Change of Left Atrial Systolic Pressure Waveform in Relation to Left Ventricular End-Diastolic Pressure

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The relation between the left atrial systolic pressure waveform and left ventricular end-diastolic pressure was observed in 17 patients who underwent diagnostic cardiac catheterization. Left atrial pressure and left ventricular pressure were simultaneously recorded from a multisensor catheter before and during angiotensin infusion. Left ventricular systolic pressure and left ventricular end-diastolic pressure were 133 ± 17 and 12.3 ± 3.2 mm Hg, respectively, before angiotensin infusion and increased to 168 ± 18 (p < 0.01) and 19.4 ± 4.5 mm Hg (p < 0.01), respectively, during infusion. The left atrial systolic pressure curve consisted of two positive waves—a first wave (A) and a second wave (A'). The A and A' wave pressures were 11.6 ± 2.3 and 10.2 ± 3.9 mm Hg, respectively, before angiotensin infusion and 16.5 ± 2.9 (p < 0.01) and 18.1 ± 4.7 mm Hg (p < 0.01), respectively, during infusion. The ratio of A'/A of left atrial systolic pressure was 0.81 ± 0.27 before angiotensin infusion and 1.08 ± 0.14 (p < 0.01) during infusion. The ratio of A' to A of left atrial systolic pressure was linearly related to left ventricular end-diastolic pressure before and during (p < 0.01) angiotensin infusion. The amplitude of the A wave exceeded that of the A' wave at normal left ventricular end-diastolic pressures. However, as the left ventricular end-diastolic pressure increased either at rest or during angiotensin infusion, the amplitude of the A' wave increased and often exceeded that of the A wave. These results suggest that the second (A') wave might be attributed to the increased reflection associated with increased left ventricular end-diastolic pressure. (Circulation 1990;82:1659–1667)

The left atrial pressure waveform consists of two major positive deflections—the a and v waves. The a wave is due to atrial systole and follows the p wave of the electrocardiogram. The rise of left atrial pressure during left ventricular systole is termed the v wave. The diastolic phase of the left ventricular pressure pulse consists of an early rapid filling wave, a slow filling period, and an atrial systolic wave caused by atrial contraction. The atrial systolic wave of the left ventricle is immediately followed by left ventricular end-diastolic pressure. Because the introduction of a catheter to the left atrium is technically complex, there have been few studies observing the relation between left atrial and left ventricular pressures.1,2 In these previous studies, the pressures were recorded with a fluid-filled catheter—external transducer system. The purpose of the present study was to examine the relation between left atrial systolic pressure waveforms and left ventricular end-diastolic pressure. A high-fidelity, micromanometer-tipped catheter was introduced into the left atrium and left ventricle, and high-gain left atrial and left ventricular pressure pulses were recorded at rapid paper speeds.

Methods

Subject Groups

Data were obtained during diagnostic cardiac catheterization in three patients with atypical chest pain and 14 with remote myocardial infarction. In all 17 patients, the changes of left atrial and left ventricular pressures were observed before and during angiotensin infusion. Those with remote myocardial infarction had no symptoms of angina. The clinical data for each patient are listed in Table 1. All patients were in normal sinus rhythm and had normal
Table 1. Patient Characteristics

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M, male; F, female; MI, myocardial infarction.

PR intervals on the electrocardiogram. None had any clinical or hemodynamic evidence of mitral valve involvement. Before cardiac catheterization, each patient gave written informed consent after full explanation of the procedure.

Catheterization

Premedication with 10 mg diazepam and prophylactic antibiotics were administered before the study of cardiac catheterization. Cardiac medications were discontinued at least 24 hours before the study. A high-fidelity, micromanometer-tipped catheter (8F, model SPC-780C, pigtail, Millar Instruments, Houston) was used for pressure measurements. Use of the catheter and its introduction to the left atrium has been described in detail elsewhere. Briefly, the catheter has two pressure sensors with one end-opening lumen. Both pressure sensors and a Statham P23ID transducer (Oxnard, Calif.) were balanced, calibrated with a mercury manometer, and adjusted for equal gain before insertion. During the procedure, the Statham gauge, which was positioned in the midstretch level, was open to the atmosphere and was used as the baseline for pressure. Zero shift during the procedure was adjusted by comparison with the pressure simultaneously obtained from a fluid-filled catheter connected to the transducer. The position of the micromanometer trace on the monitor was then matched to that of the transducer. Because both micromanometer pressures were adjusted to the same zero level, the transmitral pressure gradient could be measured directly and very accurately from micromanometer pressures at any time of diastole. Before recording, the left atrial catheter micromanometer was balanced, with its fluid-filled lumen against a transducer aligned at the midstretch level. The period of late diastasis was then used to align the micromanometric left ventricular pressure signal with its left atrial pressure signal, which may ignore a small pressure gradient of a few millimeters of mercury between the left atrium and the left ventricle during diastasis. The calibration was reconfirmed after withdrawal of the catheter. The catheter was introduced through the brachial artery by means of a guide wire. In the shallow right anterior oblique projection, the catheter was advanced through the aortic valve into the left ventricle. While the catheter was directed toward the mitral valve in a position near it, the catheter was advanced into the left atrium. The distal pressure sensor was placed in the left atrium and the proximal sensor in the left ventricle. The previous study confirmed that the retrograde catheter across the mitral valve did not produce mitral regurgitation. High-gain left atrial and left ventricular pressures were recorded on an Electronics for Medicine VR 12 recorder (Pleasantville, N.Y.) at a rapid paper speed of 100 mm/sec. After obtaining the left atrial and left ventricular pressures, the continuous intravenous infusion of angiotensin was administered. The infusion of angiotensin was titrated to maintain a steady-state infusion at a dose that produces an increase in left ventricular systolic pressure. The dose of angiotensin was 0.5–4.0 μg/min and produced a 17–53-mm Hg increase in left ventricular systolic pressure. The left atrial and left ventricular pressures were recorded in a steady state during angiotensin infusion. The five consecutive beats were analyzed before and during angiotensin infusion, and the results were averaged.

Experimental Study

Experiments were conducted on six mongrel dogs (body weight, 13±3 kg) that were anesthetized with morphine sulfate (5 mg/kg) and α-chloralose (30 mg/kg). A tracheal cannula was inserted, and ventilation was begun. The chest was opened laterally through the fifth left intercostal space. One micromanometer-tipped catheter (model PC-484A, Millar Instruments) was placed in the left atrium through the pulmonary vein, and another one was placed in the left ventricle retrogradely across the aortic valve through the left carotid artery. The circumflex coronary artery was occluded after placement of the catheters. The pericardium was closed. With this approach, the effects of catheter movement on the left atrial pressure waveforms that might occur with the transmural approach could be avoided. In the model of acute myocardial infarction, left atrial and left ventricular pressures were obtained before angiotensin infusion. The infusion of angiotensin was increased with a maximum dose of 3 μg/min to produce an increase of left ventricular systolic and end-diastolic pressures. When the increase of left ventricular end-diastolic pressure was not sufficient with angiotensin infusion, the dextran infusion was added. Left atrial and left ventricular pressures were recorded in a steady state during angiotensin infusion.
Statistics

All hemodynamic values are given as mean±SD. Statistical analysis of the results was completed by a paired t test and by linear regression analysis. In all analyses, a p value of less than 0.05 was considered significant.

Results

Clinical Studies

Left atrial and left ventricular pressure tracings are displayed in Figure 1. Left atrial systolic pressure had two positive waves—the first (A) and second (A') left atrial systolic waves. Pressure waves of the left ventricle consistent with A and A' of the left atrium were pre-A wave (pre-LVA) and A wave (LVA), respectively. The left atrial Z point pressure is the coincident time point to the left ventricular end-diastolic pressure.

Figure 2 shows left atrial pressure patterns before angiotensin infusion in a patient with atypical chest pain (2A) and those with myocardial infarction (2B and 2C). The patient with atypical chest pain had a left ventricular end-diastolic pressure of 9.2 mm Hg. Left atrial systolic pressure did not have an evident A' wave. One patient with anterior myocardial infarction (2B) had a left ventricular end-diastolic pressure of 15.6 mm Hg, and left atrial systolic pressure showed an equivalent amplitude of A and A' waves. Another patient with anterior myocardial infarction (2C) had a left ventricular end-diastolic pressure of 17.6 mm Hg. The second wave of left atrial systolic pressure was higher than the first.

Figure 3 represents left atrial and left ventricular pressure changes before and during angiotensin infusion. In the control period before angiotensin infusion (3A), the A wave of left atrial pressure was higher than its corresponding A' wave. With the infusion of angiotensin (2 μg/min) (3B), left ventricular systolic pressure increased to 152 mm Hg from the control level of 127 mm Hg. Left ventricular end-diastolic pressure increased from 9.6 mm Hg to 14.0 mm Hg; this change was associated with the increase of LVA. The A' wave of left atrial systolic pressure became prominent, and the pressure curve had a biphasic composition of A and A' waves. With the continued infusion of angiotensin (3 μg/min) (3C), left ventricular systolic pressure increased to 167 mm Hg and left ventricular end-diastolic pressure increased to 19.2 mm Hg. The A' wave of the left atrial systolic pressure curve became more prominent and higher than the A wave.

Hemodynamic parameters for all 17 patients are listed in Table 2. Left ventricular systolic pressure significantly increased from 133±17 mm Hg before to 168±18 mm Hg during angiotensin infusion. The cardiac cycle lengths were not significantly changed during angiotensin infusion. The left atrial A wave pressure before angiotensin infusion was 11.6±2.3 mm Hg and increased to 16.5±2.9 mm Hg during infusion (p<0.01). The left atrial A' wave pressure increased from 10.2±3.9 mm Hg before to 18.1±4.7 mm Hg during infusion (p<0.01). The A'/A before angiotensin infusion was 0.81±0.27 and 1.08±0.14 (p<0.01) during infusion. The left atrial Z point pressure significantly increased from 14.0±0.8 to 17.6±1.0 mm Hg during infusion (p<0.01).
pressure was 9.4±3.9 mm Hg before and increased to 16.5±4.8 mm Hg during angiotensin infusion. The pre-LVA pressure was 8.5±2.4 mm Hg before and increased to 13.4±2.9 mm Hg during angiotensin infusion. The LVA pressure increased from 11.9±3.7 mm Hg before to 20.0±4.4 mm Hg during angiotensin infusion. The left ventricular end-diastolic pressure was 12.3±3.2 mm Hg before angiotensin infusion and 19.4±4.5 mm Hg during infusion. The left atrial A wave pressure was consistently higher than the pre-LVA pressure of the left ventricle (Figure 1). Then the left atrial and left ventricular pressure curves crossed, and the LVA pressure of the left ventricle was consistently higher than the left atrial A’ wave pressure. The LVA pressure was higher than the A wave pressure in nine of 17 patients before and 16 of 17 patients during angiotensin infusion. The left atrial Z point pressure was in a decay of A’ wave and was lower than the A’ wave pressure. The left ventricular end-diastolic pressure had a significant linear correlation with the left atrial Z point pressure (Y=1.04x-3.53, r=0.98, p<0.01) and was consistently higher (2.9±1.2 mm Hg) than the Z point pressure.

Figure 4 shows the relation between the left ventricular end-diastolic pressure and the A’/A ratio of the left atrial pressure wave. The ratio of A’/A before angiotensin infusion (open circle) had a significant linear correlation with the left ventricular end-diastolic pressure (r=0.918, p<0.01). Because the
results of an analysis of covariance (ANCOVA) demonstrated no significant difference in the linear regression before and during angiotensin infusion, these data were combined. The left ventricular end-diastolic pressure correlated well with the ratio of A'/A of left atrial pressure \( (r=0.902, p<0.01) \).

**Experimental Studies**

In the experimental animals with acute occlusion of the coronary artery, left atrial pressure waveforms were observed without the transmittal approach. The left atrial A wave pressure before angiotensin infusion was 10.9±3.0 mm Hg and increased to 26.3±4.4 mm Hg during infusion \( (p<0.01) \). The left atrial A' wave pressure increased from 9.0±2.7 mm Hg before to 25.8±4.1 mm Hg \( (p<0.01) \) during angiotensin infusion. The A'/A ratio was 0.83±0.07 before angiotensin infusion and 0.99±0.08 during infusion \( (p<0.01) \). Left ventricular end-diastolic pressure was 10.9±3.2 mm Hg before and 27.6±4.0 mm Hg during angiotensin infusion \( (p<0.01) \). Left ventricular end-diastolic pressure correlated well with the ratio of A'/A of left atrial pressure \( (Y=0.009X+0.74, r=0.71, p<0.05) \). Figure 5 is a representative tracing in one dog. The change of the left atrial systolic pressure waveform is shown before (5A) and after (5B) increasing left ventricular end-
ventricle and priming it for ventricular ejection.²⁻⁵,⁷⁻¹⁶

The a wave of the left atrial pressure waveform or left atrial systolic pressure pulse is believed to result from left atrial contraction. The present study demonstrates a previously unrecognized biphasic left atrial systolic pressure waveform and describes the relation of the change in this pressure pattern to left ventricular end-diastolic pressure. The pattern of the left atrial systolic pressure waveform varied in relation to left ventricular end-diastolic pressure in the control state (Figure 2). Patients with lower left ventricular end-diastolic pressure had a higher first (A) wave of left atrial systolic pressure, and patients with higher left ventricular end-diastolic pressure had a higher second (A') wave of left atrial systolic pressure. These conclusions were reached by changing (increasing) each patient's left ventricular end-diastolic pressure with angiotensin infusion (Figure 3). As left ventricular end-diastolic pressure increased with angiotensin infusion, the amplitude of the second wave increased. The ratio of amplitude between the second and first left atrial systolic waves was linearly related to left ventricular end-diastolic pressure, either at rest or during angiotensin infusion (Figure 4).

In previous studies, left atrial pressure was recorded with a fluid-filled catheter-external transducer system, while in the present study it was obtained by a micromanometer-tipped catheter. It has been reported that the elevation in atrial pressure produced by atrial systole varies directly with the time interval between the onset and peak of the left atrial systolic pressure wave and, inversely, with the time interval between the peak of the left atrial

**Discussion**

**Present Data**

Left atrial contraction provides a significant increment of blood to the left ventricle, stretching the

![Graph showing the relation of A'/A to left ventricular end-diastolic pressure.]

**Figure 4. Scatterplot of relation of the ratio of A'/A of the left atrial systolic pressure and left ventricular end-diastolic pressure before (○) and during (●) angiotensin infusion. Patients with higher left ventricular end-diastolic pressures had higher ratio of A' to A of left atrial systolic pressure.**

**Figure 5. Representative case of experimental dog. Change of left atrial systolic pressure waveform before (A) and after (B) increasing left ventricular end-diastolic pressure. A' wave became more prominent and higher than A wave as left ventricular end-diastolic pressure increased. A, first left atrial systolic pressure; A', second left atrial systolic pressure; LVP, left ventricular pressure; LAP, left atrial pressure; LVA, A wave of left ventricular pressure; LVEDP, left ventricular end-diastolic pressure; LVP, left ventricular pressure.**
systolic pressure wave and the onset of ventricular contraction. This phenomenon might be explained from the data in the present study. The amplitude of left atrial systolic pressure was dependent on a component of the first or second wave. With lower left atrial systolic pressure, its peak was that of the first wave; it had a shorter elapsed time from the onset of the left atrial systolic pressure wave and longer elapsed time until the onset of ventricular contraction. With higher left atrial systolic pressure, its peak was that of the second wave; it had a longer elapsed time from the onset of the left atrial systolic pressure wave and shorter elapsed time until the onset of ventricular contraction.

The significant correlation between the left atrial Z point pressure and left ventricular end-diastolic pressure has been previously documented. The left atrial Z point pressure occurs during decay of the A' wave. The A' wave is a combined wave of the later component of the A wave and the wave of reflection from the ventricle back to the atrium. The Z point pressure depends on the prominence of the A or A' wave and the amplitude of these waves. In the present study, left ventricular end-diastolic pressure had a significant linear correlation with and was consistently higher than the left atrial Z point pressure.

Left atrial contraction causes the increase of the A wave and transfers the blood from the left atrium to the left ventricle. The A wave of left atrial pressure was higher than the pre-LVA of left ventricular pressure. Then the left atrial and left ventricular pressure curves crossed, and the LVA wave of left ventricular pressure was higher than the A' wave of left atrial pressure. The LVA wave of left ventricular pressure results from left ventricular filling contributed to by left atrial contraction and is influenced by left ventricular chamber stiffness and myocardial viscosity. The A' wave of left atrial pressure is a reflection of left ventricular pressure and was lower than its LVA. The LVA wave was higher than the A wave in more patients observed during angiotensin infusion than those observed before infusion, which might be explained by augmented left atrial contraction and by increased chamber stiffness in relation to left ventricular filling.

**Study Limitations**

The potential effects of catheter movement on the pressure waveforms must be considered. A catheter moved through the mitral orifice during the period between the P wave in the electrocardiogram and the upstroke of left ventricular pressure is subjected to movement due to valve motion and other stresses. However, if the biphasic left atrial systolic pressure wave is produced by movement caused by valve motion and other stresses, it should also be observed in patients with lower left ventricular end-diastolic pressure. The left atrial pressure pattern was observed in the open-chest dog, avoiding the effects of catheter movement on the pressure waveforms. When left ventricular end-diastolic pressure was low, left atrial systolic pressure did not have an evident A' wave. When left ventricular end-diastolic pressure was increased by angiotensin infusion, the left atrial systolic pressure curve showed a biphasic pattern, which revealed an evident second atrial systolic wave. These experimental results are consistent with the present clinical data and indicate that the biphasic left atrial systolic pressure pattern was not produced by catheter movement through the mitral valve.

**Mechanism of Biphasic Wave**

There have been recent investigations on the contribution of wave reflections to aortic pressure waveforms. A well-defined systolic inflection point was found to divide the aortic pressure waveform into early and late systolic phases. Pressure waveforms were divided into three groups: Group A, patients whose late systolic pressure exceeded early systolic pressure; Group B, those whose early and late systolic pressures were nearly equal; and Group C, those whose early systolic pressure exceeded late systolic pressure. Patients from Groups A and B all demonstrated oscillations of the impedance moduli about the characteristic impedance. Group C patients demonstrated flatter impedance spectra. Thus, a larger secondary rise in pressure was associated with a more oscillatory impedance spectrum. These results suggest that the differences in pressure waveforms were due to differences in reflections in the arterial tree. From these studies, the present data might suggest that the rise in the second atrial systolic wave was influenced by wave reflection from the left ventricle. However, the amplitude of the second atrial systolic wave is a combined wave of the component of the later portion of the first atrial systolic wave resulting from the combined influence of atrial contraction and a reflection wave from the ventricle back to the left atrium and not solely from a reflection wave.

Several factors could explain the changes in left atrial systolic pressure waveforms in relation to left ventricular end-diastolic pressure. First, the acute increase in left ventricular end-diastolic pressure with angiotensin infusion must be accompanied by an increase in left ventricular end-diastolic volume. If the change of reflection does account for the change of the left atrial systolic pressure waveform, does the change of reflection from the left ventricle result from the change of pressure, volume, or other causes in the left ventricle? Left ventricular end-diastolic pressure and left ventricular end-diastolic volume relation is exponential \( P = b e^{kV} \), \( P \) is left ventricular end-diastolic pressure, \( V \) is left ventricular end-diastolic volume; and volume stiffness \( dP/dV \) is \( kbe^{kV} = kP^{2} \). Left ventricular end-diastolic pressure and the \( dP/dV \) relation is linear. In the present study, the ratio of A'/:A and the left ventricular end-diastolic pressure relation was linear. Thus, the change of reflection is probably acutely related to the change of \( dP/dV \). However, it is difficult to observe the atrial systolic waves during volume loading with-
out increased left ventricular end-diastolic pressure. Second, the patients in the present study had either atypical chest pain or remote myocardial infarction. We did not consider those with increased left ventricular end-diastolic pressure from other causes, such as left ventricular hypertrophy. Third, it has been demonstrated that regional pressure differences exist in the left ventricle not only during the early rapid filling phase but also the late filling phase associated with atrial contraction. During late diastolic filling, left ventricular end-diastolic pressure decreases from apex to base. Although the most appropriate position at which to record intraventricular pressure remains unresolved, the present study did not focus on left ventricular diastolic mechanics. Furthermore, in the present study the micromanometer in the left ventricle was always placed near the mitral valve, and the position remained unchanged throughout the procedure. Fourth, in other published high-fidelity tracings from the experimental animals, no bifid left atrial systolic pressure wave was evident. However, in those studies the left ventricular end-diastolic pressures were less than 10 mm Hg. The pressure recording in one previous experimental study shows biphasic left atrial systolic pressure with increased left ventricular end-diastolic pressure in the dog with surgically induced heart block. A biphasic left atrial systolic pressure wave and an increment of the second component during ischemia were clinically recorded by micromanometer during balloon angioplasty. In our clinical and experimental studies, patients or dogs with lower left ventricular end-diastolic pressure did not have a bifid left atrial systolic pressure wave. Fifth, the relation between left ventricular end-diastolic pressure and the ratio of A′/A was linear within this range of left ventricular end-diastolic pressure. However, if left ventricular end-diastolic pressure exceeded this range, the ratio of A′/A might not partake of this linear relation.

Conclusion

The left atrial systolic pressure curve was found to comprise two positive waves, the first (A) and second (A′) waves. The first wave resulted from left atrial contraction, and the second wave was the reflection from the ventricle back to the left atrium. With the increase of left ventricular end-diastolic pressure, the amplitude of the second atrial systolic wave became higher than that of the first wave. The results of this study suggest that the a wave of left atrial systolic pressure is not a simple wave produced by left atrial systole, and it includes a component of reflection associated with increased left ventricular end-diastolic pressure.

References


KEY WORDS • left atrial pressure • left ventricular pressure • transmitral pressure
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