THEORETICAL VALIDITY OF PREDICTING RIGHT VENTRICULAR SYSTOLIC PRESSURE BY A QUANTITATIVE INDEX OF INTERVENTRICULAR SEPTAL DISPLACEMENT IN ACUTE RIGHT VENTRICULAR PRESSURE OVERLOAD

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A leftward shift of the interventricular septum (IVS) of the heart is observed in patients with right ventricular pressure overload (RVPO). We simulated the leftward displacement of IVS in a modified ellipsoidal model on the assumption that the IVS generates the same tension as the left ventricular (LV) free-wall in acute RVPO, and derived the relational equations between ventricular pressures (RVP, LVP) and eccentricity index ($EI = LVAPD/LVSLD$, LVAPD: left ventricular anterior-posterior diameter, LVSLD: left ventricular septal-lateral diameter). The equations indicate that RVP/LVP correlates with simultaneous EI, independent of the absolute LV wall tensions and the LV size. To confirm this result, we undertook recurrent pulmonary embolizations in anesthetized open-chest dogs, and analyzed the relationship between RVP/LVP and EI at four phases in systole through the course of RVPO and shock. The advance of RVPO shifted the peak of RVP toward late-systole and made the values of RVP/LVP and EI significantly greater at late-systole than at early-systole. There were significant linear relationships between instantaneous EI and RVP/LVP at each phase, except for the early systole in the shock stage, and the regression lines on all phases were similar to one another. These results are consistent with our theoretical ones. Therefore we conclude that it is reasonable to predict RVP by using EI, theoretically and experimentally.

RIGHT ventricular pressure overload (RVPO) occurs frequently in many types of patients with primary, or secondary pulmonary hypertension.¹⁻³ Since accurate assessment of right ventricular pressure (RVP) requires cardiac catheterization, a simple and noninvasive method for the assessment of RVP would be of considerable value.

Recently many echocardiographic studies have documented the leftward displacement

of the interventricular septum (IVS) in patients with chronic RVPO, and several investigators have indicated that some indices of the "end-systolic" IVS displacement correlate with the right ventricular "peak systolic" pressure (RVPSP) or the "peak systolic" pressure ratio (RVPSP/LVPSP, LVPSP: left ventricular peak systolic pressure).¹⁻³ So far, EI ,^{2,4,5} NSC (normalized septal curvature)¹ and LVSCI (left ventricular systolic circular index)³ have been proposed.

However, the relationship of end-systolic RVP with RVPSP may not remain constant, because the peak point of the RVP wave shifts toward the late-systolic phase with

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TABLE I SYMBOLS AND DEFINITIONS

Symbol	Definitions
δ_1	the latitudinal stress of the free wall
δ_2	the longitudinal stress of the free wall
δ_3	the latitudinal stress of the IVS
δ_4	the longitudinal stress of the IVS
R_1	the latitudinal radius of the free wall
R_2	the longitudinal radius of the free wall
R_3	the latitudinal radius of the IVS
R_4	the longitudinal radius of the IVS
H_1	the thickness of the free wall
H_2	the thickness of the IVS
a	LVAPD (the anterior-posterior short axis diameter)
b	LVSLD (the septal-lateral short axis diameter)
Θ	the central angle of the IVS
Area	the area of left ventricular latitudinal plane
ri	the ideal radius of the IVS = $\sqrt{\text{Area}/\pi}$
LVperimeter	the perimeter of left ventricular latitudinal plane

RVPSP elevation. Accordingly, it is controversial whether estimation of RVPSP is possible by using end-systolic IVS-displacement indices⁵. Furthermore, it has not been reported whether the estimation could be applied in situations where LVPSP is markedly reduced, as in pulmonary embolic shock.

Therefore, we attempted to show the direct relationship between IVS-displacement and the simultaneous ventricular pressures (RVP, LVP) using a theoretical model. We then studied the actual relationship in 4 points of systole through the course of RVPO and shock in a canine model of acute pulmonary microembolism and confirmed the feasibility of the prediction of RVPSP by a IVS-displacement index.

METHODS

I. Theoretical bases

In order to discuss how the right and left ventricular pressures affect IVS-displacement,

we used a modified ellipsoidal model of the left ventricle (LV). This model was conceived by Olsen et al⁶ to calculate the wall stresses of the deformed left ventricle in acute experimental RVPO.

They considered the LV free wall (FW) as a hemi-ellipsoid and the IVS as a part of another ellipsoid (Fig. 1-B), and applied the generalized Laplace's law to each ellipsoid individually⁶. [The generalized Laplace's law of ellipsoid: $P/H = \delta_i/R_i + \delta_{ii}/R_{ii}$, (δ_i , δ_{ii} , R_i , R_{ii} : latitudinal and longitudinal wall stresses and radii of curvature respectively, P : internal pressure, H : wall thickness)].

In the model, the FW was assumed to deform according to LVP and its wall tension (wall thickness $\times$ wall stress). The IVS was also assumed to deform according to the ventricular transseptal pressure (TSP = LVP - RVP) and its wall tension. Namely,

$$\text{LV-FW: } \delta_1/R_1 + \delta_2/R_2 = \text{LVP}/H \dots (1)$$

$$\text{IVS: } \delta_3/R_3 + \delta_4/R_4 = (\text{LVP} - \text{RVP})/H \dots (2)$$

where R_1 , R_2 , R_3 , R_4 are, respectively, the latitudinal and longitudinal radii of the FW and the IVS, δ_1 , δ_2 , δ_3 , δ_4 are the latitudinal and longitudinal stresses of the FW and the IVS, H_1 , H_2 are the wall thicknesses of the FW and the IVS. (See also Table I)

In these equations, the geometry of the LV is a complex function of the ventricular pressures (RVP, LVP) and wall tensions. In order to assess the direct relationship between ventricular pressures and IVS-displacement, it is necessary to cancel the wall tension in the equations, because the wall tension is also a dependent variable of the ventricular pressures.

To simplify those equations, we considered that the radii of curvature changes proportionally in both longitudinal and latitudinal directions according to the advance of RVPO, and supposed that the changes in the longitudinal/latitudinal radius ratios of the IVS and the FW are negligible throughout the course of RVPO.

$$R_1/R_2 = R_3/R_4 \text{ (through the whole course of RVPO)} \dots (3)$$

And then, to represent the LV-geometry as a function of only the right and left ventricular pressures, we postulated that the wall tension of the IVS and that of the LV-

Fig.1. Panel A: An illustration of the basic preparation for measurement of the two-dimensional geometry of the left ventricle. The location of the implanted ultrasonic crystals (A, P, S, L) allowed accurate measurement of the left ventricular anterior-posterior diameter (LVAPD: A to P) and septal-lateral wall diameter (LVSLD: S to L). Panel B: the geometrical model of the left ventricular latitudinal plane. The free wall of the LV (P-L-A) is modeled by a hemi-ellipsoid, and the IVS is composed of a small part of another ellipsoid (P-S-A). Q is the center of the ellipsoid for the IVS. θ is the central cornus of the IVS. Panel C: the model in control. Right ventricular pressure (RVP) is low and negligible compared with left ventricular pressure (LVP). The radii of curvature (R_1 , R_3) and wall tensions (δ_1 , δ_s) are similar in the IVS and the free wall. Panel D: The model of RVPO. According to the increment in RVP, the radius of septal curvature (R_s) increases and the IVS shifts proportionally to the left to maintain the wall balance.

FW are approximately equal during systole in this model,

$$\delta_1 \times H_1 \doteq \delta_3 \times H_2, \quad \delta_2 \times H_1 \doteq \delta_4 \times H \dots \dots (4)$$

This assumption is based on the report in acute RVPO experiment by Olsen⁶

These assumptions enable us to cancel the wall tensions in equations (1) and (2).

The equations (1)–(4) yield,

$$R_1/R_3 \doteq 1 - RVP/LVP \dots \dots \dots (5)$$

The radii of curvatures (R_1 , R_3) can be derived from the left ventricular short axis diameters by using the geometrical model. Re-

fering to Fig. 1-B,

$$R_1 = a/2, \quad R_3 = \frac{(a^2 - 2ab + 2b^2)/2}{(2b - a)} \dots \dots (6)$$

$$(\because R_3^2 = R_1^2 + (QO)^2, (QO) = R_3 - (OS), (OS) = b - (a/2) \text{ in Fig. 1-B})$$

where “a” is the anterior-posterior short axis diameter, “b” is the septal-lateral short axis diameter. These equations ((1)–(6)) yield the following:

$$RVP/LVP = \frac{2(a - b)^2}{(a^2 - 2ab + 2b^2)} \dots \dots (7)$$

To clarify the relationship between the degree of IVS-displacement and the ventricular

Fig.2. The theoretical relationships between RVP/LVP and the indices of IVS-displacement (EI, NSC, LVSCI) during systole. Each index correlates approximately linearly with RVP/LVP (EI: eccentricity index, NSC: normalized septal curvature, LVSCI: left ventricular systolic circular index).

pressures (RVP, LVP), we tried to use some indices of IVS-displacement, EI, LVSCI and NSC in equation (7). These are expressed below.

EI is defined as $a/b \dots \dots \dots (8)^{2,5}$

NSC is defined as $ri/R_3 \dots \dots \dots (9)^1$

LVSCI is defined as $4\pi \text{ Area} / \text{LVperimeter}^2 \times 100 \dots \dots (10)$

Where ri is the ideal septal curvature ($ri = \sqrt{\text{Area}/\pi}$)! Area is the area of left ventricular latitudinal plane.³

In Fig. 1-B,

$$\text{Area} = [R_3^2 \Theta / 2] + [R_1^2 \pi / 2] - [R_1 \times (R_1 + R_3 - b)] \dots \dots (11)$$

$$\text{LV perimeter} = R_3 \Theta + R_1 \pi \dots \dots \dots (12)$$

$$\Theta = 2\sin^{-1}(R_3/R_1) \dots \dots \dots (13)$$

Where Θ is the central angle of IVS.

We can represent the direct relationship between the IVS-displacement and RVP/LVP ratio (RVP/LVP) by using those equations theoretically. We draw the relational curves between the RVP/LVP ratio (RVP/LVP) and those indices by these formulas (Fig. 2).

The relational formula between EI and RVP/LVP is relatively simple:

$$\text{RVP/LVP} = \frac{2(EI-1)^2}{(EI^2-2EI+2)} \dots \dots (14)$$

With regard to NSC and LVSCI, the relational equations with RVP/LVP are so complex that these relational curves were drawn by a computer using equations (8)–(13).

As shown in Fig. 2, these relational curves indicate that the relationships between those indices and RVP/LVP are almost linear theoretically. These results are consistent with some clinical reports.

In equation (14), both EI and RVP/LVP are instantaneous values. Though some investigators have been reported that the end-systolic EI relates RVPSP², the instantaneous relationship between EI and RVP has not been reported yet. Therefore we attempted to analyze the instantaneous relationship between them experimentally to confirm our theory in the subsequent section.

II. Experimental acute RVPO in dogs

1) Preparation

Eight adult mongrel dogs weighing 8.8–26.4 (mean 12.4) Kg were anesthetized with pentobarbital sodium (25 mg/Kg iv) and ventilated with a Harvard respirator, type 613 (tidal volume 30 ml/Kg, 15–20/min with O₂ as needed). The heart was widely exposed in the fifth intercostal space with a trans-sternal thoracotomy.

2) Hemodynamics and Dimensional Measurements

Two solid-state pressure transducers (Millar Co. PC-350) were inserted into the right and left ventricular cavities through apical incisions. An electromagnetic flow probe (NIHON KODEN, MF-1200) was placed around the pulmonary artery trunk to measure cardiac output. Two pairs of hemispheric sonomicrometer crystals were attached to the endocardial surface of the left ventricle using the previously described techniques.⁷ The two pairs of implanted ultrasonic crystals were arranged in the maximum transverse sectional plane of the left ventricle to measure the anterior-posterior dimension (LVAPD) and the septal-to-lateral wall dimension (LVSLD) (Fig. 1-A). The pericardium was preserved as carefully as possible.

3) Experimental Procedure and Data Acquisition

To produce acute RVPO by pulmonary

Fig.3. A representative recording from a dog undergoing repetitive pulmonary microembolizations: (1) control, (2) maximum RVPSP, (3) shock stage.

microembolizations, 0.1 to 1.0 ml of saline suspension of lycopodium spores (0.05 mg/ml) was repeatedly injected through a catheter into the inferior vena cava. Hemodynamics and ventricular dimensions were continuously recorded on a multichannel recorder (NIHON KODEN, RM-85), at a paper speed of 25 mm/sec or 100 mm/sec as needed. High speed recordings were made at the control stage, at each step of 10 mmHg increment of RVPSP and at the shock stage (LVPSP below 70 mmHg) during endexpiratory states with the respirator stopped. At each increment of RVPSP, over 3 min observation was allowed for the stabilization of hemodynamic variables.

4) Data Analysis

To analyze the instantaneous relationship between EI and RVP/LVP, we selected 4 phases of systole, the beginning of ejection (EJ), the end-systole (ES), the point of one-third of systole (1/3S) and that of two-third

of systole (2/3S), and then calculated EI(LVAPD/LVSLD) and RVP/LVP at each phase. EJ and ES were determined as the corresponding points to the peak of dp/dt and 20 msec prior to the peak of negative dp/dt. 1/3S and 2/3S were determined by dividing the interval between EJ and ES into three. EI is usually used as a clinical index that is measured at end-systole and end-diastole by two-dimensional echocardiography. Therefore in the subsequent section, we will use E to represent the instantaneous value of EI, and EI for that which is limited only to end-systole.

5) Statistical Analysis

To compare E with each of the RVP/LVP ratio, RVPSP, and RVPSP/LVPSP, linear regression analysis was used. Differences between either stages or phases were determined by paired Student's *t* tests. A value of $p < 0.05$ was regarded as statistically significant.

Fig. 4. The hemodynamic variables and the left ventricular dimensional changes with the advance of RVPO. (C: control, M: maximum RVPSP, S: shock stage). N=8.

RESULTS

Figure 3 shows a representative recording from a dog undergoing repetitive pulmonary microembolizations: (1) control, (2) maximum RVPSP, and (3) shock stage. At the maximum RVPSP, the right ventricular pressure elevated with the shift of the peak RVP from the early to the late phase of systole as compared with the control. Although the LVAPD changed only minimally, the LVSLD apparently decreased, which indicated leftward displacement of the IVS. In the shock stage, the RVPSP decreased with the LVPSP fall, and the waveform of the LVSLD showed asynchrony with that of the LVAPD.

1) Hemodynamics

Figure 4a shows the hemodynamic vari-

ables in control and RVPO including the shock stage. Each point on the graph represents a mean value after each 10 mmHg increment in RVPSP. RVPSP increased progressively from a mean value of 28 mmHg to 72 mmHg in response to repeated microembolizations. Conversely, stroke volume decreased gradually to a level of 60% of the control at the maximum RVPSP. LVPSP fell only slightly, the change being without statistical significance, from the control stage until RVPSP reached a level of 60 mmHg and then began to fall significantly ($p < 0.01$). Additional embolizations following the maximum RVPSP caused cardiovascular collapse (shock), in which LVPSP fell to a level of 62 mmHg with a significant fall in RVPSP and cardiac output. Left ventricular end-diastolic pressure

Fig.5. The change in RVP/LVP during systole at each level of RVPSP. Each point is the mean value of eight dogs, and the brackets indicate standard errors.

Fig.6. The change in E throughout the cardiac cycle at each level of RVPSP. Each point is the mean value of eight dogs.

(LVEDP) decreased continuously, whereas right ventricular end-diastolic pressure (RVEDP) increased slightly but steadily and the difference between LVEDP and RVEDP became more apparent after the maximum RVPSP was achieved (from the control to the shock stage, RVEDP: 3.7 to 6.7 mmHg,

LVEDP: 4.7 to 2.7 mmHg, both $p < 0.01$). heart rate did not change significantly throughout the course of RVPO including the shock stage.

2) Dimensional Changes

Figure 4b shows the effect of RV-afterload increase on LVAPD and LVSLD at end-diastole and end-systole. LVAPD showed little change due to recurrent microembolizations, either at end-systole or end-diastole. On the other hand, LVSLD decreased gradually throughout the cardiac cycle. At a RVPSP level of 60 mmHg, LVSLD decreased by 23% at end-diastole, and by 25% at end-systole from the control value ($p < 0.01$). In the shock stage, despite the sudden fall in RVPSP, the decrease in LVSLD was accelerated, particularly at end-diastole.

3) The Change in RVP/LVP During Systole

In response to the progression of RVPO, the peak of RVP shifted from early to late systole. As a result, the RVP/LVP ratio became greater during late systole compared with early systole after RVPSP exceeded 50 mmHg ($p < 0.01$). (Fig. 5)

4) The Change in E throughout the Cardiac Cycle

In response to the progression of RV-afterload, E increased throughout the car-

Fig.7. The correlation between the instantaneous RVP/LVP and E at each phase of systole. Each point is the mean value of eight dogs, and the brackets indicate standard errors.

Fig.8. The relationships of EI with RVPSP and RVPSP/LVPSP. Each point is the mean value of eight dogs, and the brackets indicate the standard errors.

diac cycle. During systole, after RVPSP exceeded 60 mmHg, the increment in E was more remarkable during late-systole than during early-systole ($p < 0.05$). Except during shock, the end-diastolic E increased in conjunction with the increment in the end-systolic value, and there were no significant differences between the end-diastolic and the

end-systolic E. At the shock stage, the end-diastolic E showed a remarkable increment in comparison with the end-systolic E ($p < 0.01$), and the values of E at early systole (EJ, 1/3S) were greater than those at late systole (2/3S, ES) ($p < 0.01$). (Fig. 6)

5) Correlation between the Instantaneous RVP/LVP and E at each Phase of Systole

Figure 7 shows the correlations between RVP/LVP and E at the different phases in systole. Each point on the individual lines represents a mean value of RVP/LVP against simultaneous E at each RVPSP level. At the late systole (2/3S, ES), there were significant linear correlations between E and RVP/LVP, which were maintained even in the shock stage. As regards the early systole, there were also significant linear correlations between RVP/LVP and E (EJ, 1/3S), except during the shock stage. The four regression lines were similar to each other, except for shock, in which the values of early-systolic E were greater than the predicted ones from the regression lines ($p < 0.01$). At each phase, the regression formulas were as follows.

EJ: $E = 1.118 + 0.265 \times (RVP/LVP)$.
 ($r = 0.64$, $p < 0.01$, except during shock)
 1/3S: $E = 1.124 + 0.264 \times (RVP/LVP)$.
 ($r = 0.65$, $p < 0.01$, except during shock)
 2/3S: $E = 1.126 + 0.263 \times (RVP/LVP)$.
 ($r = 0.68$, $p < 0.01$)
 ES: $S = 1.127 + 0.268 \times (RVP/LVP)$.
 ($r = 0.69$, $p < 0.01$)

6) Relationship between RVPSP and EI

Figure 8 shows mean values of RVPSP against EI at each RVPSP level. Except during shock, there was a linear correlation of relatively low significance between RVPSP and EI.

$EI = 1.11 + 0.0029 \times RVPSP$ (mmHg), ($r = 0.56$, $p < 0.01$, excluding Shock.). In the shock stage, despite the significant decrease in RVPSP, EI increased further.

7) Relationship between RVPSP/LVPSP, the end-systolic RVP/LVP and EI

The RVPSP/LVPSP ratio showed a good correlation with the end-systolic RVP/LVP ratio, as follows.

$RVESP/LVESP = 0.058 + 0.85 \times (RVPSP/LVPSP)$ ($r = 0.87$, $p < 0.01$)

Where RVESP is right ventricular endsystolic pressure, LVESP is left ventricular endsystolic pressure.

Therefore, it was clear that there was a significant linear correlation between RVPSP/LVPSP and EI, which was not lost even in the shock stage.

$EI = 1.124 + 0.259 \times RVPSP/LVPSP$ ($r = 0.68$, $p < 0.01$)

DISCUSSION

The IVS has been reported to be displaced from its normal position during diastole according to the interventricular transseptal pressure gradient (TSP) under various conditions.^{8,9} Kingma et al assumed that the IVS behaves essentially as a membrane between two fluid-filled chambers and showed that there was a tight linear relationship between the end-diastolic TSP and the corresponding end-diastolic LVSLD in the experimental study.⁹ Recently in patients with RVPO, the leftward displacement of the IVS has been documented not only during diastole, but also systole!¹⁻³ However, it is difficult to explain the systolic IVS-displacement only by the same mechanisms as Kingma's proposal on diastole. During systole the LV size varies markedly and the LV wall becomes much stiffer and thicker than during diastole, so the IVS could not act as a simple membrane.

Previously the LV has been modeled in many reports by using simple geometrical figures to calculate the LV-wall tension!¹² Although the thin-walled simple ellipsoidal model has been used as one of the intact left ventricular geometrical models, it is not applicable to the deformed LV with RVP. Olsen et al⁶ conceived a modified ellipsoidal model in RVPO, and assumed that the LV deforms according to the ventricular pressures (RVP, LVP) and the wall tensions. Their model is based on Laplace's law (very thin shell theory). The very thin shell theory neglects the finite thickness, the shear and the bending moments of the wall, and the result is not quantitative. However, Hood et al reported that the overestimation of average stress due to neglecting the finite wall thickness was approximately 10%.¹³ Mirsky also indicated that the effects of shear and bending moments are negligible except near the apex of the ventricle!¹⁴ We therefore think that this model is worthy enough to discuss the qualitative relation between biventricular pressures and IVS-displacement.

We attempted to clarify the role of ventricular pressures in IVS-displacement using this model, in which the LV geometry is a complicated function of the ventricular pressures and the wall stresses, and the direct relationship between the IVS-displacement

ment and the ventricular pressures is uncertain. We found that it was possible to cancel the wall tension in the equations on the assumption that the ratio of the wall tensions (IVS-wall tension/LV-FW tension) was constant irrespective of the state of RVPO.

In acute RVPO experiments, it was reported that the LVS behaved like the LV-free wall, and the wall tension of the IVS was similar to that of the LV-FW,¹⁵ as calculated using Olsen's model.⁶ Therefore, we presupposed that the IVS generates the same tension as that of the LV-FW, and derived the relational equation that indicated the direct relationship between the IVS-displacement and the ventricular pressures.

With regard to the indices for the assessment of IVS-displacement, EI, NSC and LVSCI were first proposed clinically, and they are dimensionless indices conceived to express the degree of LV deformity independent of the LV size.¹⁻³ Though they were previously reported to correlate with RVPSP or RVPSP/LVPSP in RVPO, the mechanisms have not been clarified mathematically. As shown in Fig. 2, our equations indicated that these indices might correlate with RVP/LVP linearly in such conditions where the IVS-wall tension maintained a constant relationship with the LV-FW tension.

We undertook the acute RVPO experiments to confirm the theoretical result in anesthetized open-chest dogs, preserving the pericardium. Preservation of the pericardium was desirable because its loss resulted in an apparent change in the basic cardiac configuration.

We produced pulmonary hypertension and shock by repeated pulmonary embolizations with lycopodium spores. Regarding pressure overload on the RV, microembolization,¹⁶ constriction⁶ or balloon occlusion¹⁷ of the pulmonary artery (PA) have been reported. In the preliminary study, we tried to produce RVPO either by microembolization or by PA constriction. There was some obvious difference in the RVP waveform between the two procedures. In microembolism, the peak of RVP wave shifted to the late phase of systole. However, the main PA constriction resulted in an RVPSP increment with a mid-systolic peak of RVP, and hence the end-systolic RVP was disproportionately low. Clinically it has also been reported that

a similar difference exists in the RV waveforms observed in cases of pulmonary hypertension and RV outlet stenosis, such as PA stenosis.¹⁸ Therefore, we used experimental microembolism as the model of clinical RVPO excluding RV outlet stenosis.

In the present study, we selected four points in systole (EJ, 1/3S, 2/3S, ES) to discuss the simultaneous relationship between E and RVP/LVP in acute RVPO. Though it was reported that the degree of IVS displacement varied according to the phase of systole, there has been no report on the systolic simultaneous relationship between the ventricular pressures and the IVS-displacement in RVPO.

With the RVPSP elevation following repeated microembolization, the waveform of RVP gradually changed as mentioned above, while that of LVP changed little. As a result, RVP/LVP at late systole became significantly greater than that at early systole in the advanced stage of RVPO. In this condition, E increased throughout the cardiac cycle, and the size of the increment at late-systole was significantly larger than that at early systole (Fig. 6).

As shown in Fig. 7, the relationships between the RVP/LVP ratio and E were significantly linear at each phase throughout all stages except for early systole in the shock stage, and the four regression lines from each phase very similar to one another. This result indicated that the relationship between RVP/LVP and E was not influenced by the changes in LV size and wall tension throughout the systolic phase, and was compatible with the theoretical conclusion.

In the shock stage, E correlated with the simultaneous RVP/LVP only at the two points of late-systole, but not at those in early-systole. Though the early-systolic changes in E could not be interpreted by our theory alone, it might be explained by reference to the influence of end-diastolic LV-deformity. In this stage, E increase of end-diastolic TSP (LVP-RVP). Usually the LV wall tension is much greater during systole than during diastole, and it was considered that the systolic E could correspond to the instantaneous RVP/LVP and might not be influenced by the end-diastolic LV-configuration. However, in the shock stage, the systolic wall tension of the LV was regarded

as very small. In this condition, the effect of the end-diastolic LV-deformity might not be negligible, especially at early-systole. It rather strongly supports our theory that the late-systolic relationship between E and RVP/LVP is maintained even in the shock stage despite the remarkable decrease in systolic LVP.

It is important to predict RVPSP for the assessment of RVPO. Some clinical investigators reported that the index of IVS-displacement was related to absolute RVPSP.² In the present animal preparations, except for the shock stage, EI increased linearly with RVPSP elevation. In the shock stage, despite the decreased RVPSP, EI increased with the elevation in RVESP/LVESP due to the fall in LVP. The dissociation of RVESP/LVESP from RVPSP enables us to interpret the dissociation of EI from RVPSP. Even if the shock stage was excluded, the coefficient of correlation between RVPSP and end-systolic E (EI) was less than that between the simultaneous RVP/LVP ratio (RVESP/LVESP ratio) and EI, these results indicating that RVPSP could be estimated approximately by using EI only, while LVP was kept within normal range.

RVPSP/LVPSP is more useful than absolute RVPSP for estimating the degree of RVPO. In clinical RVPO, the relationships between the end-systolic IVS-displacement and RVPSP/LVPSP were also reported. Our theory and experimental results revealed the instantaneous relationship between the index of IVS-displacement and the simultaneous RVP/LVP, and, as RVPSP/LVPSP correlates with RVESP/LVESP significantly, showed indirectly the relationship between EI and RVPSP/LVPSP. Therefore, the coefficient of correlation between EI and RVPSP/LVPSP was as significant as that between EI and RVESP/LVESP during RVPO, including the shock stage. Now it is possible to predict RVPSP from RVPSP/LVPSP by using EI, because the systemic blood pressure can substitute for the value of LVPSP.

In this report, we demonstrated the relationship between the ventricular pressures and the IVS-displacement in acute RVPO. With regard to chronic RVPO, there is an apparent difference between the basic cardiac geometry of this condition and that of

acute RVPO. It has been reported that the heart deformed organically with right ventricular hypertrophy and dilatation, and that the LV tended to deform to crescent shape resembling the RV.¹⁹ In this type of heart, the distribution of the cardiac wall tension might be different. Furthermore, it was reported that there was an apparent difference in the degree of the IVS-displacement against the similar RVPSP between acute and chronic RVPO.¹⁹ The pathophysiology of the IVS-displacement in chronic RVPO should be clarified by further studies.

In recent years much progress has been made toward the development of mathematical models for describing large deformations of the heart more quantitatively, for example, a finite deformation theory.²¹ Those approaches may enable one to analyze the behavior of cardiac movement more precisely. However those theories are too complex to elucidate the role of IVS-displacement intuitively and clinically.

We assumed that the IVS generates a tension equal to that in the left ventricular free wall during systole in acute RVPO, and demonstrated the role of RVP and LVP in the IVS-displacement both theoretically and experimentally. Our model is simple and the result is qualitative. But this model indicates that the IVS must shift leftward to maintain the balance of the wall tensions between the IVS and the LV free wall, and some indices of IVS-displacement correlate with RVP/LVP, independent of left ventricular size and the absolute wall tension. The present concept is plain and helps in the understanding of the role of RVP and LVP in IVS-displacement. We consider that this is one of the theoretical bases for the prediction of RVPSP by using the IVS-displacement indices.

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