Importance of Left Atrial Function in Patients with Myocardial Infarction

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SUMMARY Left atrial function was evaluated in patients with and without remote myocardial infarction. The simultaneous left atrial pressure recording and left atrial and left ventricular cineangiograms were obtained with a catheter-tip micromanometer. The pressure-volume curve of the left atrium was composed of an A-loop and a V-loop. The ratio of active atrial emptying to left ventricular stroke volume in patients with myocardial infarction was significantly larger than that in normal subjects (42 ± 12% vs 29 ± 10%, p < 0.05). The left atrial work was also significantly greater in patients with myocardial infarction (1690 ± 717 mm Hg·ml) than in normal subjects (940 ± 426 mm Hg·ml, p < 0.05). The ratio of active atrial emptying to left ventricular stroke volume and left atrial work were significantly related in both normal subjects and patients with myocardial infarction (γ = 0.72, p < 0.01). The left ventricular ejection fraction correlated inversely with left atrial work (γ = −0.5, p < 0.05). Left atrial work also showed a significant linear correlation with left atrial work before active atrial emptying (γ = 0.82, p < 0.01).

We conclude that the left atrial contribution to left ventricular function is increased in patients with remote myocardial infarction. This left atrial contribution to the left ventricle is attributed to the Frank-Starling mechanism in the left atrium.

LEFT ATRIAL function and its hemodynamic importance for overall cardiac performance have been discussed.1–13 The left atrium may serve as a conduit for the passage of blood from the pulmonary veins to the left ventricle during early left ventricular filling, as a reservoir for storing blood during left ventricular systole, and as a contractile chamber for augmentation of left ventricular filling. Understanding each of these functions and the contribution of the left atrium to left ventricular function in normal and diseased hearts is important.

In this report, we analyze left atrial pressure-volume relationships in patients with remote myocardial infarction and discuss the importance of left atrial function.

Methods

Data were obtained during diagnostic cardiac catheterization in two groups of patients. The normal group consisted of eight patients who had no coronary, valvular or congenital heart disease and were hemodynamically normal. These patients were referred for diagnostic cardiac catheterization to evaluate chest pain. The myocardial infarction group consisted of 10 patients who had a documented remote transmural myocardial infarction, and no other associated heart disease. The clinical data for each patient are listed in table 1. All patients were in normal sinus rhythm and had a normal PQ interval on the ECG.

All patients were studied in the fasting state at rest. Premedication with 10 mg of diazepam and prophylactic antibiotics were routinely administered. Cardiac medications were discontinued for at least 2 days before the study, except for sublingual nitroglycerin, which was allowed for anginal attack, but withheld 12 hours before the study. A Millar catheter-tip manometer (Model PC-484A, piggtail) was used for pressure measurement and cineangiography. The transducer was calibrated electronically against mercury at the beginning of each study. The zero shift during the procedure was adjusted by comparison with the pressure obtained simultaneously from the fluid-filled catheter connected to the Statham P23ID pressure transducer. The calibration was reestablished after withdrawal of the catheter.

The catheter was introduced through the brachial artery using the guide wire. In the shallow right anterior oblique projection, the catheter was advanced through the aortic valve into the left ventricle. The catheter tip was placed near the mitral valve. A loop was formed in the left ventricle, and the catheter was directed toward the mitral valve and passed into the left atrium. In the preliminary study, no mitral regurgitation was observed during left ventriculography with the catheter in the left atrium. Therefore, we ignored the artifactual influence of the retrograde catheter across the mitral valve. Biplane cineangiograms (anteroposterior and lateral projections) were obtained at 60 frames/sec with a 35-mm Arritechno cine camera mounted on a 25-cm image intensifier (Siemens Cardoskop U). Thirty milliliters of Urografin-76 (Schering) were injected directly into the left atrium at a rate of 10 ml/sec at the end of a normal inspiration. During the left atriogram, left atrial pressure was recorded on an Electronics for Medicine VR12 recorder at a paper speed of 100 mm/sec. Frame number and ECG signals were recorded on each cine film. After the study, a 1-cm² cross-hatched grid was filmed at the same distance from the x-ray tube and image intensifier as were the left atrial and left ventricular cavities.

Left atrial angiographic volumes were calculated as

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TABLE 1. Clinical Data

<table>
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<th>Age (years)</th>
<th>Sex</th>
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<th>Months after MI</th>
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<tr>
<td>1</td>
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<td>55</td>
<td>M Atypical chest pain</td>
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<td>8</td>
<td>51</td>
<td>F Atypical chest pain</td>
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<tr>
<td>Mean ± SD</td>
<td>49 ± 13</td>
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MI group

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<th>Sex</th>
<th>Diagnosis</th>
<th>Months after MI</th>
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<td>M Anteroseptal MI</td>
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<td>11</td>
<td>65</td>
<td>M Anteroseptal MI</td>
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<td>67</td>
<td>M Inferior MI</td>
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<tr>
<td>13</td>
<td>65</td>
<td>M Anterolateral MI</td>
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<td>47</td>
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<td>47</td>
<td>M Anterior MI</td>
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<td>48</td>
<td>M Anteroseptal MI</td>
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</tr>
<tr>
<td>17</td>
<td>57</td>
<td>M Anterior MI</td>
<td>3</td>
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<td>18</td>
<td>56</td>
<td>M Anteroseptal MI</td>
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<tr>
<td>Mean ± SD</td>
<td>55 ± 8</td>
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Abbreviation: MI = myocardial infarction.

described by Sauter and co-workers.\textsuperscript{14} Biplane left atrial angiograms were traced during one cardiac cycle on a 35-mm Vanguard projector. The left ventricular volumes in end-systolic and end-diastolic biplane frames were calculated as previously described.\textsuperscript{15, 16} The same cardiac cycle was analyzed for left atrial pressure, left atrial volume and left ventricular volume. These individual left atrial volume measurements were plotted sequentially for one cardiac cycle, and connected by a line. At a film speed of 60 frames/sec, a volume observation was made every 16.7 msec. Left atrial volume changes were related to left atrial pressure changes with time common to both. A pressure-volume loop was then constructed by plotting each volume observation and the corresponding pressure using a specially programmed computer (Cardias GP 3000A, NAC).

Statistical analysis was done using the unpaired \( t \) test to compare the normal group and the myocardial infarction group. We calculated the best-fit linear regression equation by the method of least squares and the correlation coefficient \( (\gamma) \) for the pairs of data. A \( p \) value less than 0.05 was considered significant.

Results

The instantaneous changes of left atrial pressure and left atrial volume during one cardiac cycle in a representative normal subject are shown in the upper panel of figure 1. Volume changes can be divided into four phases. During phase 1, beginning with mitral valve closure, blood flows into the left atrium through the pulmonary veins, producing an increase in volume (atrial filling), the maximal level of which is the maximal left atrial volume. The increase in volume is accompanied by a continuous pressure rise ("v" wave). Phase 2 is initiated by the opening of the mitral valve. Atrial volume begins to decrease (passive atrial emptying), accompanied by a parallel fall in left atrial pressure. During phase 3, the volume remains relatively constant (atrial diastasis) and atrial pressure increases sharply. Atrial volume starts to decrease with the beginning of phase 4 (active atrial emptying), during which the peak systolic pressure is attained ("a" wave). The minimal volume at the end of this phase is the minimal left atrial volume.

The pressure-volume relationship is depicted in the lower panel of figure 1. The curve forms a double loop, with the beginning of phase 1 situated at the left lower corner. During phase 1, the curve is directed upward and to the right. After maximal diastolic pressure and volume are reached, the curve turns clockwise and downward, corresponding to phase 2. Subsequently, the curve proceeds almost parallel to the y-axis (phase 3), for systolic atrial pressure increases while the vol-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{(top) Instantaneous changes of left atrial pressure (LAP) and left atrial volume (LAV) during one cardiac cycle, starting from the onset of QRS on the ECG. (bottom) Pressure-volume relationship for the cardiac cycle in upper panel.}
\end{figure}
ume does not change. After the rising curve has closed the first loop (V-loop) by crossing the segment corresponding to atrial filling, the curve turns counterclockwise and closes the second loop (A-loop).

The hemodynamic data are shown in table 2. The cardiac cycle length and peak systolic pressure were not significantly different in the normal subjects and patients with myocardial infarction. The left ventricular end-diastolic and end-systolic volumes were significantly greater in patients with myocardial infarction than in normal subjects (169 ± 40 vs 117 ± 21 ml and 94 ± 45 vs 32 ± 14 ml, respectively; both p < 0.01). The left ventricular ejection fraction in patients with myocardial infarction was significantly lower than that in normal subjects (47 ± 16% vs 73 ± 8%, p < 0.001). However, there was no significant difference in the stroke volume between normal subjects and patients with remote myocardial infarction.

The peak pressure of the left atrial "a" wave and the nadir pressure of the x descent in normal subjects were not significantly different from those in patients with myocardial infarction. The maximal and minimal left atrial volumes, and their difference, left atrial volume change, in patients with myocardial infarction were not significantly larger than those in normal subjects. The left atrial volume before active atrial emptying, corresponding to the preload for atrial contraction, was significantly increased in patients with myocardial infarction compared with that in normal subjects (65 ± 21 vs 47 ± 10 ml; p < 0.05). The volume of left atrial passive emptying was calculated by subtracting the left atrial volume before active atrial emptying from the maximal left atrial volume. There was no significant difference in the mean volume of left atrial passive emptying between normal subjects and patients with myocardial infarction. The volume of left atrial active emptying was calculated by subtracting the minimal left atrial volume from the left atrial volume before active atrial emptying. In patients with myocardial infarction, the volume of left atrial active emptying was not significantly larger than that in normal subjects in the pressure-volume relationship, the area of the A-loop was expressed as the left atrial work of active atrial emptying. The left atrial work in patients with myocardial infarction was significantly larger than that in normal subjects (1690 ± 717 vs 940 ± 426 mm Hg·ml; p < 0.05).

The ratio of left atrial volume change to left ventric-
TABLE 2. (Continued)

<table>
<thead>
<tr>
<th>VA-Vmn (ml)</th>
<th>VC (ml)</th>
<th>Work (mm Hg·ml)</th>
<th>SV (%)</th>
<th>SV (%)</th>
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<td>±8</td>
<td>±8</td>
<td>±426</td>
<td>±10</td>
<td>±5</td>
<td>±10</td>
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26          | 43      | 896             | 63     | 25     | 38            |
30          | 46      | 2032            | 69     | 24     | 45            |
25          | 28      | 1312            | 47     | 5      | 42            |
39          | 46      | 2080            | 63     | 10     | 53            |
40          | 61      | 2992            | 90     | 31     | 59            |
32          | 49      | 2320            | 61     | 21     | 40            |
32          | 50      | 1184            | 39     | 14     | 25            |
19          | 41      | 672             | 52     | 28     | 24            |
33          | 54      | 1392            | 92     | 36     | 56            |
29          | 53      | 2016            | 71     | 32     | 39            |
31          | 47      | 1690            | 65     | 23     | 42            |
±6          | ±9      | ±717            | ±17    | ±10    | ±12           |
NS          | NS      | <0.05           | <0.05  | NS     | <0.05         |

In the present study, the left ventricular end-systolic and end-diastolic volumes were larger in patients with myocardial infarction than in normal subjects. However, the stroke volume was maintained in patients with myocardial infarction compared with that in normal subjects. There have been many studies on the left atrial contribution to left ventricular filling and stroke volume.5-8,17,18 During left ventricular filling, the left atrium has phases of passive atrial emptying, atrial diastasis and active atrial emptying in the present study (fig. 1) that were consistent with the demonstration in a previous study.20 The left atrial volume change does

Discussion

Patients with remote myocardial infarction often have some form of disturbance of myocardial function.
not provide a measure of blood entering the left ventricle from the left atrium during left ventricular diastole. In the phase of passive atrial emptying and atrial dias-
tasis, blood also flows from the pulmonary veins to the left ventricle. During active atrial emptying, some blood may flow back to the pulmonary veins. The contribution of left atrial contraction to left ventricular filling or stroke volume has been studied either by left atrial volumetric determinations or by left ventricular volumetric determinations during atrial contraction. In the present study, left atrial volumetric determinations showed that the ratio of left atrial volume change to left ventricular stroke volume in normal subjects was 48%. The ratios of passive atrial emptying and active atrial emptying to left ventricular stroke volume were 19% and 29%, respectively. These results are in reasonable agreement with previous reports of left atrial volumetric determinations, and also with the reports of left ventricular volumetric determinations. In patients with myocardial infarction, the ratio of left atrial volume change to left ventricular stroke volume was higher than that in normal subjects. The ratio of active atrial emptying to left ventricular stroke volume was significantly higher in patients with myocardial infarction than in normal subjects. Rahimtoola et al. also observed the relationship of left atrial function to left ventricular filling in patients with myocardial infarction. By the left ventricular volumetric determinations, they concluded that left atrial contraction made a larger contribution to left ventricular filling and left ventricular stroke volume in patients with myocardial infarction than in the controls.

Grant et al. reported the pressure-volume figure of the left atrium in the normal subjects and patients with various heart disease. The left atrial pressure-volume curves were composed of two loops, an A-loop and a V-loop. In the present study, we observed the instantaneous change of left atrial pressure and volume using the catheter-tip micromanometer. The pressure-volu-

tions were composed of two loops (fig. 1). The A-loop was expressed as a function of the contractile chamber during active atrial emptying and the area of A-loop was calculated as a work of the left atrium. The area of the A-loop, or left atrial work, in patients with myocardial infarction was significantly higher than that in normal subjects. The left ventricular ejection fraction showed a significant inverse correlation with the left atrial work (fig. 2). In patients with myocardial infarction, the more left ventricular function was impaired, the more left atrial work was increased. Furthermore, left atrial work correlated significantly with the ratio of active atrial emptying to left ventricular stroke volume (fig. 3).

In an experimental study of isolated rabbit left atria, Blinks demonstrated that the Frank-Starling mechanism was operative in the atrium as well as in the ventricle. Furthermore, Payne et al. reported that the atrial stroke shortening increased as atrial diameter was increased in an intact animal. Clinically, Braunwald et al. proposed that Frank-Starling’s law was operative in the human left atrium. Later, Murray et al. and Hawley et al. observed the relationship of left atrial cyclic volume change and left atrial maximal volume, and found that the larger left atrial volumes had the greater atrial volume change during the cardiac cycle. However, in these studies, they discussed left atrial function without separating passive atrial emptying and active atrial emptying. The present study shows that the left atrial work during active atrial emptying correlated significantly with the left atrial volume before active atrial emptying (fig. 4). This correlation suggests that the left atrial work depends on the left atrial volume before active atrial emptying, which corresponds to the Frank-Starling mechanism. Furthermore, the contractility of the left atrium seems to be unchanged both in normal subjects and in patients with myocardial infarction, since all patients were on the linear correlation of left atrial work and left atrial volume before active atrial emptying. Therefore, the increased left atrial work is caused by the increased left atrial volume before active atrial emptying, and not by the increased contractility of the left atrium.

In patients with myocardial infarction, left ventricular stroke volume is relatively maintained despite the impairment of left ventricular function. In these patients, the left atrium works more and transports the blood to the left ventricle during left ventricular diastole. This function of the left atrium can be attributed to the Frank-Starling mechanism. Left atrial function in patients with myocardial infarction cannot be ignored clinically or in assessing the left ventricular performance in these patients. In patients with atrial fibrillation or atrioventricular block associated with impaired left ventricular function due to myocardial infarction, the importance of proper atrial function should be recognized.

References

Importance of left atrial function in patients with myocardial infarction.
Y Matsuda, Y Toma, H Ogawa, M Matsuzaki, K Katayama, T Fujii, F Yoshino, K Moritani, T
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