Behavior of the Right Ventricle Against Pressure Loading: On its Plasticity

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The difference between the right (RV) and the left ventricle during progressive pressure loading is that the RV changes to cylindrical form without change in its enddiastolic pressure (EDP). We investigated this particular phase experimentally and clinically.

In experimental animal studies (dogs), the gradual constriction of the main pulmonary artery (PA) resulted in an elevation of RV systolic pressure (RVSP) without changes in RVEDP and PA flow (FPA). This was followed by an increase in RVEDP and a decrease in FPA. In this initial phase, the RV increased in its dimension from the free wall to the septum.

In 155 clinical cases with RV pressure loading, the relationship between RVSP and RVEDP suggested that the RV compliance of the cases with chronic RV pressure loading was less than that with subacute loading. However, these studies did not supply the expected data on the phase we mentioned. In 2 patients with primary pulmonary hypertension who showed spontaneous remission during our observation, a decrease in RVSP without change in RVEDP occurred with a change in RV form detected by 2-dimensional echocardiography. These data suggested the existence of a phase in which the RV showed a character of plasticity during pressure loading, but direct documentation was not successful.

The right ventricle has the anatomical characteristics of a volume pump and its hemodynamic response to pressure loading might be different from that of the left ventricle (LV) which acts as a pressure pump. The experiments performed in an experimental dog revealed that gradual pulmonary constriction resulted in an increase in RV systolic pressure without change in end-diastolic pressure (RVEDP) followed later by a decrease in pulmonary blood flow (FPA) with an increase in RVEDP.3

As Sarnoff et al. pointed out in their classical work on Frank-Starling mechanism, the homeometric autoregulation of the LV did not take place against an afterloading when an increase in aortic resistance was not abrupt.

The existence of the phase in which RV systolic pressure increases against an increase in PA resistance without changes in RVEDP and FPA might be a distinctive character of the RV performance which differs from the LV. It is assumed that in this particular phase, the RV changes its form to a more cylindrical shape and the distribution of the RV segmental wall tension becomes more homogenous. Therefore, an increase in its diastolic volume would occur without change in its diastolic pressure.

METHODS

Animal Experiments
Six mongrel dogs anesthetized with intravenous Nembutal of 25 to 30 mg/kg of body weight were used in this open chest study.
Fig. 1. Schematic illustration of the preparation for documentation of a phase in which RVSP increases without change in RVEDP during PA constriction.

Fig. 2. Schematic illustration of the preparation to reveal a change in RVD during constriction of the PA.

Fig. 3. Measurement of the index of RV deformity through left ventricular minor axis. IVS = interventricular septum; FW = left ventricular free wall

Under the artificial respiration with a mixture of room air and 1 L/min of oxygen by Harvard respiratory pump, a bilateral thoracotomy was performed at 5th intercostal space. A small incision of the pericardium was made to insert a large bore tubing connected to the reservoir into the right atrial appendage, and to apply an electromagnetic flow probe around the root of the pulmonary artery. Distal to the flow probe, a snare was placed around the main PA for gradual pulmonary constriction. Pulmonary and RV pressures (PPA, PRV) were measured through catheters inserted at the peripheral vein or punctured at the PA and RV (Fig. 1).

Setting the position of the reservoir to maintain right atrial (RA) pressure, the PA was gradually constricted and changes in FPA and PRV were recorded.

In two additional dogs, we measured the right ventricular dimension from the free wall to the septum (DRV) by an ultrasonic dimension meter during increasing pulmonary constriction (Fig. 2).

Clinical Observation

We reviewed 174 hemodynamic data taken from 155 patients with right ventricular pressure loading who underwent cardiac catheterization at Keio University Hospital. Basic diseases of these patients were mitral stenosis which was taken as a cause of chronic pulmonary pressure loading, primary pulmonary hypertension and recurrent pulmonary embolism as causes of subacute RV pressure loading. Ninety three two-dimensional echocardiographic data were obtained from 91 out of 155 catheterized patients. As a two-dimensional echocardiographic indicator of RV pressure loading, the ratio of the anteroposterior internal diameter and septum-lateral internal diameter in end-diastole obtained from the LV short axis view at the level of the chordae tendineae (DAp/Dsl) was used5 (Fig. 3).
RESULTS

Animal Experiments

In our preparation, we could keep RA pressure constant by the reservoir during gradual constriction of the main PA. As shown in Fig. 4, by constriction of the PA, PPA was increased without change in RVEDP and FPA followed later by a decrease in FPA. Closing the tubing of the reservoir connected to the RA, the constriction of the main PA resulted in a slight increase in PPA without changes in FPA and RVEDP followed by a marked decrease in FPA and distinct increase in PPA and RVEDP (Fig. 5). In this experiment, a change in FPA was assumed from a change in aortic blood flow (FAO). In the additional experiments, this increase in systolic PRV without change in FAO and RVEDP were associated with an increase in DRV (Fig. 6).

Clinical Observation

The relationship between RV systolic pressure (RVSP) and RVEDP was depicted in Fig. 7. As a whole, there was a statistically significant relationship between them \(r = 0.56\). In the cases with mitral stenosis, this regression line was less steep than that of the cases with subacute RV pressure loading. But a phase of increasing RVSP without change in RVEDP was not observed in these relationship.

The relation between \(D_{AP}/D_{SL}\) and RVEDP was also linear and the difference of this relationship between the cases with subacute and chronic RV pressure loading could not be found (Fig. 8).

DISCUSSION

In the animal experiments, the data suggested that the RV could increase in its systolic pressure without change in RVEDP but with change in its end-diastolic geometry within some range of RVEDP. This phenomenon may be explained by the compliant quality of the RV, however, if a minimal change in RVEDP could be detected
in our experiments. The term “compliance” of the whole ventricular chamber has been used to refer to the ratio dV/dP. In this meaning, the RV compliance can be said extremely high in the phase we mentioned. In this particular phase, RV is assumed to change to a more cylindrical form. Unfortunately, in our clinical observation, a curvilinear relationship was not established between the index of RV deformity and RVEDP to support the observations in the animal experiments. In this phase, the distribution of RV wall tension might be non-homogenous and the concept of compliance defined in the field of physics may not be appropriate. A more applicable factor may be that of plasticity.

There are many variables which determine the deformity of the RV. Pressure difference between the LV and RV, the stiffness of the ventricular muscle and the contributers of change in preload to each ventricle might affect its deformity. Individual factors did not reveal a close relation to the index of the RV deformity, e.g., the relationship between LV-RV pressure difference in end-diastole and $D_{AP}/D_{SL}$ was not significant (Fig. 9).

The duration of RV loading is also considered to be one of the factors which determine the RV deformity, although we could not demonstrate that relationship. The difference of the steepness of the regression lines in the RVEDP-RVSP relationship between the cases with subacute and chronic RV pressure loading

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**Fig. 6.** Effect of PA constriction on the right ventricular dimension from the free wall to the septum (DRV). PA constriction resulted in an end-diastolic prolongation of DRV and an increase in RVSP without change in RVEDP.

**Fig. 7.** Relationship between RVEDP and RVSP in the cases with RV pressure loading.
Fig.8. Relationship between RVEDP and the index of RV deformity (DAP/DsL) in end-diastole.

Fig.9. Relationship between LV-RV pressure difference and DAP/DsL in end-diastole.

(Fig. 7) would, at least in part, be affected by the stiffness of the RV. A curve formed at plotting systolic ventricular pressure against their EDP has been called a pressure-function curve of the ventricle. It is known that ventricular function curve constructed by using EDP is shifted with a change in ventricular compliance, i.e., the more compliant the steeper. Here again, the confusion between compliance and plasticity of the RV could not be clarified from our clinical data.

In our clinical experiences, RV plasticity was seen in two cases of primary pulmonary hypertension that showed spontaneous remission of pulmonary hypertension. Their RVSP decreased without change in RVEDP while an index of the RV deformity changed (Fig. 10), as we reported previously.

REFERENCES
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