Myocardial blood supply through a direct left ventricle–coronary artery shunt is not aided by augmented coronary capacitance

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Objectives: Left ventricle–coronary artery shunting is proposed as an alternative means of myocardial revascularization when standard methods are not an option. During diastole, however, regurgitant coronary flow to the left ventricle decreases the efficacy of the left ventricle–coronary artery shunt. We investigated whether augmented coronary compliance would improve net forward shunt flow.

Methods: In 11 anesthetized pigs a specially designed stent was placed through the lateral wall of the left ventricle. Through an arterial graft, it was connected to the proximal left anterior descending coronary artery. A blind stump of the right internal thoracic artery (15 cm) was anastomosed to the distal left anterior descending coronary artery to serve as added coronary compliance chamber. Blood flow was measured in the coronary artery just distal from the left ventricle–coronary artery shunt, as well as in the shunt and in the compliance chamber entrance-exit.

Results: The left ventricle–coronary artery shunt decreased the net forward mid-coronary flow to $53\% \pm 18\%$ (mean $\pm$ SD) of native flow (8 ± 4 vs 16 ± 5 mL/min at baseline, $P < .01$). The augmented compliance did not significantly increase net forward coronary flow ($61\% \pm 25\%$ of native flow, $P < .01$ vs baseline and $P = .21$ vs left ventricle–coronary artery shunt with normal compliance). The increase in systolic forward flow (53 ± 23 vs 37 ± 19 mL/min with normal compliance) was accompanied by a similar increase in diastolic regurgitant flow (-26 ± 20 vs -16 ± 16 mL/min).

Conclusion: In healthy pigs a left ventricle–coronary artery shunt decreased net forward coronary flow to $53\% \pm 18\%$ of native flow. Augmentation of coronary artery compliance did not improve shunt performance.

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Left ventricle–coronary artery (LV-CA) shunt implantation is proposed as an alternative revascularization procedure when percutaneous transluminal coronary angioplasty and coronary artery bypass surgery are not an option. Already in the late 1960s, direct ventricular sourcing was studied by several groups,¹⁻³ who concluded that direct sourcing would make the myocardium ischemic and was unlikely to be useful.² Recently, the concept was revisited by Tweden and colleagues⁴ and Suehiro and associates,⁵ who showed that in their model direct sourcing from the LV resulted in a net forward coronary artery flow of approximately 50% to 70% of native flow. Because these shunts do not include a valve-like mechanism, during diastole, substantial backflow to the left ventricular cavity occurs (pressure sink). It is obvious that the efficacy of the LV-CA shunt will improve with minimization of the backward flow to the LV during diastole.^{5,6}

We hypothesized that augmentation of the compliance of the epicardial coronary artery might enhance net forward flow of the LV-CA shunt by enhancing forward flow in early diastole without reducing forward flow in systole. Augmentation of the

coronary compliance, which is defined as the change in luminal volume divided by the corresponding change in pressure, might be obtained by enlarging the total volume of the epicardial coronary bed.

In the present study phasic flow was measured simultaneously in an LV-CA shunt and in the revascularized coronary artery with and without augmented vascular compliance. The results were compared with those of both native coronary flow and conventional coronary bypass flow. Increased compliance was obtained by connecting a blind stump of an internal thoracic artery (ITA) to the distal coronary artery ("added Windkessel").

Methods

Animals

All experiments were performed in accordance with the "Guide for the Care and Use of Laboratory Animals" prepared by the Institute of Laboratory Animal Resources, National Research Council, and published by the National Academy Press (revised 1996) and with prior approval of the Animal Experimentation Committee of the Faculty of Medicine, Utrecht University, the Netherlands. One day before the experiment, all animals received 560 mg of acetylsalicylic acid orally.

Anesthesia

Eleven pigs of either sex (77 ± 6 kg) were sedated with ketamine (10 mg/kg administered intramuscularly) and atropine (0.01 mg/kg administered intramuscularly) and anesthetized with thiopental (4 mg/kg administered intravenously) before they were intubated and connected to a respirator for intermittent positive pressure ventilation with a mixture of oxygen and air (1:1 vol/vol). Anesthesia was maintained with 0.5% to 1.0% halothane and with continuous infusion of midazolam ($0.3 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ administered intravenously), analgesia was obtained by means of continuous infusion of sufentanil citrate ($1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ administered intravenously), and muscle relaxation was obtained through infusion of pancuronium bromide ($0.1 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ administered intravenously). A bolus of propranolol was administered (0.05 mg/kg administered intravenously) before the operation to reduce the mechanical irritability of the heart during the operation.

Surgical Intervention

Through a midline pretracheal incision, both the left and right common carotid arteries were exposed, through which 2 Millar pressure catheters were placed in the aorta and LV to measure aortic pressure and left ventricular pressure, respectively. A venous catheter was placed in the jugular vein for continuous administration of saline and anesthesia.

After a midsternotomy, the left and right ITAs were harvested in a skeletonized fashion. The animal was heparinized during the entire procedure to obtain an activated clotting time of at least 300 seconds (150 IU/kg administered intravenously). The pericardium was opened, and the heart was suspended in a pericardial cradle. Before dissecting, the left anterior descending coronary artery (LAD) was stabilized with an Octopus 2 Tissue Stabilizer (Medtronic, Inc, Minneapolis, Minn) by using suction along the side of the right ventricle. Before creating an anastomosis between

the distal part of the LAD and the right ITA, myocardial preconditioning, used only to protect the heart against arrhythmias, was performed with 5 minutes of total coronary occlusion by use of a microvascular Acland clamp (S&T Marketing Ltd, Neuhausen, Switzerland), followed by 5 minutes of reperfusion. The distal end of the right ITA was cleaned from loose periadventitial tissue and cut at an angle of 45° . After clamping the distal LAD, followed by an arteriotomy of about 5 mm, a right ITA-LAD anastomosis (end-to-side fashion) was constructed on the beating heart.

For creation of an LV-CA shunt, a segment of 4 to 5 cm of the ITA was sutured in an end-to-end fashion to a specially designed polytetrafluoroethylene-covered stent (23 or 25 mm in length; Percardia Inc, Merrimack, NY). Subsequently, the conduit was positioned over a polytetraethylene 2.5-mm balloon. The implantation side of the stent was determined at the anterior free wall of the LV by using B-mode epicardial echocardiography. Before stent placement, a marked hollow needle was punctured through the wall and replaced with a guidewire. The stent was then stabbed through the left ventricular wall and led by the guidewire. After the stent was expanded for 30 seconds by means of balloon inflation (pressure, 16 atm), the balloon was removed from the left ITA graft-stent conduit. Myocardial preconditioning, used only to protect the heart against arrhythmias, was then obtained through 5 minutes of segmental LAD occlusion between the first and second diagonal branch, followed by 5 minutes of reperfusion. Subsequently, the ITA conduit was anastomosed to the proximal part of the LAD between the first and second diagonal branches. During segmental LAD occlusion needed for the construction of the left ITA graft-proximal LAD anastomosis, the distal right ITA anastomosis perfused the myocardium retrogradely. A snare was placed around the LAD proximal from the LV-CA shunt-LAD anastomosis for occlusion of the proximal LAD during direct blood supply from the LV.

Transit time flow probes (Transonic Systems, Inc., Ithaca, NY) were placed around the distal right ITA (closely to the anastomosis with the distal LAD), the mid-LAD, and the LV-CA shunt (Figure 1). In 5 animals a flow probe was placed around the LAD proximally from the LV-CA shunt, whereas in the other 6 animals a flow probe was placed around the LAD distally from the right ITA-LAD anastomosis.

Experimental Protocol

The present model enabled the comparison in the same animal of myocardial blood supply (1) by using its native coronary artery, (2) by using the LV-CA shunt, and (3) by using conventional coronary artery bypass.

First, during native flow, both the left and right ITAs were occluded near the side of the anastomosis (Figure 2, phase 1), which was referred to as baseline. In phase 2 phasic blood flow through the LV-CA shunt (ie, systolic and diastolic blood flow separately) was studied when both the proximal LAD and LV-CA shunts were fully patent. In the next phases, after blocking the proximal LAD, phasic blood flow through the LV-CA shunt was studied during normal compliance (phase 3) or enlarged compliance (phase 4). This added Windkessel was created by means of ligation of the distal right ITA bypass more distally from the LAD, whereby the length of the right ITA was approximately 15 cm. Finally, blood supply by conventional coronary artery bypass was

Figure 1. Schematic presentation of the surgical procedure. A distal bypass was created by anastomosing the right ITA to the distal part of the LAD. The LV-CA shunt, which consisted of a part of the left ITA (arterial conduit) and a stent, established a direct ventricular sourcing of the coronary artery. Blood flows were measured with transit time flow probes at the site of the proximal LAD ($Q_{Prox\ LAD}$), the mid-LAD ($Q_{Mid\ LAD}$), the distal LAD ($Q_{Distal\ LAD}$), the right ITA ($Q_{Right\ ITA}$), and the LV-CA shunt ($Q_{LV-CA\ shunt}$).

compared with direct blood supply by the LV-CA shunt (phase 5), which was accomplished by means of occlusion of both the LV-CA shunt and the proximal LAD.

Data and Statistical Analysis

All hemodynamic data were digitized at a minimal sampling rate of 300 Hz and stored for off-line analysis. Instantaneous, direction-sensitive volume flow data (in milliliters per minute) were averaged during systole and diastole separately. End-diastole was defined as the onset of rapid increase in left ventricular pressure, whereas end-systole was defined at the maximal rate of decrease in left ventricular pressure ($LV\ dp/dt_{min}$). Net forward flow was obtained by averaging the instantaneous volume flow data (in milliliters per minute) during the cardiac cycle. Forward flow was defined as flow toward the myocardium (positive flow). Calculated flows were each averaged over at least 5 successive cardiac cycles and presented as mean systolic and mean diastolic flows and mean net forward flow.

All data are presented as means $\pm$ SD. For comparison of means, the Student t test was used when data were normally distributed, whereas a Wilcoxon rank sum test was used when data were not normally distributed.

Results

Coronary Blood Flow

When both the proximal LAD and LV-CA shunt were fully patent (phase 2), proximal LAD flow increased from 38 ± 16 mL/min at baseline to 126 ± 66 mL/min ($P = .04$, Table 1). The increase in flow was merely caused by an increase in diastolic forward flow (203 ± 94 vs 54 ± 22 mL/min at

baseline, $P = .04$) because systolic forward flow remained unchanged (24 ± 10 vs 21 ± 13 mL/min at baseline, $P = .68$). The large increment in diastolic flow was lost through the LV-CA shunt to the left ventricular cavity (-174 ± 139 mL/min). Mid and distal net forward LAD flow (both measured distally from the LV-CA shunt) remained unchanged.

When the myocardial blood supply originated directly from the LV (proximal LAD totally occluded, phase 3) the blood flow pattern in the LV-CA shunt showed a systolic forward flow (72 ± 31 mL/min) and a diastolic regurgitant flow (-32 ± 37 mL/min) that resulted in a net forward LV-CA shunt flow of 16 ± 9 mL/min. In addition, the blood flow pattern in the epicardial coronary artery changed from diastolic inflow to enhanced systolic inflow and diastolic regurgitant flow (Figure 3), resulting in a decrease in net forward mid-LAD flow to $53\% \pm 18\%$ ($P < .01$) of net forward mid-LAD flow during baseline (phase 1). Even though systolic mid-LAD forward flow increased 5-fold, the decrease in net forward mid-LAD flow was caused by diastolic regurgitant flow (-16 ± 16 vs 22 ± 9 mL/min at baseline, Figure 4).

When epicardial coronary compliance was enhanced by including the right ITA stump (phase 4), both systolic and diastolic LV-CA shunt flow increased from 72 ± 31 to 91 ± 40 mL/min ($P = .01$) and -32 ± 37 to -44 ± 46 mL/min ($P = .02$), respectively. In addition, in the mid-LAD both systolic and diastolic blood flow increased significantly to 53 ± 23 mL/min ($P < .01$) and -26 ± 20

Figure 2. Experimental protocol comprising various coronary artery blood sources. Blood supply was accomplished consecutively by native flow (phase 1), by both native flow and the LV-CA shunt (phase 2), by the LV-CA shunt alone with normal compliance (phase 3) and increased compliance (phase 4), and by conventional bypass (phase 5).

mL/min ($P < .01$), respectively. Although peak systolic mid-LAD flow was similar to the peak systolic mid-LAD flow when the compliance was normal, mean systolic inflow was enhanced because volume flow over time was increased, whereas diastolic regurgitant flow was increased because diastolic peak mid-LAD flow was increased (Figure 3). This increase in phasic blood flow could be ascribed to the inflow during systole and outflow during diastole of the right ITA stump (-18 ± 18 mL/min and 14 ± 14 mL/min, respectively). However, net forward flow remained the same in both the LV-CA shunt ($P = .47$), mid-LAD ($P = .21$), and distal LAD ($P = .07$), indicating no or minimal contribution of increased coronary compliance to the efficacy of the LV-CA shunt.

Central blood sourcing from the fully patent distal right ITA bypass (conventional bypass, phase 5) showed a mean blood flow of 44 ± 21 mL/min over the right ITA to the relevant myocardium, whereas the distal LAD flow remained unchanged (3 ± 2 vs 3 ± 1 mL/min at baseline, $P = .25$). In comparison, mean LV-CA shunt flow during direct ventricular sourcing to the relevant myocardium was $41\% \pm 30\%$ of mean right ITA flow during conventional bypass (16 ± 9 vs 44 ± 21 mL/min, $P < .01$). In addition,

blood flow to the upper LAD region during blood supply by the LV-CA shunt (phase 3, upper LAD region $\approx$ LV-CA shunt flow – mid-LAD flow: 16 ± 9 to 8 ± 4 mL/min) is approximately 50% of the blood flow during blood supply by means of a conventional coronary artery bypass (phase 5, upper LAD region $\approx$ mid-LAD flow: 20 mL/min).

Discussion

The principal findings of the present study are as follows: (1) direct ventricular sourcing results in a net forward epicardial coronary blood flow of $53\% \pm 18\%$ of native epicardial coronary blood flow, and (2) increasing epicardial compliance does not contribute to LV-CA shunt performance, as demonstrated by the unchanged mid-LAD net forward flow.

This is the first animal study to show the direct measurement of blood flow through an LV-CA shunt. In the proposed clinical procedure,⁶ the LV-CA shunt is placed through the posterior wall of the LAD, which provides blood flow to both the proximal and distal segments of the LAD. In our experimental model we chose to interpose an ITA graft segment between the stent and the LAD. Al-

TABLE 1. General hemodynamics and coronary blood flow

	Phase 1: Baseline	Phase 2: Native + LV- CA shunt	Phase 3: LV- CA shunt	Phase 4: LV-CA shunt; increased compliance		Phase 5: Conventional bypass
				1 compliance chamber (n = 11)	2 compliance chambers (n = 3)	
HR, beats/min	77 ± 25	73 ± 19	74 ± 19	74 ± 22	74 ± 12	74 ± 22
MAP, mm Hg	82 ± 17	73 ± 15*	71 ± 22*	75 ± 18*	85 ± 14	74 ± 15
RPP, mm Hg · beats/min	6159 ± 2056	5202 ± 1355*	5139 ± 1782*	5474 ± 1747	6289 ± 1213	5356 ± 1483*
LV dP/dt max, mm Hg/s	1520 ± 360	1370 ± 280*	1170 ± 280*	1260 ± 240*	1170 ± 400	1390 ± 300*†
LV dP/dt min, mm Hg/s	−1100 ± 260	−1030 ± 260*	−940 ± 320*	−1000 ± 240*	−1010 ± 150	−1060 ± 290†
LVEDP, mm Hg	13 ± 4	13 ± 6	14 ± 6	15 ± 5	9 ± 3	14 ± 5
Proximal LAD flow, mL/min (n = 5)						
Diastolic	54 ± 22	203 ± 94*	—	—	—	—
Systolic	21 ± 13	24 ± 10	—	—	—	—
Net forward	38 ± 16	126 ± 66*	—	—	—	—
Mid-LAD flow, mL/min (n = 11)						
Diastolic	22 ± 9	20 ± 15	−16 ± 16*	−26 ± 20*†	−13 ± 19	−23 ± 12*
Systolic	8 ± 4	10 ± 5	37 ± 19*	53 ± 23*†	53 ± 21	−13 ± 7*†
Net forward	16 ± 5	16 ± 10	8 ± 4*	9 ± 5*	16 ± 15	−20 ± 13*†
Distal LAD flow, mL/min (n = 6)						
Diastolic	3 ± 2	3 ± 2	−2 ± 3*	−1 ± 2*	—	4 ± 3†
Systolic	2 ± 1	2 ± 1	7 ± 4*	6 ± 4*	—	2 ± 2†
Net forward	3 ± 1	3 ± 1	2 ± 1*	2 ± 1*	—	3 ± 2†
LV-CA shunt flow, mL/min (n = 11)						
Diastolic	—	−174 ± 139	−32 ± 37	−44 ± 46†	−40 ± 15	—
Systolic	—	−4 ± 11	72 ± 31	91 ± 40†	109 ± 36	—
Net forward	—	−103 ± 94	16 ± 9	17 ± 10	27 ± 16	—
Right ITA flow, mL/min (n = 11)						
Diastolic	—	—	—	14 ± 14	19 ± 14	51 ± 13
Systolic	—	—	—	−18 ± 18	−28 ± 19	26 ± 13
Net forward	—	—	—	0 ± 1	2 ± 5	44 ± 21

LV-CA shunt, Left ventricle–coronary artery shunt; HR, heart rate; MAP, mean arterial pressure; RPP, rate pressure product; LV dP/dt max, maximal rate of increase in left ventricular pressure; LV dP/dt min, maximal rate of decrease in left ventricular pressure; LVEDP, left ventricular end-diastolic pressure; LAD, left anterior descending coronary artery; ITA, internal thoracic artery.

*P < .05 versus baseline (phase 1).

†P < .05 versus LV-CA shunt (phase 3).

though this is unlike the proposed clinical setting, our goal was to measure LV-CA shunt flow directly.

When the LV-CA shunt was operational in competition with the native LAD, proximal mean LAD flow increased 4-fold. Approximately 80% of this flow, however, was lost through the LV-CA shunt into the LV during myocardial relaxation, when arterial blood pressure in the coronary system exceeded left ventricular pressure. Distal to the LV-CA shunt, there was no change in mean coronary blood flow, which was expected because in a comparable pig model, it was shown that the mean diastolic coronary pressure remained unchanged.⁶

When the native LAD was totally occluded, perfusion by means of direct sourcing from an LV-CA shunt was predominantly during systole. Although systolic flow was substantial, diastolic regurgitant flow reduced net forward mean LAD flow because of the absence of valves in the LV-CA shunt. Consequently, net forward flow was 53% ± 18% of

native flow, which was comparable with the results reported in other recent studies that range between 45% and 76% of native flow.^{4,7} The considerable variation in the efficiency of the LV-CA shunt between these studies is irrespective of animal model (dog versus pig) and implantation method (off artery versus on artery). Moreover, in our study large differences were also found between animals (net flow range, 20% to 80% of native flow), although anesthesia, implantation method, LV-CA shunt, and rate–pressure product were similar in these animals. The changes in flow we observed in the LV-CA shunt and the coronary artery are out of range of physiologic blood flows. This increased shuttle flow during blood supply by the LV-CA shunt (ie, forward blood flow during systole and regurgitant blood flow during diastole) might result in high shear rates and consequently lead to arterial remodeling like an increase in vessel diameter.^{8,9} The effect of this vascular change might be subject for further research.

Figure 3. Representative recording of mid-LAD flow pattern during native blood supply and during direct ventricular sourcing with an LV-CA shunt. The *top graphs* show the left ventricular pressure (LVP, in millimeters of mercury), the *middle graphs* show the mid-LAD flow (in milliliters per minute), and the *bottom graphs* show the LV-CA shunt flow (in milliliters per minutes). S, Systole; D, diastole. End-diastole was defined as the onset of rapid increase in left ventricular pressure, whereas end-systole was defined at the maximal rate of decrease in left ventricular pressure. Note 1: Giant sink flows toward the LV during diastole at phase 2. Note 2: At phase 5, negative flow was caused by reversed flow direction in the mid-LAD segment by the right ITA blood supply.

The effect of the diastolic interval on myocardial perfusion is well known. During direct left ventricular revascularization, LV-CA shunt performance is expected to be positively affected by an increased heart rate because in the cardiac cycle the relative duration of the systole will increase. In addition, the relative decrease in diastolic duration will diminish diastolic regurgitation.

During blood sourcing from a (distal) conventional coronary artery bypass, mid-LAD flow was reversed and showed a physiologic normal flow pattern with mainly diastolic perfusion. The difference in myocardial perfusion between conventional coronary artery bypass and direct ventricular sourcing could be explained by the precipitous decrease in coronary artery perfusion pressure during diastole when the coronary capacitance is emptied rapidly through the low resistance shunt into the LV cavity low pressure sink. During systole, the developed extravascular pressure in the myocardial wall limits perfusion of the

myocardium. In particular, the subendocardium is only minimally perfused, whereas the subepicardium receives normal blood supply.^{10,11} Thus adequate perfusion of the subendocardial bed is achieved during diastole only. In the healthy animal low diastolic perfusion pressure is likely to jeopardize the subendocardium. However, it is unknown what the effect of systolic blood supply through the LV-CA shunt will be on subendocardial perfusion in an animal model of chronic intermittent or progressive coronary artery occlusion resulting in collateral development.

We hypothesized that an increase in epicardial compliance might contribute to the overall efficacy of the LV-CA shunt by enhancing forward flow in early diastole without reducing forward flow during systole. In an in vivo dog model Eng and colleagues¹² added an artificial compliance chamber (Windkessel) to the LAD distal from a stenosis that reduced the mean distal perfusion pressure to about 35 mm Hg. The added compliance chamber produced a 40%

Figure 4. Systolic, diastolic, and mean mid-LAD flow presented as percentage of baseline (phase 1: native blood supply) measured just distally from the LV-CA shunt, during baseline (*open bars*), and during direct blood sourcing from the LV-CA shunt with normal compliance (*hatched bars*) and with increased compliance (*filled bars*). Instantaneous, direction-sensitive volume flow data (in milliliters per minute) were measured by using transit time flow probes and averaged during systole and diastole separately. Net forward flow (mean) was obtained by averaging the instantaneous volume flow data (in milliliters per minute) during one cardiac cycle. Data are presented as means $\pm$ SD.

increase in myocardial blood flow, as measured by microspheres. Systolic pressure was decreased, whereas diastolic coronary pressure was increased, keeping the mean coronary pressure unchanged. In the study by Eng and colleagues, however, coronary blood flow originated from its natural pressure source in the ascending aorta. In the present study, in contrast, direct left ventricular sourcing of the LAD created an entirely different situation, with myocardial perfusion being predominantly during systole rather than during diastole. The compliance chamber would absorb almost 1.5 mL of blood during systole and would empty (part of) this blood volume in the endocardium during diastole. Theoretically, this extra blood volume of the compliance chamber could account for about 20% to 50% of the blood supply needed to the area perfused by the LV-CA shunt, given the fact that the blood volume of the coronary vessels is estimated at 6 to 15 mL/100 g left ventricular mass¹³ and that the perfusion area of the LV-CA shunt in our model comprises about 50 g. Flow measurement in the distal right ITA, which served as a compliance chamber, showed filling and emptying of this blind stump, and systolic mid-LAD flow was increased when the epicardial compliance was expanded. However, diastolic mid-LAD regurgitant flow was also increased, suggesting more shuttle flow between the coronary artery and the LV instead of redistributing blood volume to the distal coronary bed during diastole. In 3 additional experiments a second blind stump of the left ITA, near the site of the LV-CA shunt–LAD anastomosis, was used to further augment the com-

pliance. Systolic inflow through the LV-CA shunt increased to 109 ± 36 mL/min, but diastolic regurgitant flow equally increased to -40 ± 15 mL/min (Table 1). As a result, again, mean net forward mid-LAD flow did not differ from direct left ventricular sourcing with normal compliance. Thus augmented coronary compliance, as created in our model, did not improve the efficacy of an LV-CA shunt. A valve or an element with a valve function should be incorporated in the shunt to improve the LV-CA shunt function.

Conclusion

In the healthy pig direct blood sourcing of the myocardium by an LV-CA shunt resulted in a net forward mid-LAD flow of $53\% \pm 18\%$ of native flow. This reduction in net forward flow was not mitigated by augmentation of epicardial coronary compliance. Incorporation of a valve mechanism in the design of the shunt is expected to improve net forward flow adequately.

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