

# Right ventricular stiffness constant as a predictor of postoperative hemodynamics in patients with hypoplastic right ventricle: a theoretical analysis

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**Abstract** One and a half ventricle repair (1.5VR) is a surgical option for hypoplastic right ventricle (RV). The benefits of this procedure compared to biventricular repair (2VR) or Fontan operation remain unsettled. To compare postoperative hemodynamics, we performed a theoretical analysis using a computational model based on lumped-parameter state-variable equations. We varied the RV stiffness constant ( $B_{RV}$ ) to simulate the various RV hypoplasia, and estimated hemodynamics for a given  $B_{RV}$ . With  $B_{RV} < 150\%$  of normal, cardiac output was the largest in 2VR. With  $B_{RV} > 150\%$ , cardiac output became larger in 1.5VR than in 2VR. With  $B_{RV} > 250\%$ , RV end-diastolic volume was almost the same between 1.5VR and 2VR, and a rapid increase in atrial pressure precluded the use of 1.5VR. These results indicate that the beneficial effect of 1.5VR depends on the RV stiffness constant. Determination of management strategy should not only be based on the morphologic parameters but also on the physiological properties of RV.

**Keywords** One and a half ventricle repair · Right ventricular stiffness · Hypoplastic right ventricle · Computational model

## Introduction

One and a half ventricle repair (1.5VR) is a surgical option for hypoplastic right ventricle (RV) caused by various congenital heart diseases including pulmonary atresia with intact ventricular septum (PA/IVS), Ebstein's anomaly or their relatives. In this procedure, the superior vena cava (SVC) is directly connected to the pulmonary artery (PA). Therefore, the blood from SVC directly enters PA, whereas the blood from the inferior vena cava (IVC) is pumped by RV to PA. This procedure is clinically acceptable because of its low surgical risk [1, 2]. However, the benefits of this procedure on postoperative hemodynamics in patients with a wide spectrum of RV hypoplasia compared to other procedures such as biventricular repair (2VR) and Fontan operation remain unsettled [3]. Furthermore, conversion to Fontan circulation was required late after 1.5VR in a possibly inappropriate candidate [4].

Although various authors reported an arbitrary selection scheme for the procedures based on RV morphology such as RV end-diastolic volume (RVEDV) [1, 2, 5], the long-term outcomes of 1.5VR have remained insufficiently known [5]. The previous criteria do not likely predict postoperative hemodynamics of these complex circulations accurately because morphological values measured preoperatively largely depend on the RV preload and afterload conditions, which change remarkably between subjects and between before and after the operation.

Hypoplastic RV is physiologically characterized by increased RV stiffness, caused by hypertrophy and

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fibroelastosis of RV muscles [6]. However, how RV stiffness influences the postoperative hemodynamics has not been reported. Given the small number of patients with each of the wide variety of preoperative RV conditions [7, 8], the influence of RV stiffness on 1.5VR, 2VR, and Fontan operation cannot be examined by clinical study. It is also difficult to experimentally reproduce hemodynamics before and after 1.5VR for hypoplastic RV with various stiffness. In view of the above, we attempted to clarify postoperative hemodynamics by a theoretical analysis using a computational model based on lumped-parameter state-variable equations. The present results indicate that the RV stiffness constant may provide selection criteria for 1.5VR.

## Materials and methods

The electrical analogs of the model used to simulate the cardiovascular system are shown in Fig. 1. We modeled the postoperative cardiovascular system mathematically by a combination of the time-varying elastance cardiac chamber model and the three-element Windkessel vascular model. We set the normal values of parameters to be appropriate for a 75-kg man. These values were obtained from the literature [9–13] and are listed in Table 1. Since

the data of the pressure–volume relationship of the atrium were scarcely available, parameters of the atrium were surmised from the literature [10–12].

## Heart

The right and left ventricular chambers as well as the atrial chambers are represented by the time-varying elastance model [9, 10, 13]. The end-systolic pressure–volume relationship is described by a linear formula:

$$P_{es,cc} = E_{es,cc} [V_{es,cc} - V_{0,cc}] \quad (1)$$

where  $P_{es,cc}$  and  $V_{es,cc}$  are end-systolic pressure and volume, respectively;  $E_{es,cc}$  is the maximal volume elastance;  $V_{0,cc}$  is the volume at which  $P_{es,cc}$  is equal to 0 mmHg. cc denotes each chamber, i.e., RA for the right atrium, LA for the left atrium, RV for the right ventricle, or LV for the left ventricle. The end-diastolic pressure–volume relationship is represented by a non-linear formula:

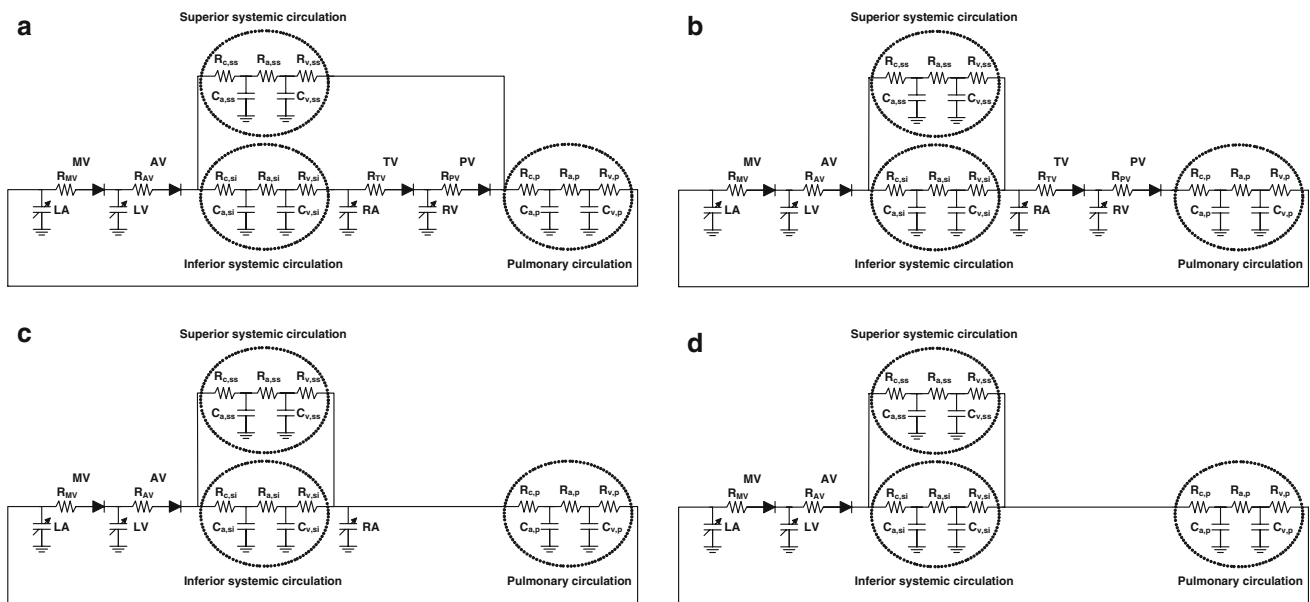
$$P_{ed,cc} = A_{cc} \left[ e^{B_{cc}(V_{ed,cc} - V_{0,cc})} - 1 \right] \quad (2)$$

where  $P_{ed,cc}$  and  $V_{ed,cc}$  are end-diastolic pressure and volume, respectively;  $A_{cc}$  and  $B_{cc}$  are constants [9, 10, 13]. We assumed the time course of the time-varying elastance by defining normalized elastance curve  $e_{cc}(t)$  as:

**Table 1** Parameters used in modeling

Heart rate (HR), beats/min	75			
Duration of cardiac cycle ( $T_c$ ), ms	800			
Time advance of atrial systole (DT), ms	16			
Total stressed blood volume ( $V_s$ ), ml	750 (control only)			
	LV	RV	LA	RA
Time to end systole ( $T_{es}$ ), ms	200	200	120	120
End-systolic elastance ( $E_{es}$ ), mmHg/ml	3.0	0.7	0.5	0.5
Scaling factor of EDPVR ( $A$ ), mmHg	0.35	0.35	0.06	0.06
Exponent scaling factor for EDPVR ( $B$ ), ml <sup>-1</sup>	0.033	0.023	0.264	0.264
Unstressed volume ( $V_0$ ), ml	0	0	5	5
	Aortic	Pulmonary	Mitral	Tricuspid
Valvular resistance (forward), (mmHg s)/ml	0.001	0.001	0.001	0.001
	Systemic		Pulmonary (p)	
	Superior (ss)	Inferior (si)		
Arterial resistance ( $R_a$ ), (mmHg s)/ml	2.25	1.5	0.03	
Characteristic impedance ( $R_c$ ), (mmHg s)/ml	0.075	0.05	0.02	
Venous resistance ( $R_v$ ), (mmHg s)/ml	0.0375	0.025	0.015	
Arterial capacitance ( $C_a$ ), ml/mmHg	0.528	0.792	13	
Venous capacitance ( $C_v$ ), ml/mmHg	28	42	8	

LV Left ventricle, RV right ventricle, LA left atrium, RA right atrium, EDPVR end-diastolic pressure–volume relationship



**Fig. 1** **a** The electric equivalent circuit of one and a half ventricle repair. **b** Biventricular repair (normal circulation). **c,d** Variations of Fontan operation [**c** atriopulmonary connection (APC); **d** total cavopulmonary connection (TCPC)]. *LV* and *RV* left and right ventricles, *LA* and *RA* left and right atria, *AV* and *MV* aortic and mitral

valves, *PV* and *TV* pulmonary and tricuspid valves,  $C_a$  and  $C_v$  lumped arterial and venous capacitances,  $R_c$  characteristic impedances,  $R_a$  lumped arterial resistances,  $R_v$  venous resistances, *ss* superior systemic circulation, *si* inferior systemic circulation, *p* pulmonary circulation

$$e_{cc}(t) = 0.5[1 - \cos(\pi t/T_{es,cc})] \quad (0 \leq t < 2T_{es,cc})$$

$$e_{cc}(t) = 0 \quad (2T_{es,cc} \leq t < T_c) \tag{3}$$

where  $t$  is the time from the start of systole,  $T_{es,cc}$  is the duration of systole, and  $T_c$  is the duration of cardiac cycle. Using  $e_{cc}(t)$ , the instantaneous pressure,  $P_{cc}(t)$ , is described by:

$$P_{cc}(t) = [P_{es,cc}(V_{cc}) - P_{ed,cc}(V_{cc})]e_{cc}(t) + P_{ed,cc}(V_{cc}) \tag{4}$$

Ventricular systole is preceded by atrial systole. The time advance of atrial systole ( $DT$ ) is calculated as the fixed fraction of  $T_c$  ( $DT = 0.02T_c$ ). Function of each chamber is characterized by the parameters  $E_{es,cc}$ ,  $T_{es,cc}$ ,  $V_{0,cc}$ ,  $A_{cc}$ ,  $B_{cc}$ , and  $e_{cc}(t)$ . The same  $e_{cc}(t)$  was used for all chambers, but the other parameters were different between chambers, as shown in Table 1.

**Vascular system**

Basically, the pulmonary and systemic circulations are modeled as modified Windkessel impedances. Each vascular system is modeled by lumped venous ( $C_v$ ) and arterial ( $C_a$ ) capacitances, a characteristic impedance ( $R_c$ ) that is related to the stiffness of the proximal aorta or pulmonary artery, a lumped arterial resistance ( $R_a$ ), and a resistance proximal to  $C_v$  ( $R_v$ ). This framework is similar to that used in deriving Guyton’s resistance to venous return [14].

To simulate the postoperative hemodynamics of 1.5VR, the systemic circulation is divided into two parts, the superior and the inferior circulation. Therefore, the parameters of the systemic circulation are also divided into the superior and inferior ones, as shown in Fig. 1. Blood flow in the descending aorta is reported to be 63.8% of the left ventricular output [15]. The compliance of the IVC is considered to be 66.6% of the total venous compliance [16]. Thus, in our model, arterial and venous compliances of the inferior systemic circulation are adjusted to 0.6 times those of the compliance of the total circulation, and the blood flow of the inferior systemic circulation is controlled to be 60% of the left ventricular output by adjusting the resistances of  $R_c$ ,  $R_a$ , and  $R_v$ .

The capacitance of the superior systemic circulation is also divided into arterial ( $C_{a,ss}$ ) and venous ( $C_{v,ss}$ ). Similarly, arterial and venous capacitances are defined for the inferior systemic circulation ( $C_{a,si}$  and  $C_{v,si}$ ) and for the pulmonary circulation ( $C_{a,p}$  and  $C_{v,p}$ ). The ratio of  $C_a$  to  $C_v$  was obtained from the literature [9, 10, 13]. The relationship between pressure ( $P_c$ ) and volume ( $V_c$ ) in each capacitance is described by the following linear formula.

$$P_c = \frac{V_c}{C} \tag{5}$$

The changes in volume in each capacitance ( $dV(t)/dt$ ) are described by the differential equations below

$$\frac{dV(t)}{dt} = \sum Q_{in-flow}(t) - \sum Q_{out-flow}(t) \tag{6}$$

where  $\sum Q_{in-flow}(t)$  and  $\sum Q_{out-flow}(t)$  indicate the sum of instantaneous volumetric flow rates at the inlet and outlet of each compartment, respectively. Each of the aortic, mitral, pulmonary, and tricuspid valves is described as an ideal diode with a serially connected small resistor.

In the 1.5VR model, the superior circulation flows from SVC to PA, while the inferior blood flow returns to RA through IVC as shown in Fig. 1a. The models of 2VR (Fig. 1b) and variations of Fontan operation [Fig. 1c, atriopulmonary connection (APC); Fig. 1d, total cavopulmonary connection (TCPC)] are constructed for comparisons. Although the superior and inferior systemic circulations return to RA in both 2VR and APC models, RA is directly connected to PA in the APC model. In the TCPC model, SVC and IVC are directly connected to PA. All parameter values were the same for all of these models except total stressed blood volume (see below) (Table 1).

### Hypoplastic RV

Hypoplastic RV is physiologically characterized by an increase in RV stiffness caused by hypertrophy and fibroelastosis of RV muscles [6]. Recalling Eq. 2 for RV, we have:

$$P_{ed,RV} = A_{RV} \left[ e^{B_{RV}(V_{ed,RV} - V_{0,RV})} - 1 \right] \tag{7}$$

where  $B_{RV}$  is stiffness constant of RV. The value of  $B_{RV}$  was changed stepwise from 0.023/ml (normal RV) to 0.143/ml (extremely stiff RV) in increments of 0.01/ml to simulate the various degrees of RV stiffness associated with hypoplasia (Fig. 2).

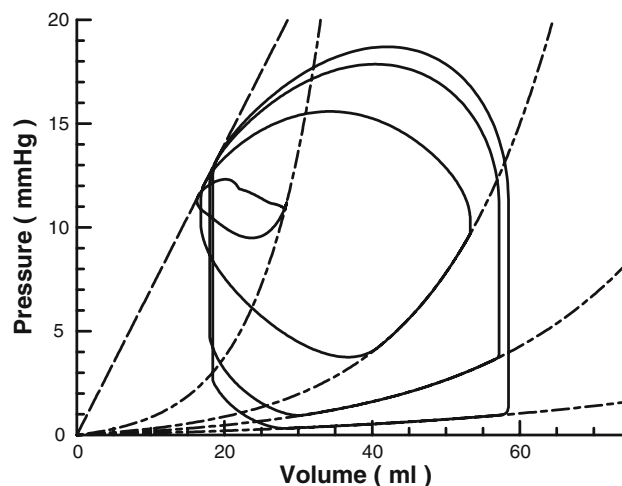
### Protocols

First, the control state was simulated by the 2VR model with normal RV stiffness constant ( $B_{RV} = 0.023$ ). The total stressed blood volume ( $V_s$ ), equal to the sum of the stressed volumes in each capacitance and the volume of each chamber, was set as 750 ml to reproduce normal hemodynamics.

$$V_s = V_{LV} + V_{RV} + V_{LA} + V_{RA} + V_{Ca,ss} + V_{Cv,ss} + V_{Ca,si} + V_{Cv,si} + V_{Ca,p} + V_{Cv,p} \tag{8}$$

We solved these simultaneous equations (Eqs. 1–8) using the component ODE45 of MATLAB, based on the Runge–Kutta method (MathWorks). The hemodynamic parameters of 2VR with normal RV stiffness constant are listed in Table 2.

Next, systemic cardiac output, pulmonary arterial pressure (PAP), right atrial pressure (RAP), and RVEDV after



**Fig. 2** Right ventricular pressure–volume loops (PV loop) after one and a half ventricle repair. With the increase in the right ventricular stiffness constant, the PV loop became smaller. The horizontal axis is the instantaneous right ventricular volume (ml) and the longitudinal axis is the instantaneous right ventricular pressure (mmHg)

**Table 2** Control hemodynamic parameters (2VR with normal RV stiffness constant)

Parameter	Value
Heart rate (HR), beats/min	75
Mean systemic arterial pressure (MAP), mmHg	80.3
Mean pulmonary arterial pressure (PAP), mmHg	13.6
Mean right atrial pressure (RAP), mmHg	2.34
Mean left atrial pressure (LAP), mmHg	8.26
Left ventricular cardiac output (CO), l/min	4.95

each procedure were calculated for each RV stiffness constant. Heart rate was kept constant and mean systemic arterial pressure (MAP) was controlled at the same value as that of the control state, by adjusting the total stressed blood volume.

### Results

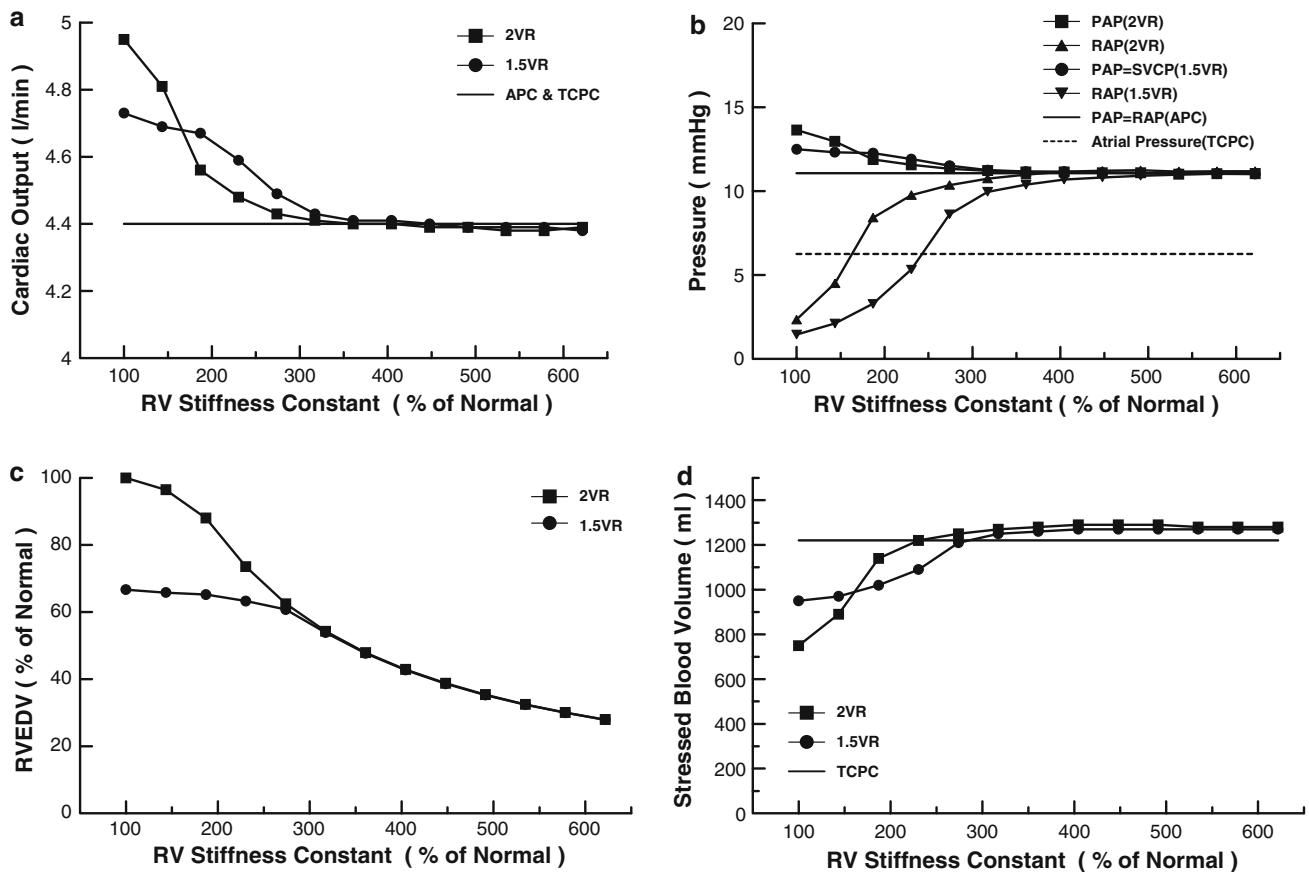
Figure 3a shows the impact of the RV stiffness constant on systemic cardiac output after each procedure. In the Fontan circulation (APC and TCPC), systemic cardiac output was independent of the RV stiffness constant and remained at 4.40 l/min. Under the condition of normal RV stiffness constant, systemic cardiac output was 4.95 l/min in 2VR and 4.73 l/min in 1.5VR, being 13 and 8% greater than that of Fontan circulation, respectively. As the RV stiffness constant was increased from the control value to mimic increased severity of RV

hypoplasia, systemic cardiac output decreased in both 2VR and 1.5VR circulations. Within the range between 100 and 150% of the control RV stiffness constant, systemic cardiac output of 2VR circulation was obviously greater than those of other two circulations. With the RV stiffness constant >150%, systemic cardiac output became greater in 1.5VR than in 2VR. In this situation, 2VR needed larger stressed blood volume than 1.5VR to maintain MAP (Fig. 3d).

The results for PAP and RAP are shown in Fig. 3b. As the RV stiffness constant increased, PAP decreased and RAP increased in both 2VR and 1.5VR circulations. In 2VR circulation, RAP increased steeply as the RV stiffness constant increased up to 150% of normal, and exceeded the atrial pressure of TCPC when the RV stiffness constant increased above 150% of normal. In 1.5VR circulation, RAP also increased but more slowly and exceeded the

atrial pressure of TCPC only when the RV stiffness constant increased above 250% of normal. PAP in 1.5VR circulation, which was equal to SVC pressure, became higher than PAP in 2VR circulation in the range of RV stiffness constant >150% of normal.

In the control state, RVEDV in 2VR was 87.7 ml, which was treated as the value of 100% of RVEDV. The influence of the RV stiffness constant on RVEDV is shown in Fig. 3c. In 2VR circulation, RVEDV decreased as the RV stiffness constant increased. In 1.5VR circulation, RVEDV reduced only slightly with an increase in the RV stiffness constant until 250% of normal. In the range of RV stiffness constant >250% of normal, RVEDV showed a relatively linear decay in both 2VR and 1.5VR circulations, and there was no difference in RVEDV between 2VR and 1.5VR. In this situation, both 1.5VR and 2VR needed larger stressed blood volume than Fontan circulation (Fig. 3d).



**Fig. 3** a The relationship between systemic cardiac output (l/min) and % stiffness constant of hypoplastic right ventricle. The horizontal axis is the ratio of RV stiffness constant (% stiffness constant) to the normal value. b The relationship between pulmonary arterial pressure or right atrial pressure (mmHg) and % stiffness constant of hypoplastic RV. Pulmonary arterial pressure is the same as right atrial pressure in APC. c The relationship between % RVEDV and

% stiffness constant of hypoplastic RV. d The relationship between stressed blood volume (ml) and % stiffness constant. 2VR biventricular repair, 1.5VR one and a half ventricle repair, APC and TCPC variations of Fontan operation (APC atriopulmonary connection, TCPC total cavopulmonary connection); PAP pulmonary arterial pressure, RAP right atrial pressure, SVCP superior vena caval pressure, RVEDV right ventricular end-diastolic volume

## Discussion

The results of this theoretical analysis suggest that, in patients with hypoplastic RV, postoperative hemodynamics depends largely on the RV stiffness constant. PA/IVS, Ebstein's anomaly or their relatives are characterized by varying degrees of underdevelopment of RV. For a severely hypoplastic RV, the definitive treatment is single ventricular circulation. For a mildly hypoplastic RV, biventricular circulation is expected to have merit. Recently, 1.5VR has been proposed to reduce the surgical risk of 2VR. The use of 1.5VR has lowered the early or midterm mortality, and adequate growth of RV and the tricuspid valve has been documented in some patients [2]. However, the postoperative RV dysfunction or arrhythmic event has also been reported, in particular, when the patients are on the borderline of criteria between 1.5VR and Fontan operation [4, 5].

For the choice of surgical options among Fontan operation, 1.5VR, and 2VR, the previously used criteria were based on morphologic characteristics of the hypoplastic RV, such as RVEDV. However, simple anatomic indices may be inaccurate, since these values are dependent on the afterload and preload conditions. For that reason, the treatment strategy for hypoplastic RV based on the anatomic indices remains controversial. We focused on the intrinsic property of hypoplastic RV, i.e., RV stiffness constant. The fact that the RV stiffness constant, an index of chamber property, is relatively independent of the loading condition is important for the accurate prediction of postoperative hemodynamics. Based on the results of the present study, we propose that patients with hypoplastic RV can be classified into three groups according to the RV stiffness constant. The first group consists of patients with mild RV hypoplasia (RV stiffness constant <150% of normal), in whom enlargement of RV is expected after the operation. At the other extreme, the second group consists of patients with severe RV hypoplasia (RV stiffness constant >250%), in whom no RV reconstruction is expected to have merit. In addition, we have shown that there certainly exists a third group consisting of patients with intermediate RV hypoplasia (RV stiffness constant between 150 and 250%), who would benefit more from 1.5VR than from 2VR or Fontan operation.

### Mild RV hypoplasia

When RV hypoplasia is mild (RV stiffness constant <150% of normal), systemic cardiac output is greater in 2VR than in 1.5VR or Fontan operation (APC or TAPC). Therefore, we recommend that 2VR should be chosen in the mild RV hypoplasia group. Although systemic cardiac output in 1.5VR is also greater than that in Fontan operation,

SVC pressure (which is equal to PAP) is higher than that of APC. Accordingly, the upper part of the body is exposed to higher SVC pressure in 1.5VR, which may cause postoperative pleural effusion [2]. A large pressure gradient between SVC and IVC also results in abnormal venous collaterals from SVC to IVC [17–20], and they could effectively increase the venous return to RA in 1.5VR.

### Intermediate RV hypoplasia

When RV hypoplasia is intermediate (RV stiffness constant between 150 and 250% of normal), systemic cardiac output in 1.5VR exceeds that in 2VR. Although SVC pressure is still higher in 1.5VR than in APC, RAP is lower in 1.5VR than in the other procedures. This condition is favorable to reduce supraventricular arrhythmias related to high RAP during the perioperative periods. This beneficial effect is not expected for 2VR since RAP in 2VR is higher than the atrial pressure of TCPC. Furthermore, 1.5VR is advantageous from the viewpoint of stressed blood volume because 1.5VR needs smaller stressed blood volume than does 2VR to maintain MAP (Fig. 3d).

In these patients, RVEDV in 1.5VR is relatively independent of the RV stiffness constant. However, abnormal systemic venous collateral channels might open after 1.5VR. These collateral channels would increase RV preload wastefully and decrease systemic cardiac output in the late postoperative phase. In such conditions, conversion to the Fontan circulation may be required in the late phase [4, 5]. Nevertheless, 1.5VR should be recommended for the intermediate RV hypoplasia group because high cardiac output and low RAP are anticipated.

### Severe RV hypoplasia

When RV hypoplasia is severe (RV stiffness constant >250% of normal), neither 1.5VR and 2VR are expected to improve systemic cardiac output. In this condition, RVEDV is almost the same between 1.5VR and 2VR, and linearly decreases with an increase in the RV stiffness constant in spite of a rapid elevation in RAP. This indicates that RVEDV might be independent of the venous return to RA. Since RAP becomes higher than the atrial pressure of TCPC even in 1.5VR, supraventricular arrhythmias caused by high RAP are liable to occur [2, 5]. In this condition, 1.5VR is considered to have hemodynamics equivalent to APC and needs larger stressed blood volume than does TCPC to maintain systemic arterial pressure (Fig. 3d).

Therefore, TCPC should be chosen for patients with severe RV hypoplasia. In these patients, the arrhythmic events after TCPC are less than that after APC [21, 22]. Although a small pressure gradient between SVC and IVC

remains in 1.5VR, this may not be of clinical significance. Systemic venous collateral channels are expected to be rare, and an increase of RV volume after the operation is unlikely.

### Clinical implication

The management strategy for patients with hypoplastic RV has been based on the morphological characteristics, which are dependent on the loading conditions. In contrast, we used a relatively load-independent index, RV stiffness constant, and simulated the postoperative hemodynamics. As a result, we identified the characteristics of hemodynamics after each of the surgical options, and clearly defined the indications of these operations.

Moreover, our results may be useful to theoretically speculate the reason for contrasting clinical findings. Chowdhury and colleagues [2] reported that the event rate of supraventricular arrhythmia was about 15% in the late postoperative phase of 1.5VR. On the other hand, Numata et al. [5] reported higher arrhythmic event rate. In the former report, the patients had a relatively high postoperative RV volume (45–75% of predicted normal RV; Fig. 3c) and a large pressure gradient between SVC and IVC (mean 7.6 mmHg; Fig. 3b) after 1.5VR. Indeed, there was significant pleural effusion in 22.7% of patients. Our results suggest that good systemic cardiac output, low IVC pressure, and high SVC pressure after 1.5VR can be expected under a condition of a relatively small RV stiffness constant. A great difference between SVC and IVC pressures may cause pleural effusion. Therefore, patients in the former report are likely to have low RV stiffness. In the latter report, the average RVEDV at 1 year after 1.5VR was about 50% of normal and there was no obvious collateral after the surgery in the patients examined. These data suggest a high RV stiffness (Fig. 3c), and a small difference between SVC and IVC pressures (Fig. 3b). Since higher arrhythmic event rate is likely to be associated with high RAP in patients with high RV stiffness, we can interpret the marked difference in arrhythmic event rate in these studies based on postoperative hemodynamics. Operations with 1.5 VR in potentially inappropriate patients (i.e., patients with stiffer RV) might impair

long-term outcomes by continued high RAP-induced arrhythmia.

If we can assess the RV stiffness constant and other hemodynamic data in a catheter laboratory before operation, we will be able to select the most suitable operation for patients with hypoplastic RV. Recently, noninvasive methods for predicting LV chamber stiffness using echocardiography have been reported [23–25]. For example, LV chamber stiffness has been estimated from the deceleration time of LV early filling, effective mitral area and length. Such a method may be applied to estimate RV chamber stiffness using the deceleration time of RV early filling, effective tricuspid area and length. Moreover, it may be possible to choose an appropriate procedure for individual patients by performing simulation of postoperative hemodynamics from individual data using our model. Further clinical studies are needed to precisely assess the RV stiffness constant, including the above methods.

### Limitations

A major limitation of this study is related to the parameters we used for the model. In our model, all parameters other than the RV stiffness constant are fixed. It is reported that RV end-systolic elastance as well as the RV stiffness constant depend upon RV histological changes such as RV hypertrophy [26]. The increase in RV end-systolic elastance moves the beneficial range of 1.5VR toward the stiffer range of the RV stiffness constant. The increase of heart rate also moves the range toward the stiffer range (Table 3). Moreover, ischemia caused by long-standing hypoxemia and hypertension of RV may influence other variables [6]. The existence of pulsatility of the pulmonary circulation may also affect the pulmonary vascular resistance [27]. Tricuspid regurgitation may also impair the postoperative hemodynamics. These limitations may be solved by using the preoperative data of individual patients. Santamore and Burkhoff have already reported the importance of ventricular interdependence using a computer model [13]. However, ventricular interdependence between small hypoplastic RV and relatively large left ventricle may be negligible.

**Table 3** The influence of right ventricular end-systolic elastance and heart rate on the beneficial range of the one and a half ventricle repair

	Lower limit of RV stiffness constant (% of normal)	Upper limit of RV stiffness constant (% of normal)
$E_{es,RV} = 0.7, HR = 75$	150	250
$E_{es,RV} = 1.4, HR = 75$	200	300
$E_{es,RV} = 0.7, HR = 100$	175	275

RV Right ventricle,  $E_{es,RV}$  right ventricular end-systolic elastance (mmHg/ml), HR heart rate (beats/min)

## Conclusion

Using a model analysis, we have shown that the beneficial effect of 1.5VR depends on the RV stiffness constant. 1.5VR is the most beneficial for hypoplastic RV with 150–250% of normal RV stiffness constant. The beneficial range of 1.5VR may also be changed by individual parameters other than the RV stiffness constant, but the beneficial range certainly exists. Therefore, determination of management strategy should be based not only on the morphologic parameters but also on the physiologically determined properties.

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