Early mitral deceleration and left atrial stiffness

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Marino, Paolo, Giuseppe Faggian, Paolo Bertolini, Alessandro Mazzucco, and William C. Little. Early mitral deceleration and left atrial stiffness. Am J Physiol Heart Circ Physiol 287: H1172–H1178, 2004.—Left ventricular (LV) filling deceleration time (DT) is determined by the sum of atrial and ventricular stiffnesses (KI,A + KI,V). If KI,A, however, is close to zero, then DT would reflect KI,V only. The purpose of this study was to quantify KI,A during DT. In 15 patients, KI,A was assessed, immediately after cardioplegic bypass, from E wave DT as derived from mitral tracings obtained by transesophageal echocardiography and computed according to a validated formula. In each patient, a left atrial (LA) volume curve was also obtained combining mitral and pulmonary vein (PV) cumulative flow plus LA volume measured at end diastole. Time-adjusted LA pressure was measured simultaneously with Doppler data in all patients. KI,A was then calculated during the ascending limb of the V loop and during DT. LA volume decreased by 7.3 ± 6.5 ml/m² during the first 6% of mitral DT, whereas LV volume increased 9.4 ± 8.4 ml/m² (both P < 0.001). There was a small amount of blood coming from the PV during the same time interval, with the cumulative flow averaging 3.2 ± 2.4 ml/m² (P < 0.001). Mean LV pressure was 10.0 ± 5.1 mmHg, and it did not change during DT [from 7.8 ± 4.3 to 8.0 ± 4.3 mmHg, not significant (NS)], making KI,V, which averaged 0.46 ± 0.39 mmHg/ml during the V loop, close to zero during DT [KI,A+DT, from −0.002 ± 0.08 to −0.001 ± 0.031 mmHg/ml NS]. KI,V, as assessed noninvasively from DT, averaged 0.25 ± 0.32 mmHg/ml. In conclusion, notwithstanding the significant decrement of LA volume, KI,A does not change and can be considered not different from zero during DT. Thus KI,A does not affect the estimation of KI,V from Doppler parameters.

METHODS

Patient population. The population consisted of 15 consecutive patients (11 men and 4 women, with a mean age of 60.3 ± 9.5 yr [range: 38–78 yr]) who underwent open heart surgery for significant coronary artery disease (n = 7, associated with aortic valve replacement in 3 patients and with mitral valve repair in 1 patient) or isolated aortic valve disease (n = 4; Table 1). One patient suffered a previous myocardial infarction in the anteroseptal region and underwent pllication of the aneurysm besides coronary surgery. Another patient suffered a previous transapical infarction, but no regional wall motion abnormalities were evident at inspection and preoperative two-dimensional (2-D) echocardiography. In all cases, there was no or only mild mitral insufficiency as judged by postoperative color Doppler echocardiography. Similarly, major aortic insufficiency was also excluded as well as the presence of any shunt at atrial or ventricular level. The patient population had been retrospectively selected from a larger series (27 patients) according to the presence of a clear separation between the E and A waves of the mitral flow velocity profile as assessed in the immediate postcardiopulmonary bypass period. Informed consent had been obtained from each patient.

Operative techniques. The patients had been instrumented, after the induction of anesthesia, with a transesophageal 5-MHz 2-D echocardiographic probe (Toshiba PEF-511SA) connected to an electronic ultrasonograph (Toshiba Sonolayer SSH-140A, Toshiba Medical; Tokyo, Japan). Images and Doppler traces were recorded at a speed of 100 mm/s (Fig. 1) on videotape (Sony V0–5800) together with a surface electrocardiogram and the aortic pressure as derived from a radial artery catheter in each patient. Coronary artery bypass, aortic valve replacement, and mitral valve repair were carried out on cardiopulmonary bypass and moderate systemic hypothermia. Myocardial protection was performed by continuous profound topical hypothermia and potassium crystalloid cardioplegia. The distal anastomoses were sutured during aortic cross-clamping; the proximal ones during cardiopulmonary bypass as the beating nonworking heart was rewarmed. The pericardium was left open in all patients.

Data acquisition. After discontinuation of cardiopulmonary bypass, before chest closure and with the patient in critical but stable hemodynamic conditions, Doppler traces of the mitral flow and of the left lower pulmonary vein (PV) velocities were recorded together with simultaneous LA pressure as derived from an intra-atrial catheter (1.7 mm in diameter and 30.5 cm long, Intracath, Deseret; Sandy, UT) sequentially connected to a strain gauge transducer (Transpac II, Abbott; Sligo, Ireland). A line 1.0 mm in diameter and 150 cm long (Plastimed; Saint-Leu-La-Foret Cedex, France) with a three-way stopcock was used to display the LA pressure on the screen of a physiological recorder (Sirecuit, Siemens Medical Systems; Iselin NJ). The catheter, routinely used in our institution for the monitoring of LA pressure during the immediate postoperative period, was placed by the surgeon, filled with saline, and visually leveled to the right of measuring the LA volume curve throughout the cardiac cycle (17).

A SIMPLE NONINVASIVE METHOD OF evaluating left ventricular (LV) diastolic stiffness would be of great clinical utility. The deceleration time (DT) of early diastolic filling measured by Doppler mitral inflow velocity provides a potential method to do this. Theoretic considerations indicate that the DT is determined by the sum of left atrial (LA) and LV chamber stiffness (KI,A + KI,V) (8, 13). Little et al. (14) proposed that KI,A could be ignored because, during the time of DT, the atrium behaves as a conduit, and there is little change in LA volume or pressure (14). This simplifying assumption allows the calculation of KI,V from the DT. The accuracy of determining KI,V from DT has been supported by limited observations in experimental animals and humans (8, 14, 16). However, LA pressure and volumes during DT have not been measured in humans.

This study was undertaken to determine LA pressure and volume during DT in patients with a wide range of KI,V. This was accomplished using a previously validated method.
atrium. The characteristics of this monitoring system had been previously defined in an in vitro condition relative to a micromanometer-tipped catheter, creating pressure waves from 10 to 30 mmHg with a frequency response ranging from 1.3 to 5 Hz. The resonant frequency of the system was shown to be 5 Hz, with a flat response until 2 Hz and then an amplified response from 2 to 5 Hz. The time delay of the system, relative to the micromanometer catheter, ranged from 0 to 60 ms, with a mean delay of 50 ms, which was subsequently used as a time correction factor for the LA pressure tracing in our patients. In three patients, LA pressure was measured using a micromanometer-tipped catheter (Mikrotip model PC-350, Millar Instruments; Houston, TX), which was introduced into the atrial cavity through the upper right PV. In one of these patients, the LV pressure was also sequentially assessed, advancing the micromanometer from the atrial to ventricular cavity. The electrocardiographic trace and the radial pulse pressure were also recorded in real time. All the traces (electrocardiographic trace, radial and LA pressures) were also simultaneously displayed on the screen of the echocardiographic equipment. The LA pressure trace of those three patients who received the micromanometer catheter was displayed directly only on the screen of the echocardiographic equipment.

A standard four-chamber view of the heart, minimizing foreshortening, was recorded first, with minor adjustments made to maximize the mediolateral diameter of the mitral anulus. Pulsed-wave Doppler

Table 1. Patient data

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Surgery Type</th>
<th>Age, yr</th>
<th>Heart Rate, beats/min</th>
<th>E/A</th>
<th>DT, ms</th>
<th>Mean AoP, mmHg</th>
<th>Mean LAP, mmHg</th>
<th>LV Volume, ml/sqm</th>
<th>LA Volume, ml/m²</th>
<th>$K_{LV}$, mmHg/ml</th>
<th>$K_{LA}$, mmHg/ml</th>
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<tr>
<td>1</td>
<td>Mechanical Ao prosthesis</td>
<td>38</td>
<td>80.2</td>
<td>1.03</td>
<td>135</td>
<td>76.8</td>
<td>12.6</td>
<td>143</td>
<td>57</td>
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<td>0.39</td>
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<tr>
<td>2</td>
<td>BYP</td>
<td>51</td>
<td>100.4</td>
<td>0.96</td>
<td>191</td>
<td>65.5</td>
<td>13.6</td>
<td>61</td>
<td>17</td>
<td>0.17</td>
<td>1.18</td>
</tr>
<tr>
<td>3</td>
<td>BYP</td>
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<td>81.6</td>
<td>0.66</td>
<td>226</td>
<td>89.1</td>
<td>4.4</td>
<td>44</td>
<td>36</td>
<td>0.12</td>
<td>0.12</td>
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<tr>
<td>4</td>
<td>BYP</td>
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<td>0.79</td>
<td>195</td>
<td>78.1</td>
<td>12.1</td>
<td>47</td>
<td>9</td>
<td>0.16</td>
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<td>39.4</td>
<td>0.70</td>
<td>542</td>
<td>70.6</td>
<td>14.1</td>
<td>105</td>
<td>59</td>
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<td>6</td>
<td>Mitral repair + BYP</td>
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<td>67.9</td>
<td>0.81</td>
<td>444</td>
<td>65.8</td>
<td>13.3</td>
<td>61</td>
<td>24</td>
<td>0.03</td>
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<tr>
<td>7</td>
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<td>91.9</td>
<td>0.76</td>
<td>84</td>
<td>91.9</td>
<td>1.7</td>
<td>59</td>
<td>22</td>
<td>1.20</td>
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<td>Mechanical Ao prosthesis + BYP</td>
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<td>108.5</td>
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<td>BYP</td>
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<td>101.3</td>
<td>0.59</td>
<td>224</td>
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<td>5.1</td>
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<td>Biological Ao prosthesis</td>
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<td>84.5</td>
<td>0.47</td>
<td>266</td>
<td>78.9</td>
<td>20.6</td>
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<td>1.41</td>
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<td>43.1</td>
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<tr>
<td>12</td>
<td>BYP</td>
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<td>0.62</td>
<td>150</td>
<td>73.4</td>
<td>11.1</td>
<td>36</td>
<td>30</td>
<td>0.29</td>
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<td>Biological Ao prosthesis + BYP</td>
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<td>0.17</td>
<td>705</td>
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<td>12.8</td>
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<tr>
<td>14</td>
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<td>0.50</td>
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<td>4.3</td>
<td>34</td>
<td>67</td>
<td>0.05</td>
<td>0.33</td>
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</tr>
<tr>
<td>15</td>
<td>Biological Ao prosthesis</td>
<td>78</td>
<td>81.5</td>
<td>0.83</td>
<td>97</td>
<td>81.7</td>
<td>3.4</td>
<td>117</td>
<td>54</td>
<td>0.83</td>
<td>0.09</td>
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<tr>
<td>Mean</td>
<td>60.27</td>
<td>86.78</td>
<td>0.71</td>
<td>271.80</td>
<td>72.67</td>
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<tr>
<td>SD</td>
<td>9.45</td>
<td>17.15</td>
<td>0.28</td>
<td>168.93</td>
<td>11.84</td>
<td>5.09</td>
<td>44.15</td>
<td>23.33</td>
<td>0.32</td>
<td>0.39</td>
<td></td>
</tr>
</tbody>
</table>

Ao, aortic; BYP, coronary artery bypass surgery; DT, deceleration time; AoP, Ao pressure; LAP, left atrial (LA) pressure; LV, left ventricular (LV) pressure; $K_{LA}$, atrial stiffness; $K_{LV}$, ventricular stiffness; E/A, ratio of E wave to A wave.

Fig. 1. A: 2-dimensional 4-chamber view with left atrial (LA) and radial pressure of patient 5 of Table 1. B: same case with color Doppler flow showing a mild degree of mitral regurgitation. C and D: mitral flow velocity profile (C) and pulmonary vein (PV) flow velocity (D) of the same case together with LA and radial pressure (originally shown in green on the screen of the equipment).
measurements of the mitral valve flow velocity were then obtained using a pulse repetition rate of 4.5 kHz, with the Doppler beam aligned perpendicular to and bisecting the plane of the mitral anulus and with the 3-mm-wide sample volume placed at the level of the anulus, together with synchronized LA and radial pulse pressures (17). Subsequently, the probe was slightly withdrawn and rotated to visualize the lower left PV. PV flow velocity was then assessed by positioning the Doppler sample ~1–2 cm proximal to the entrance of the inferior vein into the LA, with the Doppler beam as parallel as possible to the vein long axis.

A superior-inferior systolic dimension of the LA cavity was also obtained, by M-mode echocardiography, by directing the interrogating beam toward the anterior leaflet of the mitral valve. The base-to-apex dimension of the LA cavity could thus be defined, on the M-mode tracing, as the distance between a point 0.5 cm below the first echo generated from the surface of the transducer and the anterior mitral leaflet, immediately after the valve closure, in systole.

Care was taken to obtain Doppler recordings of the mitral valve and of the left lower PV flow velocities within <2 min from one another. Recordings were made, without the respirator being stopped, with inotropic support (dopamine) or vasodilators (nitrates) as necessary.

Data analysis. For each patient, the diameter of the mitral anulus was measured offline from the video frame containing the maximal separation of the leaflets (first or second frame after mitral valve opening) on three consecutive sinus or right atrially paced beats and averaged using a commercially available ad hoc software package (Echoport, Sigma; Pavia, Italy) and a personal computer (Vectra ES/12, Hewlett Packard) provided with a digitizing board (Targa 16, AT&T). Mitral valve area was then calculated assuming a circular shape according to the following formula: \( \pi \times D^2/4 \), where \( D \) is the mediolateral diameter of the mitral anulus in early diastole.

Synchronized LA and radial pressures and Doppler recordings were then digitized from selected video frames using the equipment described above (Fig. 2). Ectopics and immediately postectopic beats were excluded. Because the mitral and PV flow velocities could not be recorded simultaneously, they were analyzed by matching records at approximately the same R-R interval, which never exceeded 5% of their mean (0.7 ± 0.5%, range from 0.0% to 1.5%). Mitral and PV tracings were digitized along the outer darkest line on the Doppler spectrum. For each point of the Doppler profile, mitral inflow velocity was multiplied by maximal annular area (as measured in early diastole) to yield the filling rate, which was then integrated to obtain cumulative filling volume and calibrated to the 2-D echocardiographic (vide below) stroke volume (coefficient of variation for the correction: 0.67) (16). The 2-D echocardiographic ventricular volume was computed assuming the ventricle to be an ellipsoid of revolution, its volume being equal to \( 0.85 \times (A)^2/L \), where \( A \) is the LV area in the four-chamber view and \( L \) is the LV apex-to-base wall distance (23).

The end-systolic volume was then used to quantify the volume of the cavity at the beginning of the filling curve (Fig. 2). Similarly, the PV flow was obtained as PV velocity \( \times \) PV area, where PV area = (mitral valve velocity integral \( \times \) mitral valve area)/PV velocity integral. The LA volume curve was then derived as instantaneous PV flow minus mitral valve flow (17). The LA volume at the beginning of the cardiac cycle (computed modeling empirically the atrial cavity as a sphere according to the following formula: \( V = 0.52 \times D^2 \), where \( V \) is the LA volume and \( D \) is the base-to-apex dimension of the atrium immediately after closure of the mitral leaflets in the M-mode tracing) was then used to quantify the LA volume at the beginning of the filling curve. Previous studies have shown that the LA size calculated from a single echocardiographic dimension correlates highly with LA area and volume as measured from cineangiography (21) and that any LA dimension should be sufficient to evaluate the chamber size (11).

We checked this assumption in five patients by relating the base-to-apex dimension to the atrial cavity area. The \( r \) value for pooled systolic and diastolic values was 0.96 (\( r = 0.2x + 1.8, P < 0.001 \)). The LA pressure-volume loop was then generated for each patient, and the average atrial stiffness (\( K_{LA} \)) was computed as the change in pressure divided by the change in volume during atrial filling. From the Doppler mitral profile, peak velocities of E and A waves and the E-to-A ratio (E/A) were also calculated (Fig. 2), together with the DT of the E wave taken as the time interval between the peak E wave and the zero intercept of the regression line of the deceleration profile [mean \( r \) value 0.97 (range: 0.94–0.99) over 18 (range 10–26) points]. \( K_{LA} \) during the DT [\( K_{LA(DT)} \)] was also assessed, as the change in pressure divided by the change in volume during progressively increasing fractions of DT. Average ventricular stiffness (\( K_{LV} \)) was finally computed from the E-wave DT according to the following formula originally proposed by Little et al. (14): \( K_{LV} = [70 \text{ ms} / (\text{DT} - 20 \text{ ms})] \).

Statistics. All results are expressed as means ± SD. Differences between means were assessed by paired or unpaired t-tests, as appropriate. One-way ANOVA with repeated measures was used to analyze pressure and volume changes during DT. Nonparametric tests were used if data were not normally distributed. Regression analysis (least-squares method) was used as necessary. Statistical analyses were performed using Sigmastat (version 2.0 for Windows, Jandel; San Rafael, CA) statistical software.
RESULTS

There were no complications related to the use of the echocardiographic equipment or the LA monitoring system in the operating room. In two patients, sequential atrioventricular pacing was adopted by applying epicardial electrodes because of complete atrioventricular block. For the entire patient population, the correlation coefficient for the LA stroke volume as obtained from the single-dimension method and the Doppler method was $r = 0.67$ ($y = 1.52x + 0.37$, $P < 0.001$, standard error of estimate $= 7.9 \text{ ml/m}^2$; Fig. 3A). A plot of the average between the two methods was also compared with their difference according to Bland and Altman (Fig. 3B) (3). The Doppler method systematically overestimated the single-dimension volume-derived method ($\pm 5.6 \pm 15.1 \text{ ml/m}^2$, $P < 0.02$).

Hemodynamic and echocardiographic parameters for the entire patient population are shown in Table 1. Average $K_{LA}$ during the V loop averaged $0.46 \pm 0.39 \text{ mmHg/ml}$, slightly more than the average $K_{LV}$ as assessed from DT ($0.25 \pm 0.32 \text{ mmHg/ml}$, $P = 0.055$). $K_{LA(DT)}$, however, dropped to $-0.002 \pm 0.08 \text{ mmHg/ml}$ during first $\frac{1}{6} \text{ DT}$, a value very close to zero, and it did not change thereafter [to $-0.001 \pm 0.031 \text{ mmHg/ml}$ at $\frac{5}{6} \text{ DT}$, not significant (NS)]. This happened while LA volume decreased $7.3 \pm 6.5 \text{ ml/m}^2$, notwithstanding continuous flow from the PVs averaging cumulatively $3.2 \pm 2.4 \text{ ml/m}^2$ (Fig. 4). Through the combination of conduit and capacitance function, the LA cavity exhibited stable pressure during $\frac{4}{6} \text{ DT}$ (from $7.8 \pm 4.3$ to $8.0 \pm 4.3 \text{ mmHg}$, NS), thus acting as a constant pressure source for LV filling. These findings confirm previous experimental data suggesting stable LA pressure during the first half of filling deceleration at all stages of congestive heart failure in dogs and even during low-dose phenylephrine infusion in normal animals (14, 19) and challenge more recent human data suggesting a significant pressure drop during that same period (8).

Finally, average $K_{LA}$ modulated directly mean LA pressure ($r = 0.69$, $P < 0.01$; Fig. 5A) and inversely cardiac index [computed as (ventricular stroke volume $\times$ heart rate)/body surface area; $r = 0.70$, $P < 0.01$; Fig. 5B], in accordance with previous theoretical analysis (20).

DISCUSSION

It has been previously shown in patients with a variety of LV sizes and ejection fractions and under varying loading conditions, but with a narrow and normal range of mitral annular areas, that DT of early diastolic mitral valve flow velocity is directly related to the net stiffness of the atrium and ventricle and that it is not significantly influenced by mitral valve opening pressure, mitral valve area, and ventricular systolic volume (8, 14, 16). The effect of ventricular relaxation, instead, is variable, depending on whether relaxation is complete.
change of volume \( > 0 \) portion of the diastolic interval, excluding the suction-initiated (negative stiffness) portion of early filling, and that, even with the above limitations, it can predict quantitatively \( K_{LV} \) in patients with cardiac disease (8, 16).

**Atrial stiffness and cardiac performance.** The physiological significance of atrial stiffness is not completely appreciated, but some indirect evidence does suggest that it is important in hemodynamics. It is well known, in fact, that the addition of a flexible atrium to the inlet of an artificial heart substantially improves the heart’s output (20). Conversely, an increase in atrial stiffness should reduce stroke volume and cardiac output. In our study, average \( K_{LA} \) was inversely related to cardiac index \( (r = -0.69, P < 0.01) \) and stroke volume \( (r = -0.55, P < 0.05) \) and directly related to mean atrial pressure \( (r = 0.70, P < 0.01; \text{Fig. 5}). \) Thus the stiffer the atrium, the greater are the amplitude and mean level of atrial pressure (20), whereas smaller should be the gradient and peak E wave (10), unless conduit flow from the pulmonary reservoir restrains pressure from rapidly falling. In contrast, when the atrial compliance is large, the atrial pressure variation is small, and the mean level of atrial pressure during ventricular filling, and filling itself, is maintained at a relatively high level (20). In our study, the amount of conduit flow during E-wave acceleration increased progressively according to higher values of average \( K_{LA} \), making the peak E wave higher in these stiffer atria compared with those more compliant (Table 2).

As shown in Fig. 4, during the deceleration portion of the Doppler E wave, the atrial volume continues to decline (from 40 \( \pm \) 22 to 30 \( \pm \) 18 ml/\( m^2 \), \( P < 0.001) \), whereas atrial pressure is minimally changed (from 7.8 \( \pm \) 4.3 to 8.0 \( \pm \) 4.4 mmHg, NS). The essentially constant atrial pressure during DT, together with the continuous reduction of atrial volume, indicates that \( K_{LA} \) is negative during DT. This phenomenon be related to the continuous inflow from the PVs or to the effects exerted by atrial contraction, particularly if DT considerably prolonged (7). In any case, the relative stability of atrial pressure during DT might also reflect the measurements were made while the chest was open. Thus the constant volume property of the four-chamber heart is no longer applicable (4, 18). In particular, it has been shown that pericardiectomy increases LA compliance (9) and that this, in turn, affecting LA pressure variation, might mitigate the “y” descent and the ensuing ascent of LA pressure toward the a wave, as we observed (Table 2).

![Graph](http://ajpheart.physiology.org/)

**Table 2.** Peak mitral E-wave velocity, PV flow during E-wave acceleration, and change in LAP during “y” descent and “a” ascent divided according to the median of atrial stiffness

<table>
<thead>
<tr>
<th>( K_{LA} ) (mmHg/ml)</th>
<th>( n )</th>
<th>Peak E wave, cm/s</th>
<th>PV flow during E wave acceleration, ml/min/m²</th>
<th>Change in LAP during “y” descent, mmHg</th>
<th>Change in LAP during “a” ascent, mmHg</th>
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<tbody>
<tr>
<td>( \leq 0.33 )</td>
<td></td>
<td>27.0 ( \pm ) 9.4</td>
<td>1.8 ( \pm ) 1.0</td>
<td>(-1.2 \pm 0.6)</td>
<td>3.8 ( \pm ) 2.5</td>
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<tr>
<td>( &gt; 0.33 )</td>
<td></td>
<td>50.6 ( \pm ) 29.5</td>
<td>5.1 ( \pm ) 6.1</td>
<td>(-9.3 \pm 5.6)</td>
<td>5.0 ( \pm ) 2.1</td>
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<table>
<thead>
<tr>
<th>( n )</th>
<th>( K_{LA} )</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>0.19 ( \pm ) 0.10</td>
<td>0.009</td>
</tr>
<tr>
<td>7</td>
<td>0.75 ( \pm ) 0.43</td>
<td>0.04</td>
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</table>

Values are means \( \pm \) SD; \( n \), no. of patients. PV, pulmonary vein.
Overall, the values for atrial compliance (determined as the reciprocal of average \( K_{LA} \)) in our study are similar to the average distensibility of the postmortem human heart in the study by Liu et al. (15) and as estimated by Arakawa et al. (2). According to these authors, average distensibility, estimated as the ratio of the fractional change in volume to the fractional change in pressure, were 9.2 ml/mmHg between 0 and 5 mmHg, 2.3 ml/mmHg between 5 and 10 mmHg, and 1 ml/mmHg between 10 and 20 mmHg. In our study, the corresponding values averaged 9.2 ml/mmHg between 0 and 7.5 mmHg, 2.8 ml/mmHg between 7.5 and 13 mmHg, and 1.5 ml/mmHg between 13 and 21 mmHg.

**Limitations and conclusion.** Some limitations of this study must be discussed. A single-plane echocardiographic volumetric assessment was adopted for a patient population with a high incidence of ischemic heart disease and potential regional wall motion abnormalities. An analogous approach, however, was adopted for a patient population with a wide range of ejection fraction and a high proportion of ischemic etiology, giving satisfactory results in 25 patients with a wide range of ejection fraction and a high proportion of systolic function from DT compared with angiography (16). It is likely, however, that the adoption of a biplane approach could have improved our results, minimizing the scatter for the atrial stroke volume computation.

A fluid-filled catheter system was used to estimate LA pressure, with only a minority of patients (3/15) instrumented with a micromanometer. The characteristics, however, of our monitoring system had been previously tested in an in vitro condition, relative to a micromanometer-tipped catheter, showing that the response of the system should have been adequate for our purposes.

DT measured in our study appears substantially longer than the normal range. This might reflect the limited ability of reliably computing LV chamber stiffness from E waves, which have an inflection point in their deceleration portion (12). However, in a large population of 131 subjects with normal ejection fraction, in vivo validation of Little et al.’s (14) average chamber stiffness \( K_{LV} \) as assessed by a cosine (linear) function from DT compared with that assessed using a model-based image-processing parameterized formalism using a damped, simple harmonic oscillator has shown a highly significant correlation (13).

It could be hypothesized that, as hearts were being rewarmed, much of the effects of potassium cardioplegia may still be present and affect atrial and ventricular muscleularity, minimizing stiffness and filling parameters (6). We assessed the potential effects of cardioplegia on ventricular filling and stiffness by analyzing data in 10 patients, selected from a previously published larger series who underwent coronary bypass surgery (5). In these subjects, the mitral velocity profile could be assessed in the immediate pre- and postcardiopulmonary bypass period. End-diastolic LV diameter did not change after the cardiopulmonary bypass period in that study (from 53.5 ± 8.1 to 53.5 ± 6.5 mm, NS), whereas mean aortic pressure decreased (from 85.3 ± 17.5 to 71.7 ± 12.4 mmHg, \( P < 0.001 \)) and heart rate increased significantly (from 61.8 ± 13.2 to 77.4 ± 13.3 beats/min, \( P = 0.005 \)). \( K_{LV} \), however, which preoperatively was not different from the corresponding value in the present study, did not change postbypass (from 0.14 ± 0.08 to 0.20 ± 0.17 mmHg/ml, NS), as did E/A (from 1.32 ± 0.29 to 1.34 ± 0.26, NS). These observations suggest that the contribution of potassium cardioplegia to our results in the present study should have been minimal.

Finally, the estimation of atrial stiffness in the present study was not pressure independent (1), and this might have impacted on our results. However, our methodological approach was chosen to relate the atrial to the ventricular stiffness data, as assessed according to Little et al.’s (14) formula, which computes average \( K_{LV} \) from DT. Our data support the view that \( K_{LV} \) can be accurately calculated from noninvasively measured DT, because \( K_{LA} \) does not change and can be considered not different from zero during this period. These findings further underline the importance of noninvasive methods for the assessment of diastolic function, methods that avoid the risk, discomfort, limited repeatability, and expenses intrinsic to the invasive approach.

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