Journal of Cardiography 15: 1215-1224, 1985

Two-dimensional echocardiographic evaluation of right ventricular function during left heart bypass

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Summary

Right ventricular (RV) function in terms of hemodynamics and RV wall motion was studied in 14 mongrel dogs during left heart bypass (LHB) using a centrifugal blood pump. The wall motion was analyzed by two-dimensional echocardiography (2D-echo). Incremental changes in LHB flow ratios of 0% (controls), 25%, 50%, 75% and a maximum 85–100% were accompanied by decrements of segmental shortening of the interventricular septum (IVS) by 54±12%, 43±5%, 42±2%, 35±0% and 0%, respectively. In addition to akinesis of the IVS during maximum flow, a specific part of the RV free wall adjacent to the IVS also had marked depression of contractions and overall RV contraction was nearly dependent on the RV free wall opposite to the IVS. Maximum LHB flow induced complete depression of the left ventricular cavity, a marked increase in RV volume, and depression of the RV ejection fraction on 2D-echo. Excessive or prolonged LHB reduces the RV wall motion capability and may lead to right heart failure. Our results suggest that an LHB ratio of about 75% is optimum to maintain normal cardiac function, particularly that of the right heart.

Key words

Right ventricular function | Right ventricular wall motion | Interventricular septal wall motion | Two-dimensional echocardiography | Left heart bypass

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Received for publication April 30, 1985; accepted June 13, 1985 (Ref. No. 30–56)

Introduction

The left ventricular assist device (LVAD) has been used for mechanical circulatory assistance in marked refractory heart failure after cardiac surgery. However, right ventricular (RV) failure is a recognized complication or natural consequence of mechanical assistance to the left ventricle (LV). Intensive pharmacologic therapy (i.e. catecholamine) or supplemental mechanical support of the RV is often required, either in the form of a biventricular bypass or total cardiopulmonary bypass with extracorporeal membrane oxygenation[1~4].

Functional interaction between the ventricles has been evaluated by many researchers using various experimental models[5~7]. This study characterized RV functional changes during highly effective mechanical assistance to the LV as evaluated by two-dimensional echocardiography.

Material and methods

Left atrial (LA)–descending aortic (Ao) bypass was performed in fourteen mongrel dogs (22±4 kg) using a bio-pump (centrifugal pump). The animals were anesthetized with 20 mg per kg sodium pentobarbital intravenously followed by a constant infusion at 3 mg per kg per hour, and ventilation with a respirator to achieve an arterial Po_2 greater than 100 torr and Pco_2 of 35 to 45 torr. Acidosis was corrected and positive end-expiratory pressure (PEEP) of 5–8 cm H_2O was applied to prevent atelectasis. The heart was exposed via a thoracotomy through the left fifth intercostal space. Right atrial pressure (RAP), right ventricular pressure (RVP), left atrial pressure (LAP), left ventricular pressure (LVP) and descending aortic pressure (AoP) were determined by cannulation of the RA, RV, LA, LV and Ao. The right cardiac output and main pulmonary arterial pressure were measured using a Swan-Ganz catheter. The ascending aortic and left heart bypass (LHB) flows were measured using a popular electromagnetic flow probe (Nihon-Koden Inc. Nagoya, Japan). A heat exchanger was used on the LA-Ao bypass circuit inflow side to maintain the blood temperature between 37°C–38°C. Appropriate cannulations and connections were made to form the LA-Ao bypass circuit. A cannula, made by 36Fr. Sarns Inc. Ann Arbor, Michigan, was inserted into the

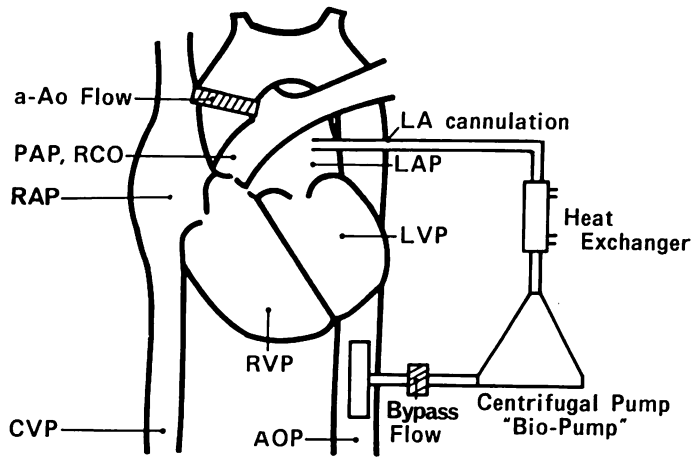

Fig. 1. Experimental setup of left ventricular assist device (LVAD).
a-Ao Flow: ascending aortic flow; LAP: left atrial pressure; RCO: right cardiac output; LVP: left ventricular pressure; PAP: pulmonary arterial pressure; RVP: right ventricular pressure; RAP: right atrial pressure; AOP: aortic pressure; CVP: central venous pressure.

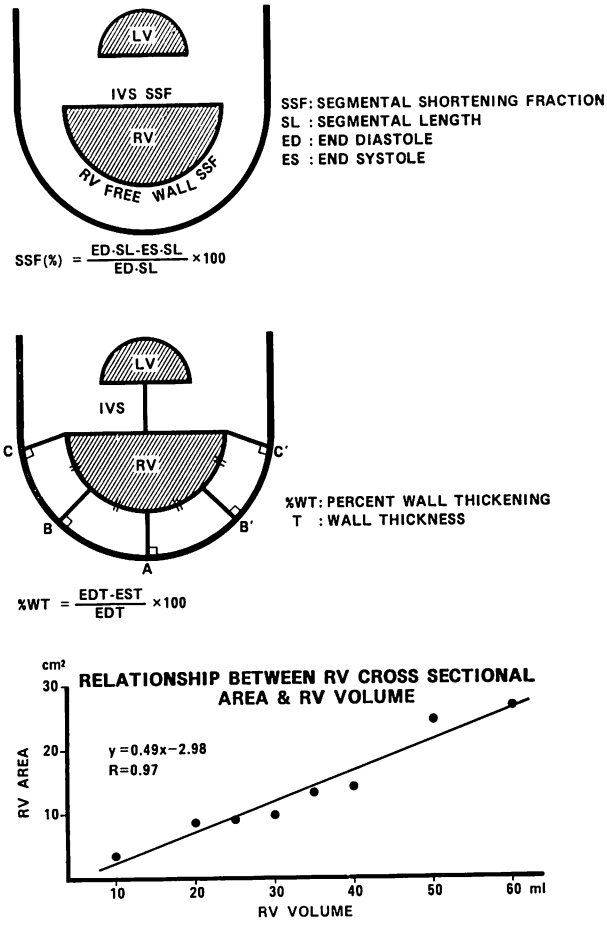

Fig. 2. Measurement method of SSF and %WT based on echocardiography.

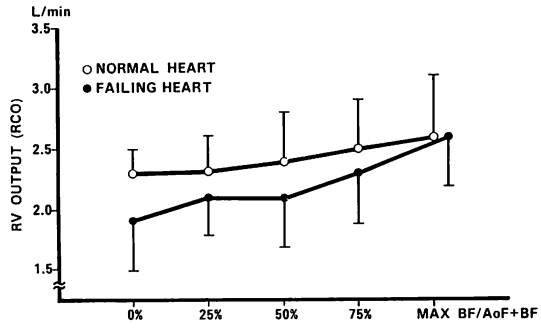

Fig. 3. Changes of right heart output at each bypass flow rate.

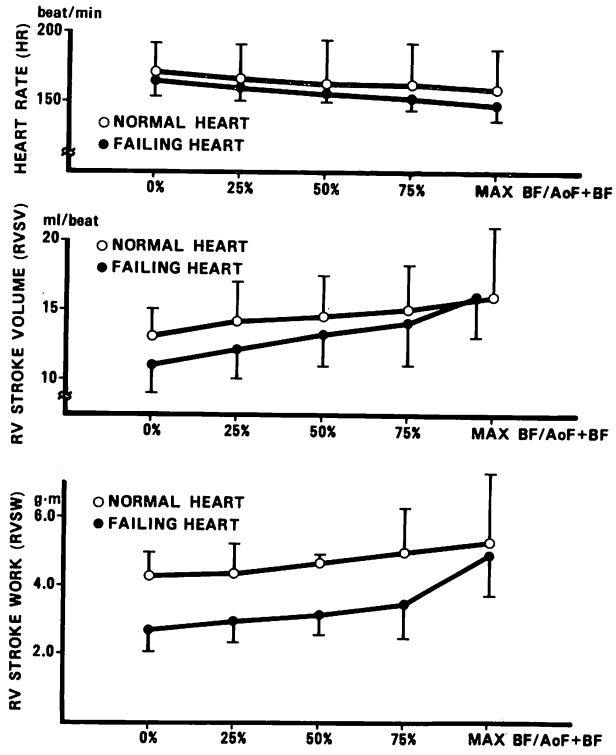

Fig. 4. Changes in heart rate, RV stroke volume and RV stroke work at each bypass flow rate.

left atrium, and an improvised T-type tube (3/8 inch) was inserted in the descending aorta (**Fig. 1**). Anticoagulation during the LHB was achieved with heparin 1 mg per kg. The LHB flow ratio (LHB flow×100/LHB flow+ascending aortic flow) was changed in a stepwise fashion from zero to maximal LHB to achieve total LV decompression. Stabilization periods of 15 min were allowed at each of the LHB flow ratios of 25%, 50%, 75% and a maximum flow (about 85~100%) to facilitate making measurements. Fourteen mongrel dogs were categorized in two groups, of normal and failing hearts. The latter was induced by left coronary artery ligations. The RV functions were evaluated hemodynamically and by two-dimensional echocardiography (YHP Inc. Boston, Mass. & Tokyo, Japan). The infarcted area, produced by left coronary artery ligation and measured by Nitroblue tetrazolium (NBT) stain, accounts to 30~40% of all left ventricles. The echo probe was positioned on the surface of the posterolateral wall of the LV and a cross-sectional image of the RV was obtained at the tricuspid valve level. This approach provided good LV and RV short-oxis views with good reproducibility sufficient for the quantitative analysis. The segmental shortening fractions [SSF=(EDSL−ESSL)/EDSL] of the IVS, the RV free wall and the RV total SSF (IVS SSF+RV free wall SSF) were measured as shown in the first diagram in **Fig. 2**. The methods for calculating the thickness of the IVS and RV free wall are shown in the second diagram of **Fig. 2**. The IVS wall thickness was determined at its mid-point. The RV free wall was divided into four equal sections, and measurements were made along perpendicular lines originating from the RV internal surface to ex-

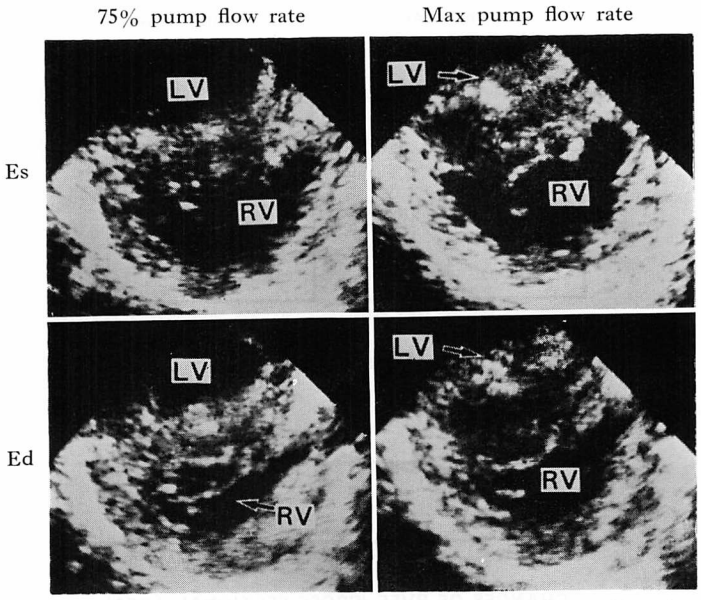

Fig. 5. Two-dimensional echocardiograms during left heart bypass at the 75% and the maximum bypass flow rate.
Ed=end-diastole; Es=end-systole.

ternal surface points denoted by A, B, B′, C and C′. Using the computer trace, percent syetolic wall thickening (%WT), defined as (EDT−EST)/EDT, changes in the RV area calculated from the end-diastolic to end-systolic areas (RVEDA−RVESA=⊿RV area), and the RV ejection fraction [RVEF=(RVEDA−RVESA)/RVEDA] were determined. The cross-sectional area of the RV determined echocardiographically correlated well with the RV volume of the isolated heart as shown in **Fig. 2** (Y=0.49X −2.98, R=0.97).

Results

The entire series of measurements was completed for each of the 14 dogs. The changes in the RV output in the LHB are shown in **Fig. 3**. As the LHB flow ratio increased, the RV output tended to increase. This was more obvious in the failing heart group than in the normal heart group.

The heart rate tended to decrease as the LHB flow ratio increased. The RV stroke volume and the RV stroke work increased with further increases in the LHB flow ratio, and became significantly higher than the initial control data when total decompression of the LV was achieved. These changes were more evident in the failing heart group than in the normal heart group (**Fig. 4**).

Fig. 5 shows changes in the LV and RV dimensions in the short-axis 2D-echocardiograms during LHB, when the LV was completely decompressed and the RV was obviously enlarged. At that time, the ascending aortic flow decreased to approximately zero, the aortic pressure patterns becamenearly pulseless, and by two-dimensional echocardiograms, the aortic and mitral valves remained in the closed position and no movement of the valve leaflets was evident.

The changes in the RV cross-sectional areas obtained by the M-mode echocardiograms are shown in **Fig. 6**. When the LHB flow ratio increased from 75% to the maximum, significant increases in both the RVEDA and the RVESA were observed ($p<0.002$). The increase in the RV area and the reduction in the RV ejection fraction were most marked when the LHB flow

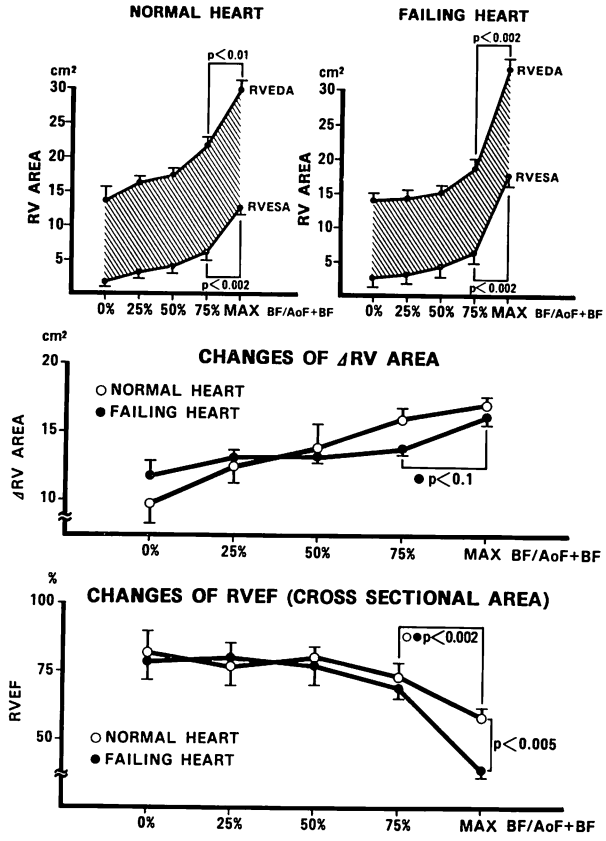

Fig. 6. Changes in RVEF at each bypass flow rate.

ratio increased from 75% to the maximum, with total decompression (**Fig. 6**). These findings were more marked in the failing heart group.

The SSF of the IVS decreased progressively as the LHB flow ratios were increased (**Fig. 7**). When the LHB flow ratio increased from 75% to the maximum of total LV decompression, the SSF of the IVS decreased significantly from 35±7% to 0%. There was also appreciable reduction in the SSF of the RV free wall during maximum bypass. The SSF of the RV free wall ranged from 44 to 49% during 75% bypass, and although it decreased to 25±2.5% and 15±1.1% during 100% bypass in the normal and failing hearts, respectively. As a result, the RV total shortening fraction decreased significantly ($p<0.005$) as the LHB flow ratios increased from 75% to the maximum (**Fig. 7**).

The %WT changes in the M-mode echocardiogram during the LHB are shown in **Fig. 8**. The %WT of the IVS in the normal and failing heart groups were 24~25% in the control state, 22~23% with a 75% LHB flow ratio, and 0~5% with the maximum LHB ratio ($p<0.005$). The %WT of the RV free wall in the failing heart group was different as shown in **Fig. 8**. The %WT of point A was 39±4% in the control state, 35±7% with a 75% LHB flow ratio, and 27±5% with a maximum LHB ratio. Those of points B and B′ were 36±5% in the control state, 34±9% with a 75% LHB ratio, and 22±4% with a maximum LHB ratio. Those of points C and C′ were 34.5±7% in the control state, and 40±9% with a 75% LHB ratio. Thus, the

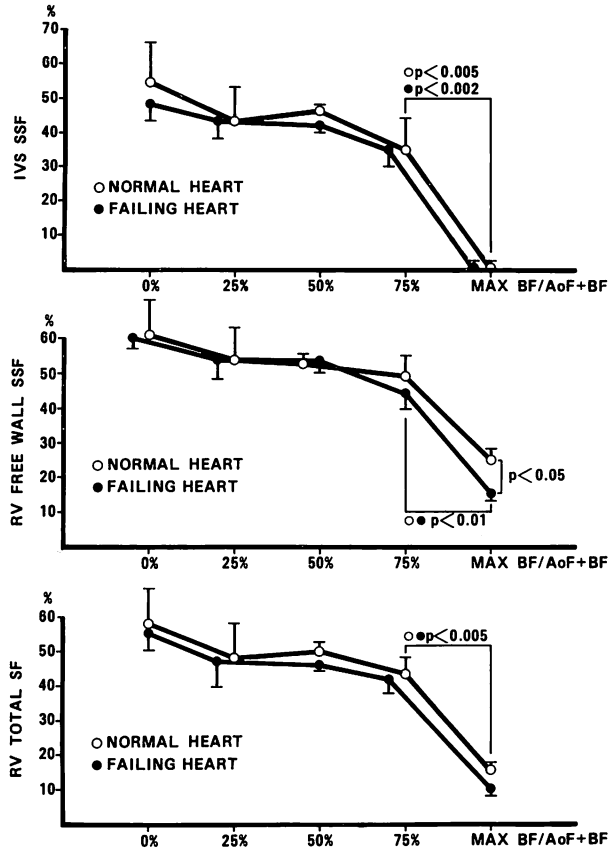

Fig. 7. Changes in SSF at each bypass flow rate.

RV free wall relatively near the IVS had less contractility during the LHB as compared to other parts of the free wall.

Discussion

There are some reports of RV function during the LHB, but the results are controversial. Pierce et al[8,9]. reported that RV failure occurred in six of 22 patients who had had LV assists after cardiac surgery. Minor deviations in RV function occur as a result of LV decompression associated with left ventricular assist device (LVAD). Pierce's observations suggest that the RV failure results primarily from RV muscle dysfunction. He also observed poor myocardial preservation or ischemia of the RV myocardium. Farrar et al[10]. advanced the ventricular interdependence hypothesis, in which RV function is dependent upon unassisted and undecompressed LV contraction. This situation is possible through forces which develop in the interventricular septum.

Rushmer[11] described four mechanisms for the contraction function of the RV, as follows: (1) papillary muscles in the RV contract with the tricuspid valve moving downward to the apex; (2) the curvature of the IVS increases due to LV myocardial contraction, and the IVS protrudes into the RV; (3) the RV free wall moves close to the IVS; and (4) the adherent portions of the RV shift to the LV side and contribute to the movement of the RV free wall close to the IVS when the LV contracts. These mechanisms (2), (3), (4) are related to the IVS, then,

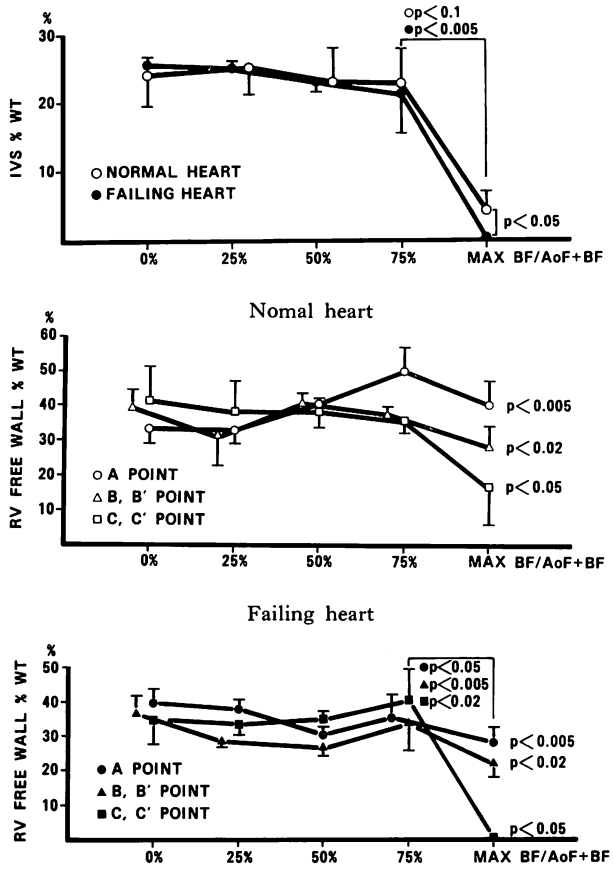

Fig. 8. Changes in %WT at each bypass flow rate.

the function will decrease whenever the IVS is injured. This shows the importance of the IVS to RV function.

The experimental results of Harada et al[12]. suggest that the detrimental effects on the RV function due to afterload reduction of the LV (intraaortic balloon pumping) in case of septal infarction depend on the dysfunction of the ventricular septum. Miyamoto et al[13~16]. reported that the maximum LHB ratio resulted in significantly depressed RV dp/dt and that the depressed RV contractility might have been caused by the markedly reduced IVS contractions which were observed by two-dimensional echo images[13,17,18].

In the present study, LV decompression and concomitant RV distension occurred with the start of the LHB observed, and these were more marked in the animals with the failing hearts. With increases in the LHB ratio, the heart rate decreased, the SV and RVSW increased, and the RVEDA and LVEDA/RVEDA increased by two-dimensional echocardiography. When the bypass ratio was increased from 75% to the maximum, RVEF on echocardiography decreased significantly, and the SSF and %WT of the IVS became motionless, with only the central portion of the RV remaining contractile. The suppressed LV wall movement during LV decompression which is mediated through the IVS to the RV free wall seems to restrict the movement of the wall near the IVS.

Excessive and prolonged LHB not only reduces the RV wall motion capability but may

lead to right heart failure. Our results suggest that LHB flow ratio of approximately 75% is optimum to maintain normal cardiac function, particularly that of the right heart.

断層心エコー図による人工心臓左心補助循環時の右心機能の検討

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雑種成犬(平均体重 22±4 kg) 14頭を用い，遠心ポンプ (bio-pump) にて左房－下行大動脈間バイパス(以下バイパス)を行った．バイパス率は全心拍出量の 25%，50%，75%，及び最大バイパス率(約85～100%の間)とし，正常心及び左冠状動脈前下行枝結紮により作成した左心不全に対し，各流量において，血行動態及び断層心エコー図所見による右室局所壁運動を中心に検討した．

バイパス開始とともに，左室の縮小と右室の拡大を認め，同時に心拍数低下，右室一回拍出量および拍出係数の増加が認められた．バイパス流量を 75% から最大バイパス率に変えると，心エコー図上，左室腔は完全に虚脱された状態となり，右室腔が著明に拡大するため，⊿RV area (RVED area－RVES area) は増加したが，右室駆出率は逆に低下した ($p<0.002$).

一方，心室中隔と右室自由壁の segmental shortening fraction (SSF) 及び心筋壁厚増加率の検討では，最大バイパス率時，心室中隔の SSF と壁厚増加率はほぼ 0% となり，心室中隔はほとんど収縮しなかった，また右室自由壁の SSF も著明な低下を認め，心室中隔に近い部位の右室自由壁の壁厚増加率はほぼ 0% となった．つまり最大バイパス流量においては右室駆出率の低下は著しく，それを右室局部壁運動でみると，心室中隔及びその付近の右室自由壁はほぼ akinesis の状態となっており，右室の収縮性は心室中隔より離れた右室自由壁にのみ依存した状態であり，ポンプ効率から見ると，右室は極めて低下した状態におかれているものと判断した．その原因として，最大バイパス流量時には左室は完全に虚脱状態で収縮性を失っており，この左室の非収縮性が，心室中隔を介して右室に悪影響を与えたものと考えられた．

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