.8</td>
<td>95</td>
<td>0.150</td>
<td>0.070</td>
<td>46.7</td>
</tr>
<tr>
<td></td>
<td>MR</td>
<td>130</td>
<td>&quot;</td>
<td>&quot;</td>
<td>0.130</td>
<td>0.060</td>
<td>46.1</td>
</tr>
</tbody>
</table>

Mean ± 1 se  Control 0.148 ±0.005 ±0.005 ±2.1
MR 0.136 ±0.04 ±0.005 ±2.3
P <0.01 <0.05 <0.05

*The differentiating circuit was not calibrated in these experiments, and maximal acceleration is expressed in arbitrary units (mm deflection).

†SEP = systolic ejection period.
### Results

The detailed observations made in nine dogs before and after the creation of mitral regurgitation are presented in table 1. When heart rate, aortic pressure, and forward left ventricular stroke volume were constant, the average changes in the pattern of aortic flow which accompanied the production of mitral regurgitation were: (1) systolic seconds per minute decreased from 20.7 ± 0.8 sec to 19.1 ± 1.0 sec \( (P < 0.01) \); (2) the time-to-peak flow decreased from 0.073 ± 0.005 to 0.063 ± 0.005 \( (P < 0.05) \); (3) peak flow increased from 137 ± 4.7 to 148 ± 4.8 ml/sec \( (P < 0.01) \); (4) the mean ejection rate rose from 78.9 ± 4.9 to 86.1 ± 5.4 ml/systolic second \( (P < 0.02) \); (5) peak acceleration of flow was measured in eight animals, and increased from 12.5 ± 0.9 to 14.1 ± 1.0 \( (P < 0.01) \); and (6) the portion of flow which occurred during the first half of the ejection period increased from 43.7 ± 2.1% to 51 ± 2.3% \( (P < 0.01) \), while that during the last one fourth of the ejection period decreased from 17.2 ± 1.2% to 12.5 ± 1.3% \( (P < 0.01) \). Stroke work and power were computed in four animals; peak power increased by 12.8 ± 2.2% with the creation of mitral regurgitation, while stroke work remained constant. Portions of typical recordings made in two dogs during the control state and after the production of mitral regurgitation are shown in figures 3 and 4.

The hemodynamic observations made in the five patients are summarized in table 2 and representative records of aortic flow, left atrial, and left ventricular pressures obtained in one patient before and after mitral valve replacement are reproduced in figure 5. In every patient, the percentage of flow which occurred during the first half of the systolic ejection period was greater during mitral regurgitation than during this same period following valve replacement. Flow during the last one fourth of the systolic ejection period, however, was significantly lower during regurgitation than after valve replacement. The absolute duration of ejection and the systolic

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Table 2
Intraoperative Hemodynamic Observations in Five Patients with Severe, Pure Mitral Regurgitation Before (Preop) and After (Postop) Mitral Valve Replacement

<table>
<thead>
<tr>
<th>Patient</th>
<th>Condition</th>
<th>Heart rate (beats/min)</th>
<th>Rhythm</th>
<th>Mean aortic pressure (mm Hg)</th>
<th>Forward stroke vol. (ml)</th>
<th>Syst. eject. period (sec)</th>
<th>Peak eject. time/sec</th>
<th>Percent of forward LV stroke vol. ejected during each 1/4th of SEP</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.S.</td>
<td>Preop</td>
<td>91</td>
<td>AF</td>
<td>50</td>
<td>20.1</td>
<td>0.235</td>
<td>0.090</td>
<td>38.0</td>
<td>13.8</td>
<td>42.9</td>
<td>32.4</td>
<td>9.9</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>90</td>
<td>AF</td>
<td>85</td>
<td>36.8</td>
<td>0.258</td>
<td>0.128</td>
<td>49.6</td>
<td>10.7</td>
<td>34.2</td>
<td>35.8</td>
<td>19.2</td>
</tr>
<tr>
<td>A.L.</td>
<td>Preop</td>
<td>84</td>
<td>NSR</td>
<td>70</td>
<td>21.9</td>
<td>0.235</td>
<td>0.111</td>
<td>47.2</td>
<td>11.2</td>
<td>36.8</td>
<td>35.9</td>
<td>15.9</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>75</td>
<td>Nodal</td>
<td>80</td>
<td>34.5</td>
<td>0.275</td>
<td>0.135</td>
<td>49.0</td>
<td>13.2</td>
<td>34.2</td>
<td>35.3</td>
<td>17.4</td>
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<tr>
<td>E.B.</td>
<td>Preop</td>
<td>55</td>
<td>AF</td>
<td>65</td>
<td>20.1</td>
<td>0.237</td>
<td>0.085</td>
<td>35.9</td>
<td>17.8</td>
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<td>32.1</td>
<td>10.5</td>
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<tr>
<td></td>
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<td>72</td>
<td>Nodal</td>
<td>65</td>
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<td>0.241</td>
<td>0.104</td>
<td>43.1</td>
<td>14.3</td>
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<tr>
<td>J.S.</td>
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<td>81</td>
<td>NSR</td>
<td>78</td>
<td>58.3</td>
<td>0.274</td>
<td>0.106</td>
<td>38.7</td>
<td>18.9</td>
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<tr>
<td></td>
<td>Postop</td>
<td>94</td>
<td>NSR</td>
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<td>70.3</td>
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<td>10.4</td>
<td>37.1</td>
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<tr>
<td>B.M.</td>
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<td>58</td>
<td>NSR</td>
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<td></td>
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<tr>
<td>Mean</td>
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<td></td>
<td></td>
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<td>41.2</td>
<td>14.5</td>
<td>40.0</td>
<td>33.7</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>51.6</td>
<td>10.8</td>
<td>33.0</td>
<td>37.0</td>
<td>19.3</td>
</tr>
</tbody>
</table>

Abbreviations: SEP = systolic ejection period; AF = atrial fibrillation; NSR = normal sinus rhythm.

Intraoperative hemodynamic observations in five patients with severe, pure mitral regurgitation before (Preop) and after (Postop) mitral valve replacement were recorded. The hemodynamic changes were characterized by a decrease in mean aortic pressure and an increase in stroke volume. The peak systolic pressure was observed during the ejection period. The results showed that the stroke volume increased with the increase in heart rate. The peak systolic pressure was higher in the postoperative period compared to the preoperative period. The peak ejection time was shorter in the postoperative period compared to the preoperative period. The percent of forward LV stroke volume ejected during each 1/4th of the ejection period was also recorded. The results showed that the percent of forward LV stroke volume ejected during each 1/4th of the ejection period increased in the postoperative period compared to the preoperative period. The hemodynamic changes were correlated with the degree of mitral regurgitation.

Comment
An abnormal pattern of instantaneous aortic blood flow was demonstrated in each of the five patients after valve replacement. The pattern of flow was characterized by a decrease in aortic flow during the late ejection period and an increase in aortic flow during the early diastolic period. The peak ejection time was observed during the ejection period. The increase in stroke volume was observed during the ejection period. The peak systolic pressure was observed during the ejection period. The percent of forward LV stroke volume ejected during each 1/4th of the ejection period increased in the postoperative period compared to the preoperative period.

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PATTERN OF AORTIC FLOW

Figure 4

Segments of continuous recordings of aortic flow and intracardiac pressures in a dog during control conditions and after the creation of mitral regurgitation. Heart rate, stroke volume, and aortic pressure were maintained constant. During mitral regurgitation the characteristic abnormalities of the aortic flow pattern were evident. In addition, increases in left atrial pressure, left ventricular end-diastolic pressure, and rate of change of left ventricular pressure (dp/dt) resulted.

decline in left ventricular pressure. The abnormal patterns of both forward aortic flow and left ventricular pressure are attributable to inability of the ventricle to maintain ejection of blood into the aorta during that portion of the cardiac cycle when regurgitant flow through the mitral valve is maximal.

When mitral regurgitation was induced in the experimental animals, in which forward stroke volume was maintained constant by means of the extracorporeal pump, an increase in the acceleration of flow into the aorta was noted, and the achievement of peak aortic flow occurred earlier than normal during the systolic ejection period. In these experiments, the increase of total left ventricular stroke volume (the sum of forward and regurgitant flow) which occurred was attributable in part to the operation of the Frank-Starling mechanism, that is, to elevations of left ventricular end-diastolic pressure and volume (figs. 3 and 4). Earlier studies on the effects of acutely induced, metered, mitral regurgitation in the dog indicated that

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Simultaneous records of instantaneous aortic flow, left atrial (LA) and left ventricular (LV) pressures obtained in patient J.S. before and after mitral valve replacement for severe mitral regurgitation. Also plotted are the percentages of the total forward stroke volume ejected during each one fourth of the systolic ejection period (SEP). During mitral regurgitation (pre-valve replacement), a significantly larger fraction of forward flow occurred during the first half of the ejection period and an abnormally small amount during the last one fourth. The reduction of mean left atrial pressure and the change in the contour of the left atrial pressure pulse which followed valve replacement are also evident.

this valvular lesion, in addition to augmenting left ventricular end-diastolic volume, also reduced ventricular systolic volume below control levels. Perhaps, forward flow into the high pressure aorta cannot be sustained during late systole in mitral regurgitation because of the diminished shortening capacity of the left ventricular fibers, which are already shortened to subnormal levels.

The rapid early ejection of blood into the aorta in patients with mitral regurgitation is reflected in the sharp, water-hammer-like arterial pulse which is characteristic of this condition. In addition, clinical evidence of an inability to sustain forward flow into the aorta is not uncommon. Brigden and Leatham, for example, called attention to the occurrence of unusually wide splitting of the second heart sound in patients with mitral regurgitation, and Perloff and Harvey showed that 14 of 33 patients with pure mitral regurgitation had abnormally wide splitting of the second sound. Phonocardiographic analyses indicated that this was associated
Single aortic flow complexes recorded in one animal under control conditions and during mitral regurgitation. They were photographically superimposed for comparison. Stroke volume, heart rate, and aortic pressure were identical. During mitral regurgitation the duration of ejection decreased, peak flow and acceleration of flow increased, and a larger fraction of the stroke volume was ejected during the first half of the ejection period.

with premature closure of the aortic valve, that is, a reduction in the duration of left ventricular systole.

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The Effects of Mitral Regurgitation on the Pattern of Instantaneous Aortic Blood Flow Clinical and Experimental Observations
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