Effect of Left Ventricular Volume on Right Ventricular End-Systolic Pressure-Volume Relation
Resetting of Regional Preload in Right Ventricular Free Wall

Seiji Yamaguchi, Kai Tsuiki, Hiroshi Miyawaki, Yoshiaki Tamada, Ikuro Ohta, Hiroyasu Sukekawa, Masayuki Watanabe, Tadashi Kobayashi, and Shoji Yasui

Effect of left ventricular (LV) volume on right ventricular (RV) end-systolic pressure-volume relation (ESPVR) was investigated, and the mechanism was examined from a standpoint of the alteration of RV free wall mean fiber length. Twelve cross-circulated isovolumically contracting canine hearts in which both ventricular volumes were controlled independently were used, and RV-ESPVR was determined at three different LV volume levels. At small (10.2±0.6 ml), middle (15.3±1.0 ml), and large (20.5±1.4 ml) LV volume, the slope of the RV-ESPVR was 2.63±0.13, 2.74±0.13, and 2.89±0.12 mm Hg/ml, respectively, and each value was significantly different from the others (p<0.01). The volume intercept (V₀) of the relation (RV-V₀) was significantly decreased with the increment of LV volume (RV-V₀ in small, middle, and large LV volume; 3.92±0.68, 3.39±0.67, and 2.87±0.71 ml, respectively; p<0.01). In nine hearts, RV free wall lengths in latitudinal and meridional direction were measured at three LV volume levels when RV volume was held constant (16.1 ±1.1 ml). RV latitudinal end-diastolic length was significantly augmented with increasing LV volume (latitudinal length in small, middle, and large LV volume; 9.68±0.55, 9.81±0.56, and 9.92±0.55 mm, respectively). RV meridional end-diastolic length also increased significantly with increasing LV volume. We concluded that RV-ESPVR showed upward-leftward shift with increasing LV volume and that this shift could be, at least in part, explained by the alteration of end-diastolic length in RV free wall that occurred with constant RV volume (resetting of regional preload), probably due to the deformation of RV becoming more crescent. (Circulation Research 1989;65:623–631)

Right ventricular (RV) performance is determined, in part, by its specific geometry,1 which, in turn, is affected by the functional interaction between right and left ventricles. RV function could be directly influenced by left ventricular (LV) activity and vice versa. Using a variety of preparations, previous investigators have established that direct mechanical interdependence exists between the ventricles during diastole2–8 and systole.9–17 However, there have been few studies with quantitative analysis of the systolic influence,17 especially from LV to RV. The first purpose of this study is to examine the effect of LV volume on the RV end-systolic pressure volume relation (ESPVR) with a cross-circulated isolated contracting canine heart.

Interventricular septal shifting has been proposed as a mechanism of explaining the direct ventricular interdependence.8,17 There is another possible modulating factor; we hypothesized that LV volume change could affect RV shape, resulting in the change of RV mean fiber length under the condition that RV volume was constant. This hypothesis was tested by measuring latitudinal and meridional lengths in the RV free wall.

We observed that the RV-ESPVR showed upward-leftward shift by increasing LV volume and that, when RV volume was held constant, RV end-diastolic free wall lengths were augmented by increasing LV volume. Therefore, it is possible that the LV volume change alters RV regional preload with RV shape becoming more crescent and that this alteration of RV regional preload is associated with the shift of the relation line. In the present study, we describe systolic influence from LV to
An excised heart was perfused with blood from a support pressure. RV, perfusion pressure; LVP, left ventricular pressure; PP, perfusion pressure; RVP, right ventricular pressure; EMF, electromagnetic flow probe; RVP, right ventricular pressure; LVP, left ventricular pressure.

RV from a standpoint of the "resetting of regional preload" in RV free wall. This mechanism, as well as interventricular septal shifting for explaining direct ventricular interdependence, is considered to be important. Further, our observations provide implications for the mechanism of the shift of ESPVR that occurs under various circumstances.18-23

Materials and Methods

Surgical Procedure and Experimental Preparation

Twelve canine hearts were used to obtain an isolated cross-circulated heart preparation. In each experiment, both an experimental dog (12.7±0.2 kg; mean±SEM) and a larger support dog (20.1±0.9 kg) were anesthetized with sodium pentobarbital (30 mg/kg i.v.), intubated, and ventilated using room air with a respirator (model SN-480-3, Shinano, Tokyo, Japan). The chest of the experimental dog was opened bilaterally at fourth intercostal space. The thoracic cavity and the body were filled with granulated ice, which produced bradycardia and asystole after 10 minutes. Then, the pericardium was removed, and the heart was isolated from the systemic and pulmonary circulation.

The aortic valve cusps were sewn together, and a patch was sutured below the aortic valve through the mitral valve orifice (Figure 1). The chordae tendineae of the LV were cut, and a thin latex balloon (condom, 0.03 mm thick) attached to a rigid cannula was inserted into the RV cavity through the tricuspid orifice. The tricuspid orifice was fixed to a plastic ring, which was linked to the cannula. To prevent the development of the intraballoon pressure by excessively stretching the latex balloon, the maximum balloon capacity was confirmed before insertion. The tips of 6F micromanometer catheters (Millar Instruments, Houston, Texas) were positioned in the balloons of the LV and RV through the cannulae. The total volume of the balloon wall, the pressure transducer, and the adapter for mounting and securing the balloon was approximately 2.0 ml. The balloons were filled with saline, and their volume was controlled by slowly adding or subtracting saline through the cannulae. A drainage was set in the apex of the LV and RV to decompress these chambers from any Thebesian drainage. To ensure proper positioning of the balloons, strings were attached to the apical portion of each balloon and withdrawn through the drainage ports. The ascending aorta of the isolated heart was cannulated with the perfusion line. The isolated ventricle was suspended by a clamp at the tops of the three cannulae. The perfusion line was connected to a femoral artery of the heparinized support dog.

About 5 minutes after the perfusion line was opened, the isolated heart was defibrillated by a DC defibrillator on atria. The heart was paced at a constant rate of 130 beats/min with bipolar electrodes on the upper septum and atrium. Then, the isolated heart contracted isovolumically. Sixty to 90 minutes after the heart began to contract, LV and RV pressure and perfusion pressure were measured via a fluid-filled system with a P23Db strain gauge manometer (Statham, Oxnard, California) and checked for stability. After stability was confirmed, the experimental protocol started.

The perfusion and drainage circuits had a cannulation-type electromagnetic flow probe (type 20T; lumen size, 3 mm) connected to a square-wave electromagnetic flowmeter (ME-27, Nihon Koden). The flow signal was calibrated by the timed collection method with steady flows. The return flow to a support dog was set at the same rate as the perfusion flow from the support dog. The circuit contained a thermostat bath, by which the blood temperature was maintained at 37°C. The support dog was ventilated with room air, and supplemental oxygen was given when necessary to maintain arterial PO2 and PCO2 within their physiological ranges. Sodium bicarbonate and pentobarbital were infused throughout the experiment to maintain arterial pH within the physiological range and to keep the level of the anesthesia constant, respectively.

Placement of Ultrasonic Crystals

In nine of the 12 isolated hearts, RV latitudinal and meridional segment lengths were measured with two pairs of crystals (5 MHz, 2 mm in diameter) placed at the midpoint between the tricuspid...
valve and the pulmonary valve (Figure 1). Each crystal was implanted through a small stab wound on the surface of the RV. Both segments were approximately 1 cm in length, crossing with each other at their respective midpoints. The reproducibility of latitudinal and meridional RV lengths were confirmed by increasing and decreasing RV volume without the change of LV volume. End-diastole was defined as the time showing abrupt increase of LV dP/dt. Hemodynamic and sonomicrometric data were recorded on a thermal 12-channel oscillograph (Recti-Horitz-8K San-ct, San-ct, Japan).

Experimental Protocol

After confirming that the LV and RV pressure at a settled volume and perfusion pressure were stable, the data were collected according to the following protocols.

Protocol 1: Measurement of the shift of RV-ESPVR due to the change of LV volume. We defined end systole as the time showing isovolumically peak systolic pressure. In this protocol, we obtained RV-ESPVR at three LV volume levels. First, LV volume was fixed at relatively large volume (large: 20.5±1.4 ml; mean±SEM). RV volume was changed stepwise every 2 ml from 10.0±0.4 to 21.5±1.0 ml (with pressure ranging from 21.1±1.3 to 53.3±2.4 mm Hg). Second, LV volume was fixed at smaller volume (middle: 15.3±1.0 ml), and RV volume was changed. Finally, RV-ESPVR was constructed at smallest LV volume (small: 10.2±0.6 ml). In each experiment, the difference in volume between large and middle LV volumes was set to equal the difference between middle and small LV volumes. A least-squares linear regression was applied to generate the slope (Ee) and the axis intercept (Vo). The coefficients of the regression analysis were greater than 0.995 in all experiments.

Protocol 2: Measurement of the change of RV regional segment length by LV volume change.

Nine isolated hearts were used. RV volume was held constant (16.1±1.1 ml; mean±SEM), and LV volume was changed at three different levels. At each LV volume, RV latitudinal and meridional end-diastolic lengths were measured. Further, in five isolated hearts, RV volume was set at two different volume levels. RV latitudinal and meridional lengths were measured at three LV volume levels in each RV volume. RV regional area was calculated as multiplying the latitudinal end-diastolic length by the meridional end-diastolic lengths.

Statistical Analysis

All results are expressed as mean±SEM. RV-ESPVRs were fit by linear regression analysis. Student’s t test for paired observations was used to assess the significance of the differences in continuous data. The Wilcoxon U test (one-sided test) was used for length data of the five hearts. A value of p<0.05 was considered a statistically significant difference.

Results

The original pressure and flow signal tracings from a representative heart are shown in Figure 2. At each run in which LV volume was increased from 10 to 32 ml and decreased, RV volume was set constant (18, 14, and 10 ml, respectively). At 18 ml of RV volume, RV systolic and diastolic pressures were enhanced with increasing LV volume and returned with decreasing LV volume. RV pressure at 10 ml LV volume was 33.5/10.2 mm Hg (peak systolic/diastolic pressure) and increased stepwise to 46.5/19.3 mm Hg at 32 ml LV volume while RV volume was kept constant. Similarly, at 14 or 10 ml RV volume, RV pressure increased and decreased with adding and subtracting LV volume. The magnitude of the enhanced RV pressure with the increase of LV volume was greater with larger RV volume. This result indicates that the change of LV volume may influence RV systolic and diastolic performance and that the influence on RV performance may be greater in larger RV volume.

We analyzed RV systolic performance at three different LV volumes in terms of RV-ESPVR (protocol 1). RV-ESPVRs at three LV volumes in the same heart as presented in Figure 2 (heart 3 in Table 1) were illustrated in Figure 3. At 12 ml LV volume, RV-Ee was and RV-Vo were 2.59 mm Hg/ml and 7.75 ml, respectively. With the increment of LV volume, RV-Ee was increased to 2.90 mm Hg/ml, and RV-Vo was decreased to 5.23 ml at 28 ml LV volume. That is, RV-ESPVR showed a leftward-upward shift. In the RV pressure volume plane, it is clearly shown how LV volume influences RV systolic performance and why the influence is greater in larger RV volume.

The results of RV-Vo for all hearts are shown in Table 1. At small LV volume (10.2±0.6 ml), middle LV volume (15.3±1.0 ml), and large LV volume (20.5±1.4 ml), values of RV-Ee were 2.63±0.13, 2.74±0.13, and 2.89±0.12 mm Hg/ml, respectively, and each value was significantly different from the others (p<0.01). The magnitude of the change in RV-Ee was 0.26 mm Hg/ml with the increase of 10.3 ml in LV volume. In contrast with RV-Ee, RV-Vo was significantly decreased with increasing LV volume (RV-Vo in small, middle, and large LV volume; 3.92±0.68, 3.39±0.67, and 2.87±0.71 ml, respectively). The result of the steeper Ee and the smaller value for Vo of the relation line with the larger LV volume explains that the increment of RV pressure is more emphasized in larger LV volume (Figure 6).

The effects of LV volume on RV latitudinal and meridional free wall lengths were examined after the data of RV-ESPVR were obtained (protocol 2). Figure 4 shows gradual increase of RV latitudinal and meridional lengths with increment of LV volume (from 16 to 32 ml) although RV volume was constant. The systolic bulging in meridional RV length is due to the isovolumically contracting heart.
used in this study. In this model, the shortening in one direction or one area of the ventricle is considered to accompany bulging in the other direction or other area, and the former was the actual finding in most of all experiments.

The change of end-diastolic latitudinal and meridional lengths accompanied by the change of LV volume in nine hearts is shown in Table 2 and Figure 5. The RV latitudinal end-diastolic length in small LV volume (mean, 11.6 ml) was 9.68±0.55 mm and significantly increased to 9.81±0.56 mm and 9.92±0.55 mm in middle (mean, 17.1 ml) and large (mean, 22.7 ml) LV volume, respectively, although RV volume was constant (16.1 ml). The RV meridional end-diastolic length significantly increased from 11.18±0.45 mm in small RV volume to 11.3±0.46 mm in middle LV volume and to 11.38±0.47 mm in large LV volume. In Table 3, RV lengths and areas are shown at three LV volumes with two RV volume levels. In both RV volume levels, RV latitudinal and meridional end-diastolic lengths and areas were significantly increased with the increment of LV volume.

Discussion

In the present study, we observed that the increment of LV volume increased the $E_d$ of RV-ESPVR and reduced the RV-$V_o$. The increased $E_d$ and the decreased $V_o$ of the relation line both contribute to the enhancement of RV pressure at constant RV volume. A hypothesis that RV diastolic free wall length could be rearranged with the LV volume change was examined as a modulating factor of this systolic influence from LV to RV. RV free wall
end-diastolic lengths were latitudinally and meridionally increased with the increased LV volume although RV volume was constant. This phenomenon is considered to contribute, at least partly, to the changes in RV-E<sub>es</sub> and RV-V<sub>0</sub> (Figure 6).

Recently, several authors have investigated RV-ESPVR in isolated dog heart,17,24 in situ dog heart,25 and human heart.26-28 Maughan et al24 reported that RV-ESPVR was linear and sensitive to change in the contractile state, as reported for LV-ESPVR.29-31 RV-E<sub>es</sub> and RV-V<sub>0</sub> obtained under the isovolumic beat of LV enhanced RV pump function rather than that of an ejection beat of LV. However, to our knowledge, none of those authors has dealt with the phenomenon from a standpoint of RV-ESPVR; the one exception is the study of Maughan et al17 who reported that, when peak left ventricular pressure was fixed at the two stages of 0 mm Hg and 100.4±2.8 mm Hg, RV-V<sub>0</sub> in 0 mm Hg LV pressure was significantly increased as compared with that in 100.4 mm Hg LV pressure without the change of RV-E<sub>es</sub>. Our observation of the upward-leftward shift is different from their observation of parallel shift. The discrepancy may be due to the difference of the techniques used. We used fixed LV volume, which was different from their fixed peak LV pressure. When peak LV pressure is fixed, LV volume must be variable. For describing RV-ESPVR at fixed LV pressure, LV volume might be increased with the decrease of RV volume because the functional enhancement from RV to LV (contralateral influence) may become gradually lower with decreasing RV volume. As a consequence, RV-E<sub>es</sub> is considered to be unchanged in their study.

We investigated the influence of LV volume change on the RV systolic pumping function in terms of the end-systolic pressure-volume relation. It is well known that LV volume increase elevates right ventricular diastolic pressure. The analysis of systolic pumping function using ESPVR deals with the net effect of diastolic pressure and developed pressure. There have been several reports regarding the systolic interaction from LV to RV.6,9,10,12-14,17 Elzinga et al14 demonstrated in isolated cat hearts that an isovolumic beat of LV enhanced RV pump function rather than that of an ejection beat of LV. However, it has been found that E<sub>es</sub> is sensitive to inotropic interventions in LV25,30 and RV24 and practically independent of loading condition. Recently, it has been documented that LV-ESPVR was partly dependent on stroke volume23 and peripheral resistance,20,23 but at the same inotropism, E<sub>es</sub>.
has been generally considered to be unchanged in both ventricles. Our study showed that LV volume change could alter RV-E under the condition that the contractile state was considered to be constant throughout the experiment.

The term interdependence includes many different phenomena. As a factor that modulates the direct interdependence between RV and LV, the transseptal interaction has been proposed. Little et al used assumptions in modeling transseptal inter-

<table>
<thead>
<tr>
<th>Table 2. Right Ventricular Latitudinal and Meridional Lengths at Different Left Ventricular Volumes</th>
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</thead>
<tbody>
<tr>
<td><strong>Left ventricular size</strong></td>
</tr>
<tr>
<td>RV latitudinal length (mm)</td>
</tr>
<tr>
<td>RV meridional length (mm)</td>
</tr>
</tbody>
</table>

Values are mean±SEM (n=9). Right ventricular volume was held constant (16.1±1.1 ml; mean±SEM) at each experiment. RV, right ventricular.

*p<0.01 vs. large left ventricular volume.

†p<0.01 vs. middle left ventricular volume.

‡p<0.05 vs. large left ventricular volume.
actions during diastole and derived a set of equations relating the ratio of changes in LV pressure over changes in RV pressure to the ratio of septal to free wall elastance. Santamore et al.34 modeled the right and left ventricles as a two-compartment model with variable wall compliances. The study of Maughan et al.,17 based on the same assumption as that of Little et al., developed a three-elastance model of RV and LV free walls and interventricular septum. The direct interdependence from LV to RV is modulated by yet another factor; we recognized that RV free wall length changed meridionally and latitudinally with LV volume change at constant RV volume. A change of RV free wall end-diastolic length means a change of RV regional preload, which results in the shift of RV-ESPVR in our study. In other words, LV volume change can alter the setting of RV preload and this “resetting of RV regional preload” could be associated with the upward-leftward shift of the relation line with the increment of LV volume (Figure 6) although the transseptal interaction and other mechanisms would also contribute to the shift of the relation line.

An explanation for resetting of RV regional preload would be that, when LV volume increases at constant RV volume, RV cavity would result in more crescent deformation because RV that adheres to LV is latitudinally and meridionally stretched. The schematical latitudinal plane is illustrated in Figure 6. If LV volume is increased at constant RV volume, the distance of the connection points of RV free wall on LV can be elongated in anteroposterior direction and then the latitudinal length in large LV volume is longer than that in small LV volume. The latitudinal RV cavity in large LV volume becomes more crescent. In the meridional plane, the same explanation can also be applied.

It seems likely that, if RV regional preload is reset with LV volume change, RV-ESPVR would show a parallel shift. We measured RV latitudinal and meridional end-diastolic lengths and areas at

**TABLE 3. Right Ventricular Lengths and Area at Different Left Ventricular Volumes at Large and Small Right Ventricular Volume Levels**

<table>
<thead>
<tr>
<th></th>
<th>Large RV size (22.4 ml)</th>
<th>Middle RV size (17.2 ml)</th>
<th>Small RV size (12.0 ml)</th>
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</thead>
<tbody>
<tr>
<td><strong>Large LV size</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV (22.4 ml)</td>
<td>10.89±0.35</td>
<td>10.79±0.36*</td>
<td>10.65±0.32*</td>
</tr>
<tr>
<td><strong>Middle LV size</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV (17.2 ml)</td>
<td>10.42±0.50</td>
<td>10.31±0.49*</td>
<td>10.18±0.49*</td>
</tr>
<tr>
<td><strong>Small LV size</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV (12.0 ml)</td>
<td>10.04±0.32</td>
<td>9.92±0.32*</td>
<td>9.85±0.33*</td>
</tr>
<tr>
<td>RV latitudinal length (mm)</td>
<td>10.82±0.35</td>
<td>10.79±0.36*</td>
<td>10.65±0.32*</td>
</tr>
<tr>
<td>RV meridional length (mm)</td>
<td>12.42±0.50</td>
<td>12.31±0.49*</td>
<td>12.18±0.49*</td>
</tr>
<tr>
<td>RV area (cm²)</td>
<td>1.38±0.10</td>
<td>1.33±0.09*</td>
<td>1.30±0.10*</td>
</tr>
</tbody>
</table>

Values are mean±SEM (n=5). RV, right ventricular; LV, left ventricular.

*p<0.05 vs. large left ventricular volume at the same RV volume level.

*p<0.05 vs. middle LV volume at the same RV volume level.

**FIGURE 5. Bar charts showing effect of altering left ventricular volume (LV V) on right ventricular latitudinal and meridional end-diastolic lengths in nine hearts. Right ventricular volume was held constant. See also Table 2.***

**FIGURE 6. Schematic illustration for explanation of resetting of right ventricular regional preload due to left ventricular (LV) volume change. If LV volume is increased from small (S) to large (L) at constant right ventricular volume (RVV), the distance between adherence points of RV free wall on LV can be elongated in anteroposterior direction (upper panel). RV latitudinal free wall mean fiber length (l_L) in large LV volume can become longer than that (l_L) in small LV volume. This resetting can contribute, at least in part, to the upward-leftward shift in the RV end-systolic pressure volume relation (lower panel). RVSP, RV peak systolic pressure.**
three LV volume levels in two states of RV volume (Table 3). If the magnitude of the increase in RV area with the increment of LV volume at large RV volume was higher than that at small RV volume, the upward-leftward shift of the relation line observed in the present study might be explained by resetting of RV regional preload. However, the magnitude of the increase in RV area by increasing LV volume at large RV volume (6.2% increase) was greater than that at small RV volume (4.5% increase) although it was not significant. The shift of RV-V₀ with the volumetric change seems likely to depend on the "resetting of RV free wall regional preload," but the resetting may not fully explain the change of RV-Ees. If LV volume is increased at the same RV volume, RV pressure is augmented and the contralateral reflection from RV to LV may also occur probably due to a shifting of the interventricular septum. As a consequence, LV pressure is augmented and RV pressure is further increased. This complicated relation between the two ventricles may result in the shift of RV-Ees. Further, we have to consider the change of end-diastolic length in interventricular septum. We speculate that the septal end-diastolic length may be increased with the increment of LV volume as shown in Figure 6. This effect might also contribute to the shift of RV-ESPVR although it is unknown what proportion of the increase of preload in interventricular septum contributes to the contractile function of LV and RV. This "resetting of interventricular septal preload" may resemble the concept of interventricular septal shifting. We would speculate that the shift of RV-ESPVR with LV volume change might depend on the resettings of preload in RV free wall and interventricular septum. Further study is needed with regard to septal preload and septal contribution to RV and LV contractile function.

It is important to recognize that the RV geometrical change is, at least in part, responsible for the shift of RV-ESPVR. This study supports the concept that the deformation of a ventricle leads to the shift of ESPVR. We have previously shown the parallel shift of LV-ESPVR by changing coronary blood flow. It can be speculated that an increase of LV wall thickness due to an increase in coronary blood flow may result in an increase of regional preload at constant LV volume and this resetting of regional preload could be considered to shift the relation line leftward. The verification of the relation between ESPVR and "resetting of regional preload" should be investigated for various circumstances where ventricular geometry is changing.

End-diastolic volume has been considered to be a reliable index of myocardial preload, and the relation between chamber volume and mean myocardial fiber length has been believed to be linear. However, our study shows that RV geometrical change can alter mean RV fiber length with constant RV volume. Under the circumstances where ventricular geometrical changes occur, mean fiber length is a more reliable index of preload than ventricular volume.

The importance of the pericardium in modulating ventricular interaction still remains unknown. The intact and surgically reapproximated pericardium has been shown to exert a restraint on cardiac dilation and augment interactive coupling between the two ventricles in isolated perfused canine preparations and in open-chest dog. There remains little doubt that the intact pericardium modulates ventricular interaction; however, the absolute magnitude of this effect remains unclear. In the present study, the pericardium was removed because it was not the intent of the present study to determine the role of the pericardium in ventricular interaction. Further investigation is needed to quantitatively integrate these factors into the evaluation of ventricular interaction.

In conclusion, RV-ESPVR showed upward-leftward shift with the increment of LV volume, and this shift can be partly explained by the resetting of regional preload in RV free wall due to RV deformation.

Acknowledgments

We gratefully acknowledge Mr. Eiji Tsuchida, Jun Otomo, MD, and Akio Fukui, MD, for their technical assistance in this study.

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KEY WORDS: end-systolic pressure-volume relation, right ventricle, ventricular deformation
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doi: 10.1161/01.RES.65.3.623

*Circulation Research* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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