LEFT AND RIGHT VENTRICULAR PRESSURE DIAMETER RELATIONSHIPS DURING VENTRICULAR STANDSTILL IN THE DOG

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It has been assumed that ventricular standstill induces left ventricular dilation because filling continues without emptying. Left and right ventricular diameters by echocardiography and pressures were monitored continuously in 21 intact dogs before and during ventricular standstill induced by coronary embolization (E), potassium infusion (K⁺), or calcium infusion (Ca²⁺) in 7 dogs respectively. At the onset of ventricular standstill, left ventricular pressure rose from an end-diastolic of 4.6 ± 2.2 to 16.6 ± 3.6 mmHg after E, 5.4 ± 3.2 to 18.0 ± 3.3 mmHg after K⁺, and 6.3 ± 2.1 to 16.6 ± 3.6 mmHg after Ca²⁺ (all p < 0.001). Left ventricular dimension rose from an end-diastolic of 44.6 ± 8.0 to 49.4 ± 8.5 mm after E, 43.1 ± 5.1 to 46.3 ± 4.6 mm after K⁺ but fell from 40.6 ± 5.1 to 33.9 ± 3.4 mm after Ca²⁺ before ventricular standstill occurred. During the first 5 min of ventricular standstill, left ventricular pressure gradually fell from 16.6 ± 3.6 to 5.4 ± 1.1 mmHg (E), 18.0 ± 3.3 to 5.9 ± 2.0 (K⁺), 16.6 ± 3.6 to 6.6 ± 1.7 (Ca²⁺) (all p < 0.001) while left ventricular diameter gradually fell to 40.7 ± 7.0 mm (E), 35.0 ± 5.1 (K⁺), 29.6 ± 3.6 (Ca²⁺) (all p < 0.001). Right ventricular pressure initially rose from 2.9 ± 1.6 mmHg to 12.1 ± 3.1 (E), from 2.7 ± 1.3 to 12.0 ± 2.0 (K⁺), from 2.7 ± 1.0 to 12.6 ± 2.6 (Ca²⁺) within 15 sec, then gradually fell to 6.6 ± 1.1 (E), 6.1 ± 1.9 (K⁺), and 7.9 ± 1.8 (Ca²⁺) in 5 min whereas right ventricular diameter rose progressively from 4.4 ± 3.0 mm to 21.0 ± 1.7 (E), 4.7 ± 2.6 to 20.3 ± 1.3 (K⁺), 4.2 ± 1.1 to 18.1 ± 2.8 (Ca²⁺) in 60 sec (all p < 0.001). Thus ventricular standstill is characterized by early left ventricular emptying and inhibited left ventricular filling despite continued right ventricular filling, a finding that might reflect the different intrinsic properties of the two ventricles.

Key Words:
Ventricular standstill
Echocardiography
Right and left ventricle
Pressure and diameter

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**Abbreviations:** RVD = right ventricular diameter, LVD = left ventricular dimension (p = probability of significant difference between each succeeding value) VS = ventricular standstill, C = control, Dd = end-diastolic diameter, Ds = end-systolic diameter, MI = myocardial infarction, O = the onset of ventricular standstill. The onset of VS was taken at the point of the end of the last heart beat. P was calculated between LVD at 0 and the preceding Dd.
gram has been shown to be a reliable method of determining the left ventricular diameter in dogs as well as in humans. In this paper we have used non-invasive echocardiography in combination with pressure recording to determine the time course of the right and left ventricular diameters and pressures during VS.

METHODS

Twenty-one mongrel dogs weighing 17.7 to 34.3 kg (mean 26.8 kg) were used. They were anesthetized with 30 mg/kg of pentobarbital. Respiration was maintained with a cuffed endotracheal tube connected to a Harvard Respirator. They were classified as follows into 3 groups.

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Fig. 2. A representative echocardiogram during premature ventricular beats (PVC) and bradycardia induced by myocardial infarction. (A) Initial stage of PVCs and bradycardia. Note the increased RV diameter and decreased LV diameter. (B) Late stage of bradycardia and asystole. Echo clouds in the right ventricle was caused by injecting 5 ml of dextran. Note the increased right ventricular diameter. Abbreviations: The same as in Fig. 1.

according to the method used to induce VS:

Group I consisted of 7 dogs weighing 18.8 to 32.6 kg (mean 27.2 kg). VS was induced by myocardial infarction caused by a mercury injection into the circumflex or the left anterior coronary artery using a Mollar catheter. Repeated embolization was performed 15 min after initial embolization until VS was induced.

Group II consisted of 7 dogs weighing 17.7 to 33.1 kg (mean 28.1 kg). VS was induced by injecting KCl (K⁺) intravenously.

Group III consisted of 7 dogs weighing 18.5 to 34.3 kg (mean 25.4 kg). VS was induced by injecting CaCl₂ (Ca⁺⁺) intravenously.

PROCEDURE

Echocardiographic studies were performed on the dogs in the right recumbent position with the transducer placed on the fourth right intercostal space. Echocardiograms were recorded using Ekoline 20 with a repetition rate of 1,000 im-
Fig. 3. Serial changes of ventricular diameters during MI and VS. Abbreviations: The same as in Fig. 1. CCA = circumflex coronary artery. Note the increased left ventricular diameter after myocardial infarction and the decreased LV diameter after VS, indicating the terminal event of the heart.

Fig. 4. A representative echocardiogram during VS induced by K+ infusion. Abbreviations: The same as in Fig. 1. Ds = end-systolic diameter.

pulses/sec. The echo signal was fed to a Honeywell 1856 A Line Scan recorder for continuous recording on light-sensitive paper. The echocardiographic technique used has been described previously in detail. In brief, an echocardiogram was taken using scanning method and 3 ml of dextran was injected into the RV and LV whenever it was required to confirm the right ventricular chamber, intraventricular septum (IVS) and left ventricular endocardium.

Right and left heart catheterization were performed in retrograde from the incised femoral vein and artery using #7 F Coumand catheters. Pressures were recorded using Statham P23Db transducers and Hewlett Packard multichannel recorder. The zero for pressures were taken at

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Fig. 5. A representative echocardiogram during VS induced by Ca²⁺ infusion. VS was seen for 5 sec, during that time left ventricular diameter decreased from 37 to 31 mm, while right ventricular diameter increased.

Fig. 6. Time course of changes of the right and left ventricular internal diameters during VS induced by MI. Significance of difference was examined between each successive value by paired student t-test. Abbreviations: MI = myocardial infarction, LVD = left ventricular diameter, RVD = right ventricular diameter, Dd = end-diastolic diameter, Ds = end-systolic diameter.

Fig. 7. Time course of changes of the right and left ventricular internal diameters during VS induced by K⁺ infusion. Abbreviations: VB = a phase of ventricular beats, other abbreviations are the same as in Fig. 6.

Experimental Design
Animals were studied according to the following protocol:
1. Echocardiographic and pressure changes during VS were followed 10 min after VS at a paper speed of 10, 25 and 50 mm per sec.
2. Ten min after VS, the thorax was opened through the fourth left intercostal space and the pericardium was opened to expose the heart. Dimensional changes of the right and left ventri-
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Abbreviations: RVP = right ventricular pressure, LVP = left ventricular pressure, C = control, O = the onset of ventricular standstill. The onset of ventricular standstill was

ucle were observed. Echocardiogram was taken with the transducer directed to the right ventricular surface using the scanning method.

3. Right and left ventricular pressures and echocardiogram were simultaneously recorded for each dog in each group during VS.

Calculations and Data Analysis

The data were analyzed in the following manner:

1. For each study, simultaneous pressures of RV and LV were calculated at onset (0), 5, 10, 30, 45, 60 sec and 2, 3, 5 and 10 min after VS. Control pressures of mean end-diastolic RV and LV of 6 consecutive cycles were calculated before inducing VS.

2. Echocardiographically determined right and left ventricular diameters were determined during VS at the same time interval as mentioned in pressure measurements. Control values of RV diameter (RVD) and LV diameter (LVD) at end-diastole and LVD at end-systole were calculated before inducing VS. RVD and LVD were calculated in the open chest by placing the echotransducer directly on the right ventricular surface.

All comparisons were made by paired Student t test and the data presented as mean ± standard error of the mean (SEM).

Methods of Inducing VS

The amount of mercury used to induce VS was 0.3–0.6 ml (mean 0.5 ml). The K⁺ used was 47–153 mg/kg, (mean 104 mg/kg) using a solution of 70 mg/ml. The amount of Ca⁺ used was 96–256 mg/kg (mean 198 mg/kg) using a solution of 200 mg/ml.

The time required for inducing VS was 16–879 sec (mean 156 sec) after MI, 6–125 sec (mean 39 sec) after K⁺ infusion, and 5–98 sec (mean 29 sec) after Ca⁺ infusion.

RESULTS

Time Course of RV and LV Diameter Changes During VS

Japanese Circulation Journal Vol. 46, March 1982
AND LEFT VENTRICULAR PRESSURES DURING VS

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\( p = \text{probability of significant difference between each succeeding value} \)

\[ \text{taken at the point of the end of the last heart beat. } \text{VS} = \text{ventricular standstill} \]

Fig.9. Time course of changes of the right and left ventricular pressures during VS induced by MI. Significance of difference was examined between each successive value by paired Student \( t \) test. Abbreviations: MI = myocardial infarction, LVP = left ventricular pressure (end-diastolic pressure at C, MI and O), RVP = right ventricular pressure (end-diastolic pressure at C, MI and O).

Table I shows the changes of echocardiographic parameters during the course of VS.

Figures 1, 2, and 3 show representative echocardiographic tracings during VS induced by MI. Figures 4 and 5 show representative echocardiographic tracings during VS induced by \( K^+ \) and \( \text{Ca}^{++} \), respectively.

Figures 6, 7, and 8 depict the time course of RV and LV diameter changes during VS induced by MI, \( K^+ \) and \( \text{Ca}^{++} \) infusions, respectively. During the first 30 sec, LVD progressively fell in all 3 groups. Ten min after VS, LVD in the MI and \( K^+ \) groups was in between end-diastolic (Dd) and end-systolic diameter (Ds) of controls (nearer to control Dd), while the LVD in the \( \text{Ca}^{++} \) group was near control Ds.

On the other hand, RVD rose progressively during the first 30 sec in all 3 groups. During the rest of the course of VS, the RV remained enlarged.

RVD and LVD measured in the open chest correlated well with those at 10 min after VS in

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the MI and K⁺ groups. RVD and LVD in the open chest showed lower values for the Ca⁺⁺ group 10 min after VS.

Time Course of RV and LV Pressure Changes during VS

Table II shows the RV and LV pressure changes during VS. Figures 9, 10 and 11 depict the time course of pressures during VS induced by MI, K⁺ and Ca⁺⁺ infusion.

At the onset of VS, left ventricular pressure (LVP) did not change from control values of LVP at end-diastole of a heart beat just prior to the VS in the 3 groups. During the first minute of VS, LVP gradually fell while RVP gradually increased and equilibrated.

RV and LV Diameter Changes during Resuscitation from VS and Ventricular Fibrillation

Figure 12 depicts the echocardiogram during resuscitation from VS by injection of ephedrine into RV. LVD increased, while RVD decreased.

Figure 13 depicts the echocardiogram during resuscitation by counter shock on ventricular fibrillation induced by catheter impulse into LV. LVD at end-diastole increased, while RVD decreased.

DISCUSSION

Critique of the Methods

Certain assumptions inherent in evaluating the RV and LV hemodynamic changes during VS are basic to the conclusions pertaining to the effects of VS. These assumptions, therefore, deserve enumeration and evaluation.

The limitations of the echocardiographic method in determining RV and LV diameters are:

First, accurate ultrasonic measurements of RV and LV diameter can only be made after careful scanning with the ultrasonic probe at the plane of the apex of the anterior and posterior mitral valve leaflets and then being tipped lightly infero-medially to identify the septal and posterior wall of the endocardial surfaces. The transducer is placed on the fourth right intercostal space. Identification of both sides of the interventricular septum and distinction between posterior endocardial and epicardial surfaces are difficult to make on occasions, but these are the necessary prerequisites to obtaining meaningful data. Injection of 4 ml Dextran2 into the RV and LV helped identification of the RV and LV cavity by causing echo clouds that are stronger than those produced by indocyanine green8,7 or saline8.

Second, even with accurate measurements of the RV and LV diameters it is possible that the cardiac axis might be changed during VS. Thus, if major axis lengthening or shortening plays a more significant role in VS than does minor axis (internal diameter) shortening, a considerable bias might be introduced by relying on only the internal diameter to characterize changes induced by VS.9

Third, in physiological conditions, the mitral valve echoes can be taken as a landmark for measuring the LV diameter.10 However, when this landmark is lost during VS, there can be no standardization of the measurements of the RV and LV diameters. In this study, this landmark was not lost during the first minute after VS in all cases.

Fourth, when the findings among 3 independent observers in measuring RV and LV diameters were compared, highly significant correlations were obtained with a maximum error difference of 2 mm.

Different Hemodynamic Effects of VS on RV and LV

Our studies have shown that VS caused LV shortening and RV dilatation in 3 groups. There are several mechanisms that might account for this phenomenon.

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Fig.12. A representative echocardiogram during resuscitation from VS by injection of ephedrine into RV. Note that LVD increased, while RVD decreased.

Fig.13. A representative echocardiogram during resuscitation from ventricular fibrillation by counter shock. Note that LVD increased, while RVD decreased.

1. Differences in architecture between RV and LV: The great differences in pressure and diameter changes in the two ventricles is consistent with the differences in their architecture within normal physiological changes. The LV is a cylindrical contour made of a thick cuff of deep myocardial bundles which develops a very high internal pressure during contraction. On the other hand, the RV has an old-fashioned bellows-like contour, containing a relatively thin layer of these fibers. This indicates that the configuration of the RV is ideal for accepting a large volume of blood.

2. Differences in pulmonary and systemic circulation: Since pulmonary vessels constitute a low pressure, distensible system, any slight increase in outflow pressure at the left ventricle or relative increase of input from the right ventricle will cause considerable quantities of blood to accumulate within the lungs, and left ventricular pressure is transmitted across the mitral valve directly to the pulmonary veins and capil-
Therefore, the proximate hemodynamic cause of the “pulmonary barrier” which prevents an inflow of blood from the RV into the pulmonary artery is the higher pressure in the pulmonary artery which is a reflection of the higher pressure in the LV during VS. Thus, the RV progressively dilates during the course of VS.

On the other hand, systemic arteries are pressure reservoirs with the relatively fixed capacity due to the elastic properties of the walls, while venous systems are reservoirs of blood under low pressure and in the variable capacity. Thus, after VS, blood was squeezed out into the venous systems, allowing venous return to continue by a slight pressure difference between the veins and RV.

3. Strong activation of the cardiovascular reflexes: The degree of filling of the vessels is one of the major factors affecting blood flow from the peripheral circulation into the heart. Strong activation of the cardiovascular reflexes by low blood pressure induced by VS causes a high degree of sympathetic tone and thereby constricts all the peripheral vessels. Since arterial constriction is stronger than venous constriction due to the anatomical difference, venous pooling comes to the maximum and via a tergo propels venous return until the pressure gradient between RV and LV is balanced. Thus, RV is dilated, while LV is decreased during VS.

**Hemodynamic Differences of Ca**⁺ and K⁺ during VS**

In the normal heart during diastole, the ventricles are subjected to the stress of a hydrostatic pressure. The rate and extent of filling will depend not only on the hydrostatic pressure and duration of diastole, but also on the mass, elasticity and viscosity of the ventricular wall and the moving blood. This change in the ventricular pressure-inflow relationship (expressed as changes in mechanical impedance) would indicate changes in the physical properties of the ventricular wall during filling.

The antagonistic action of Ca⁺ and K⁺ on cardiac contractility is well known: increasing K⁺ causes a decreased contractile response of the isolated frog heart while the opposite effect can be produced by increasing the concentration of the extracellular Ca⁺. Changes in contractility induced by Ca⁺ and K⁺ might be accompanied by changes in the physical properties of the ventricular wall. Thus, the resting fiber length was in between the end-diastolic and end-systolic diameters in the 3 groups. According to the state of filling of the heart, the length of the “resting” muscle may be anything between that of the maximal dilatation or lengthening and maximal contraction.

**Clinical Implications of This Study**

This study provides further evidence that the terminal events of the heart are the dilatation of RV and the decrease of LV diameter. These findings are consistent with those reported in the companion paper on the effects of ventricular fibrillation on RV and LV. This “overfilled right” and “underfilled left” cardiac syndrome might indicate that the diastolic phenomena of the left ventricle is not a passive process but an active one.

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