
DIRECT CORONARY ARTERY PERFUSION FROM THE LEFT VENTRICLE

Kotaro Suehiro, MD^a
Juichiro Shimizu, MD, PhD^b
Geng-Hua Yi, MD^b
Shu-Ming Zhu, MD^b
Anguo Gu, MD^b
Robert R. Sciacca, Eng ScD^b
Jie Wang, MD, PhD^b
Daniel Burkhoff, MD, PhD^b

Objectives: Trends in coronary bypass surgery require less invasive techniques and more conduits. We investigated the ability of direct coronary perfusion from the left ventricle to support regional and global cardiac function.

Methods: A conduit was established between the left ventricle and left anterior descending coronary artery (n = 6) with an interposed Starling resistor that allowed for graded regulation of backward flow. Changes of coronary flow, regional function in the territory of the left anterior descending coronary artery, and reactive hyperemia were studied. In 3 separate dogs, functional tolerance to increased heart rate was tested. In another 3 dogs, left ventricle–left anterior descending and left ventricle–left circumflex coronary artery conduits were established simultaneously (double conduit), and global function was tested.

Results: Without flow regulation, flow through the left ventricle–left anterior descending conduit exhibited high peaking (102 ± 35 mL/min), midsystolic forward flow, and large pandiastolic backward flow (peaking at -47 ± 22 mL/min). Mean coronary flow and regional function were maintained at $46.0\% \pm 7.1\%$ (35.8%-54.2%) and $45.3\% \pm 29.1\%$ (-1.8%-74.2%) of their respective normal values. When the Starling resistor was used to regulate backward flow, these values increased to $70.8\% \pm 12.5\%$ (56.8%-90.4%) and $70.2\% \pm 27.8\%$ (23.6%-107.7%), respectively. Coronary and functional reserve with a left ventricle–left anterior descending conduit were not observed. With the double conduit, global ventricular contractility indexed by end-systolic pressure-volume relation averaged $46\% \pm 35\%$ of its normal value.

Conclusions: A left ventricle–coronary artery conduit supplied approximately 45% of normal blood flow and regional function, and both were improved by regulation of backward flow. Therefore, a conduit from the left ventricle to an epicardial vessel could serve as a rapidly deployable means of revascularizing totally occluded coronary vessels for which suitable natural conduits are not available. (*J Thorac Cardiovasc Surg* 2001;121:307-15)

From the Departments of Surgery^a and Medicine,^b Columbia University, New York City, NY.

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Address for reprints: Daniel Burkhoff, MD, PhD, 177 Fort Washington Ave, MHB 5-435, New York, NY 10032 (E-mail: db59@columbia.edu).

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Recently, interest has resurfaced in development of alternative coronary revascularization techniques. One proposed technique is to use an artificial conduit whose distal end is inserted into the vessel and whose proximal end is inserted transmurally directly into the left ventricle (LV).^{1,2} Such a device could be deployed through a minimally invasive approach on a beating heart, for example, in patients with a calcified aortic root or in redo operations where suitable graft material is lacking. However, without a valve inside a ventricle–coronary artery conduit, backflow drainage during diastole resulting in decreased mean forward flows is predicted.

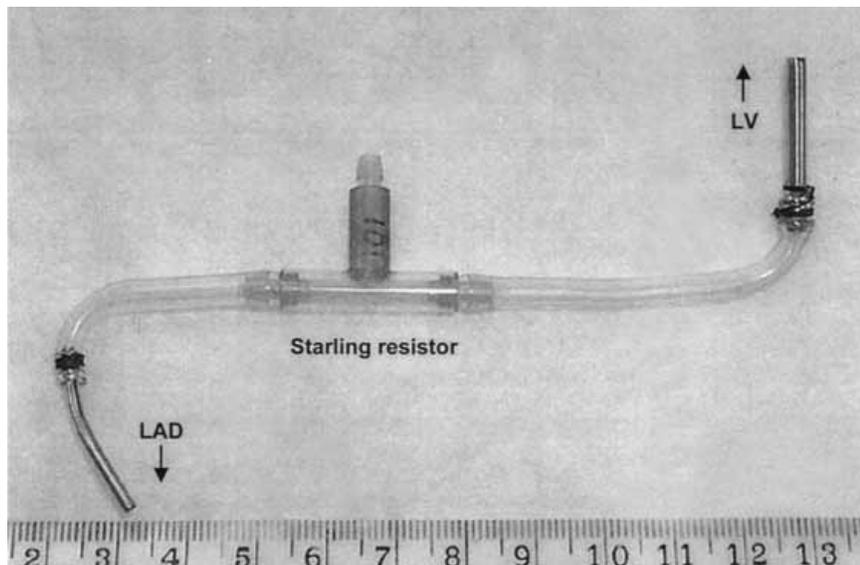


Fig 1. The left ventricle–coronary artery conduit system. The Starling flow resistor³ has a thin-walled, collapsible polyvinyl chloride inner tube (2-mm internal diameter, the “lumen”) inside an airtight T tube that can be pressurized to a desired compression pressure (P_{Starling}). When pressure inside the lumen falls below P_{Starling} , the lumen collapses, acting as a valve. The dead volume of the entire tubing system was 0.95 mL. *LV*, Left ventricle; *LAD*, left anterior descending coronary artery.

The purpose of this study was to elucidate the physiology of blood flow through a direct LV–coronary artery conduit, including the ability of that flow to maintain regional and global myocardial function, and to determine the degree to which impeding backward flow through the conduit provides more effective perfusion.

Methods

Studies were performed in compliance 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.

LV–coronary artery conduit. A system was established to create a conduit between the LV and the left anterior descending coronary artery (LAD). An LAD perfusion cannula (internal diameter of ~1.8 mm) and an LV cannula (internal diameter of ~2.5 mm) were connected with a Starling flow resistor and polyvinyl chloride tubing (Fig 1). The Starling flow resistor³ has a thin-walled, collapsible polyvinyl chloride inner tube (2-mm internal diameter, the “lumen”) inside an airtight T tube that can be pressurized to a desired compression pressure (P_{Starling}). When pressure inside the lumen falls below P_{Starling} , the lumen collapses, acting as a valve. The dead volume of the entire tubing system was 0.95 mL.

Surgical preparations. Twelve adult mongrel dogs, weighing 27 to 32 kg, were used in this study. Each dog was anesthetized with sodium pentobarbital (30 mg/kg intravenously) and ventilated. A conductance catheter with a

Mikro-Tip manometer (Millar Instruments, Inc, Houston, Tex) was inserted into the LV through the right carotid artery to measure LV pressure and volume. The left fifth intercostal space was then entered. The proximal part of the LAD was dissected for later cannulation. The more distal portion of the LAD was dissected and an ultrasonic flow probe (Transonic Systems Inc, Ithaca, NY) was placed. A pair of ultrasonic crystals was placed in the region supplied by the blood flow through the conduit and was connected to a sonomicrometer (Triton Technology, Inc, San Diego, Calif) to monitor segment shortening. So that blood flow to the tested region would be derived predominantly from the LAD, all visible collateral vessels to the LAD were ligated.

The dog was then heparinized (10,000 U intravenously). The LV cannula was inserted through the anterior wall and the proximal LAD was ligated. The coronary perfusion cannula was introduced into the LAD after an arteriotomy, which thus established the LV-LAD conduit. The myocardial ischemic time was no more than 2 minutes.

In the 3 separate double shunt studies (see below), a second conduit was established from the LV to the left circumflex coronary artery (LCx).

Protocol

Coronary flow and regional function. LAD flow and regional function were assessed in 6 animals. Baseline measurements were performed before the LAD was cannulated. The LV-LAD conduit was then established and P_{Starling} initially set at 0 mm Hg, was increased stepwise by increments of 20 to 30 mm Hg until blood flow through the conduit was

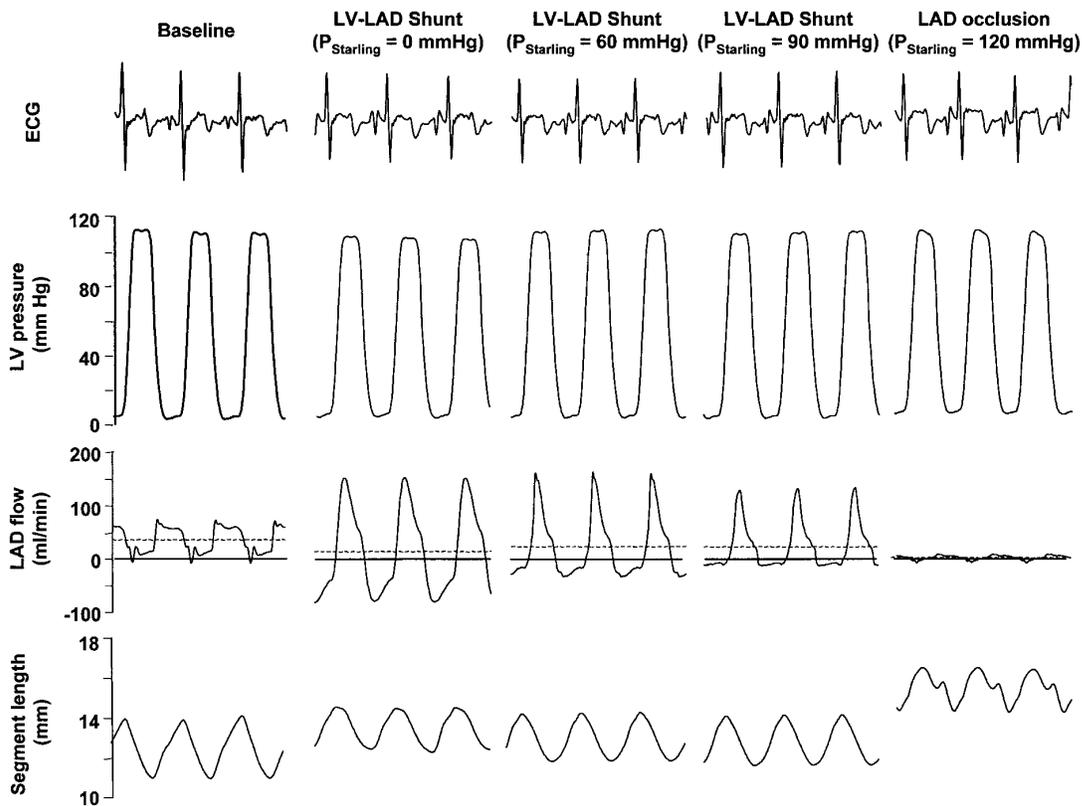


Fig 2. Representative recordings of hemodynamics at various settings of P_{Starling} , the compression pressure in the Starling flow resistor. See text for detailed description of findings. *LV*, Left ventricle; *LAD*, left anterior descending coronary artery.

totally obstructed. At each level of P_{Starling} , 2 minutes were allowed for establishment of stable hemodynamic conditions, at which time physiologic measurements were made.

Regional blood flow. In the same dogs, regional myocardial blood flow was measured under 4 conditions with the use of colored microspheres as described by Kowallik and associates.⁴ First, baseline measurements were obtained before LAD cannulation. During coronary perfusion through the conduit, regional blood flow was assessed at a P_{Starling} of 0 mm Hg, at a P_{Starling} that provided maximum total mean epicardial coronary artery flow, and finally with total occlusion of the conduit.

Reactive hyperemia. Blood flow after release of a 2-minute LAD occlusion (reactive hyperemia) before and after LV-LAD conduit placement was assessed in the same 6 dogs. After reperfusion, coronary flow and regional function were recorded every 20 seconds until each returned to its pre-occlusion value.

Coronary and functional reserve during tachycardia. In 3 dogs, the ability of the conduit without flow regulation to provide sufficient flow to maintain myocardial function at higher heart rates was assessed. Propranolol (0.1-0.2 mg/kg intravenously) was administered to decrease sinus rate to approximately 100 min^{-1} and heart rate was then increased at

20-min^{-1} steps to a maximum of 160 min^{-1} . The heart was paced for 2 minutes at each rate.

Global function during double conduit studies. In 3 dogs, pressure-volume loops were recorded with inferior vena caval occlusions⁵ before the operation (baseline), after placement of the LV-LAD conduit (single conduit), and after placement of the LV-LCx conduit (double conduit). Parallel conductance was determined by the hypertonic saline technique.⁶

Data analysis. In each experiment, except double conduit studies, LAD flow, segmental shortening, and LV pressure were measured. In double conduit studies, LV pressure and volume, LAD blood flow, and LCx blood flow were measured. All data were digitized at 200 Hz and analyzed offline. As an index of regional LV systolic function, regional stroke work (the pressure-segment length loop area) divided by end-diastolic segment length was used. End-systolic pressure-volume relationships (ESPVRs) were determined from the family of pressure-volume loops in the double conduit studies.⁷

Statistics. All data are presented as means and standard deviations. Statistical comparisons were performed with repeated-measures analysis of variance with Bonferroni correction. Linear ESPVRs were compared with analysis of covariance.

Table I. Comparison of LV-LAD conduit flow and normal LAD flow

	Baseline (normal)	LV-LAD conduit ($P_{\text{Starling}} = 0$ mm Hg)	LV-LAD conduit (maximum mean flow)
Heart rate (min^{-1})	134 \pm 12	131 \pm 10	133 \pm 16
Peak flow (mL/min)	58.1 \pm 16.3	101.5 \pm 35.6	85.1 \pm 29.0
Minimum flow (mL/min)	6.9 \pm 8.9	-46.6 \pm 22.0 [†]	-10.5 \pm 8.1 [‡]
Mean flow (mL/min)	31.0 \pm 8.1	14.0 \pm 2.9 [†]	21.1 \pm 2.7*
Positive flow ($\mu\text{L}/\text{beat}$)	223.7 \pm 66	216.6 \pm 65.6	165.7 \pm 31.1
Negative flow ($\mu\text{L}/\text{beat}$)	-0.2 \pm 0.6	-114.8 \pm 58.9 [†]	-12.8 \pm 13.6 [§]

P_{Starling} Compression pressure in Starling flow resistor. Data are expressed as mean \pm SD.

* $P < .01$ versus baseline.

[†] $P < .0001$ versus baseline.

[‡] $P < .01$ versus $P_{\text{Starling}} = 0$.

[§] $P < .0001$ versus $P_{\text{Starling}} = 0$.

Table II. Changes of regional function

	Baseline (normal)	LV-LAD conduit ($P_{\text{Starling}} = 0$ mm Hg)	LV-LAD conduit (maximum mean flow)
P-SL area/EDL (mm Hg)	13.5 \pm 4.8	6.2 \pm 5.1	9.2 \pm 5.0
Recovery ratio (%)	100	45.3 \pm 29.1*	70.2 \pm 27.8

P-SL area/EDL, Pressure-segment length area/end-diastolic length. P_{Starling} compression pressure in Starling flow resistor. Recovery ratio is expressed as percent recovery compared with baseline value. Data are expressed as mean \pm SD.

* $P < .01$ versus baseline.

The organization that funded this research (PerCardia Inc, Nashua, NH) had no role in collection or interpretation of these data.

Results

Coronary flow and regional contractility. Blood flow through the conduit without flow regulation ($P_{\text{Starling}} = 0$) peaked during midsystole and exhibited pandiastolic negative flow (Fig 2). Although the positive flow volume per beat was almost the same as under normal flow conditions, mean net forward blood flow through the conduit was significantly reduced because of the negative flow during diastole (Table I). The pressure-segment length loop after insertion of the LV-LAD conduit (Fig 3, A) was shifted to the right of the control loop, indicating a slight reduction in regional contractility.

When the Starling resistor was progressively pressurized, the negative diastolic blood flow decreased, whereas positive flow was only mildly affected (Fig 2, Table I). This was the case until P_{Starling} reached a critical point at which positive flow dropped precipitously (Fig 3, B, 4, A). On average, maximum mean conduit flow was achieved when P_{Starling} equaled 71.3% \pm 7.0% of peak LV pressure (range 65.3%-83.5%).

Coincident with the increase in mean coronary flow resulting from increases in P_{Starling} , there was a corresponding leftward shift of the pressure-segment length

loop (Fig 3, A), indicating that function improved as flow increased. Indeed, there was a roughly linear relationship between regional function and mean coronary flow as seen in Figs 3, C (typical example) and 4, B (results from all studies). As a result, regional function, which was reduced to approximately 50% of baseline with $P_{\text{Starling}} = 0$, was restored to about 70% of baseline with optimal regulation of negative flow (Table II).

Regional blood flow. Regional blood flow in the LCx territory (control region) was roughly constant throughout the experiment (Fig 5). After establishment of the LV-LAD conduit with $P_{\text{Starling}} = 0$ mm Hg, subendocardial blood flow in the LAD region was decreased. However, at the P_{Starling} that provided maximal mean LAD flow, subendocardial flow was significantly improved. Thus, it was speculated that changes in regional function during pressurization of the Starling resistor would correlate with changes in subendocardial blood flow. This was confirmed (Fig 6) by correlating subendocardial flow and regional function ($r^2 = .54$, $P = .0041$).

Postischemic reactive hyperemia. Representative tracings of coronary flow and a summary of the results from 6 dogs after 2-minute LAD occlusions are shown before (Fig 7, A and C) and after (Fig 7, B and D) creation of the LV-LAD conduit, respectively. During normal perfusion, maximum mean hyperemic LAD flow

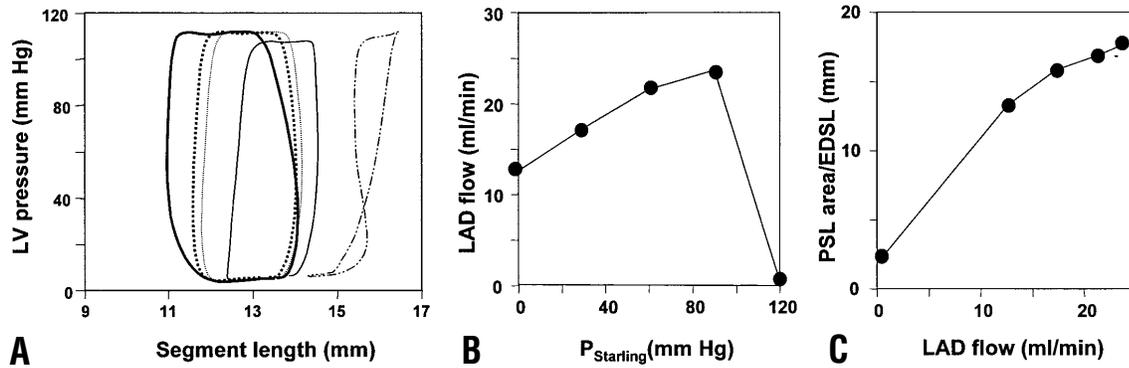


Fig 3. **A**, Representative left ventricular pressure-segment length loops. *Thick solid line*, baseline; *thin solid line*, $P_{Starling} = 0$; *dotted line*, $P_{Starling} = 60$ mm Hg; *dashed line*, $P_{Starling} = 90$ mm Hg; *dashed-dotted line*, $P_{Starling} = 120$ mm Hg. **B**, The relation between $P_{Starling}$ and mean left anterior descending flow. **C**, Relation between mean coronary flow and regional function assessed by pressure-segment length area/end-diastolic segment length (*PSL area/EDSL*). $P_{Starling}$, The compression pressure in the Starling flow resistor; *LV*, left ventricular; *LAD*, left anterior descending coronary artery.

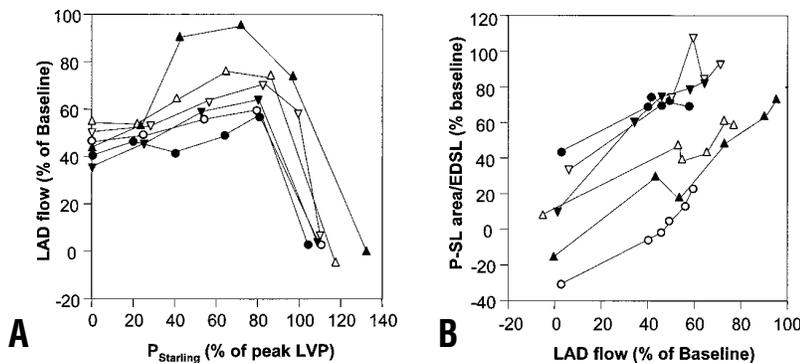


Fig 4. **A**, Relationship between $P_{Starling}$ and mean LAD flow. **B**, Relationship between the pressure-segment length area/end-diastolic segment length (*P-SL area/EDSL*) and mean LAD flow through the LV-LAD conduit. See Fig 3 for abbreviations.

reached $389\% \pm 78\%$ (296%-467%) of mean baseline LAD flow at 9.8 ± 2.9 seconds (6-12 seconds) after release of the occlusion. After LV-LAD conduit placement, however, peak, minimum, and mean LAD flows attained their steady state values within 14.3 ± 6.7 seconds (6-21 seconds) after release of the occlusion. The lack of a hyperemic response indicates that the bed is prevasodilated in the presence of the LV-LAD conduit.

Coronary flow and functional reserve during atrial pacing. Changes in mean LAD flow and regional function in each of 3 dogs studied at different heart rates are shown in Fig 8. In all hearts with natural flow through the LAD, mean coronary flow was mildly increased as heart rate was increased from 100 to 140 min^{-1} . Regional function was relatively well maintained until heart rate reached 160 min^{-1} .

In the presence of the LV-LAD conduit without flow regulation, mean LAD flow was maintained roughly constant at heart rates between 100 and 160 min^{-1} . In terms of the function with the LV-LAD conduit, there was a uniform mild decrease as heart rate increased.

“Double conduit” and global LV function. Typical pressure-volume loops obtained under baseline conditions, with a single LAD conduit, and after addition of the second conduit to the LCx are shown in Fig 9. The ESPVR changed little with the single conduit but shifted downward with the double conduit. The parameters of the ESPVRs along with other hemodynamic parameters under these conditions from the 3 animals studied are summarized in Table III. Analysis of covariance revealed a consistent downward shift of the ESPVR compared with baseline with both conduits in place.

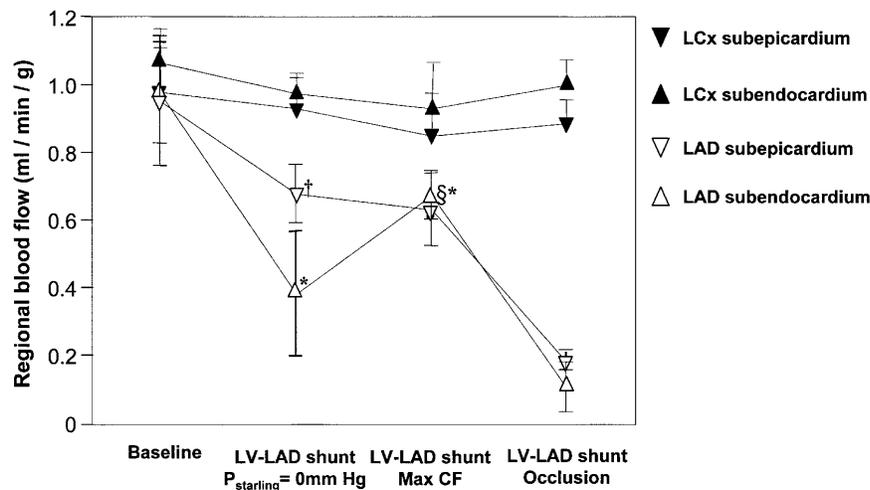


Fig 5. Relationship between subepicardial and subendocardial blood flow and P_{Starling} in the territory of the circumflex coronary artery (LCx) and left anterior descending coronary artery (LAD). LV, Left ventricle; Max CF, maximum coronary flow. * $P < .001$ versus baseline; † $P < .005$ versus $P_{\text{Starling}} = 0$ mm Hg.

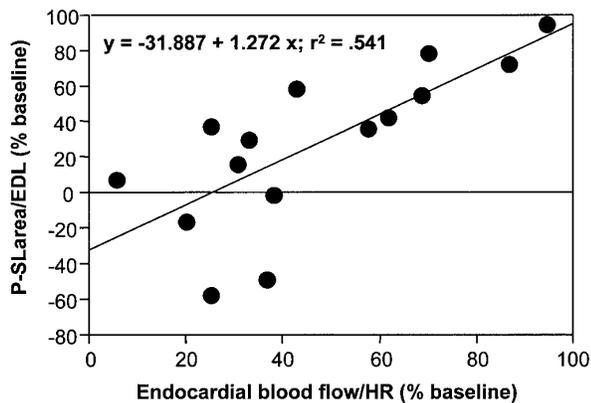


Fig 6. Relation between P-SL area/EDSL and normalized subendocardial blood flow. Subendocardial flow was divided by heart rate and then normalized. See Fig 3 for abbreviations.

When both shunts were occluded, global function deteriorated significantly within a few beats, aortic pressure decreased, and ventricular fibrillation developed relatively quickly. This confirms that blood flow through these conduits was indeed supporting myocardial function to an extent that global function and hemodynamics could be maintained.

Discussion

Use of an LV-LAD conduit without flow regulation was able to provide approximately 45% of normal net

forward flow while maintaining regional function at about 45% of its normal value, both markedly improved compared with conditions with a totally occluded vessel. Control of backward coronary flow further improved net forward flow and regional function, each increasing to approximately 70% of their normal values.

The physiology of LV-LAD conduits has been examined in two prior studies with conflicting results. Munro and Allen¹ reported a reduction of about 70% in net forward flow followed by a significant fall in aortic pressure and myocardial contractility using a plastic cannula connecting the proximal LAD to the LV. More recently, Tweden and associates² implanted a rigid L-shaped titanium cannula in pigs and reported that net forward flow was maintained at approximately 76% of baseline.

In our studies, mean total and subendocardial coronary flow ranged between 35.8%-54.2% and 25.0%-38.0%, respectively, without flow regulation. It has been shown that either 30%-40% of normal transmural flow or 20%-30% of normal subendocardial blood flow is critical for maintaining systolic function.⁸ Consistent with these prior studies, with relatively wide overall ranges of total and subendocardial blood flows observed without regulation of negative flow (ie, with $P_{\text{Starling}} = 0$), we also observed a wide range of functional preservation, ranging from -1.8% (akinesis) to 74% of baseline.

No significant coronary flow reserve was observed during use of the LV-LAD conduit at $P_{\text{Starling}} = 0$ as evi-

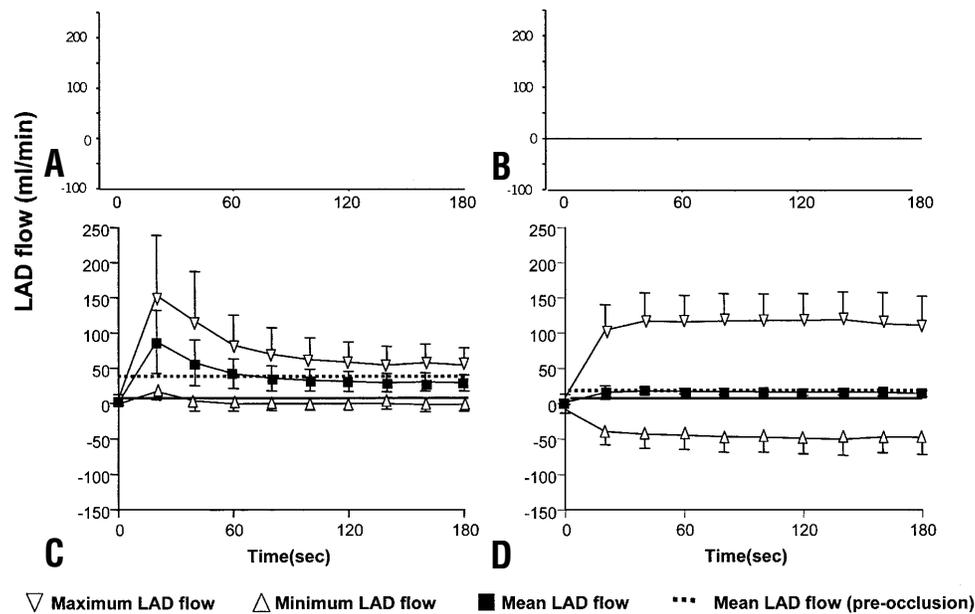


Fig 7. Representative tracings of reactive hyperemic flow and mean LAD flow after release of a 2-minute LAD occlusion before (A and C, respectively) and after (B and D, respectively) establishing an LV-LAD conduit with $P_{\text{Starting}} = 0$. See Fig 3 for abbreviations.

Table III. Global cardiac function and coronary flow with LV-coronary conduit ($n = 3$)

	Control	Single conduit	Double conduit
Peak LV pressure (mm Hg)	118.2 ± 5.5	104.7 ± 8.9	94.0 ± 13.8
Ees (mm Hg)	6.4 ± 1.6	4.8 ± 3.5	3.1 ± 2.9*
V_0 (mL)	1.5 ± 9.8	-12.2 ± 30.5	-0.5 ± 9.0*
Ejection fraction (%)	66.2 ± 9.1	40.1 ± 17.7	40.6 ± 23.6
Mean LAD flow (mL/min)	30.9 ± 12.5	12.1 ± 8.5	12.1 ± 2.5
Mean LCx flow (mL/min)	40.7 ± 8.3	37.8 ± 9.6	19.5 ± 2.7

LV, Left ventricular; Ees, end-systolic pressure-volume relation; V_0 , volume intercept; LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery.

* $P < .05$ versus end-systolic pressure-volume relationship at baseline by analysis of covariance.

denced by lack of a reactive hyperemic response or increase in blood flow with increased heart rate. This suggests that under these conditions, the vascular bed is relatively underperfused and there is a pre-existent vasodilation.

Although normal regional and global function was not fully maintained with either the single or double conduits, the coronary flow through these conduits was physiologically important and significantly better than what was observed with no flow when the shunts were occluded.

Significance of LV-coronary conduits in clinical settings. The number of redo coronary artery bypass operations accounts for nearly 20% of all coronary

revascularization procedures.^{9,10} This population tends to be older and sicker, the ascending aorta may be calcified, and performing multiple proximal anastomoses can be problematic. The number of available conduits may also be limited.¹¹ Thus, the surgical challenges associated with redo operations makes desirable the availability of alternative revascularization techniques implementable by minimally invasive approaches, preferably without cardiopulmonary bypass. Availability of a clinically applicable LV-coronary conduit that would maintain patency could overcome some of these hurdles. Such conduits could be implanted via a limited thoracotomy because the procedure requires only a small area surrounding the target vessel.

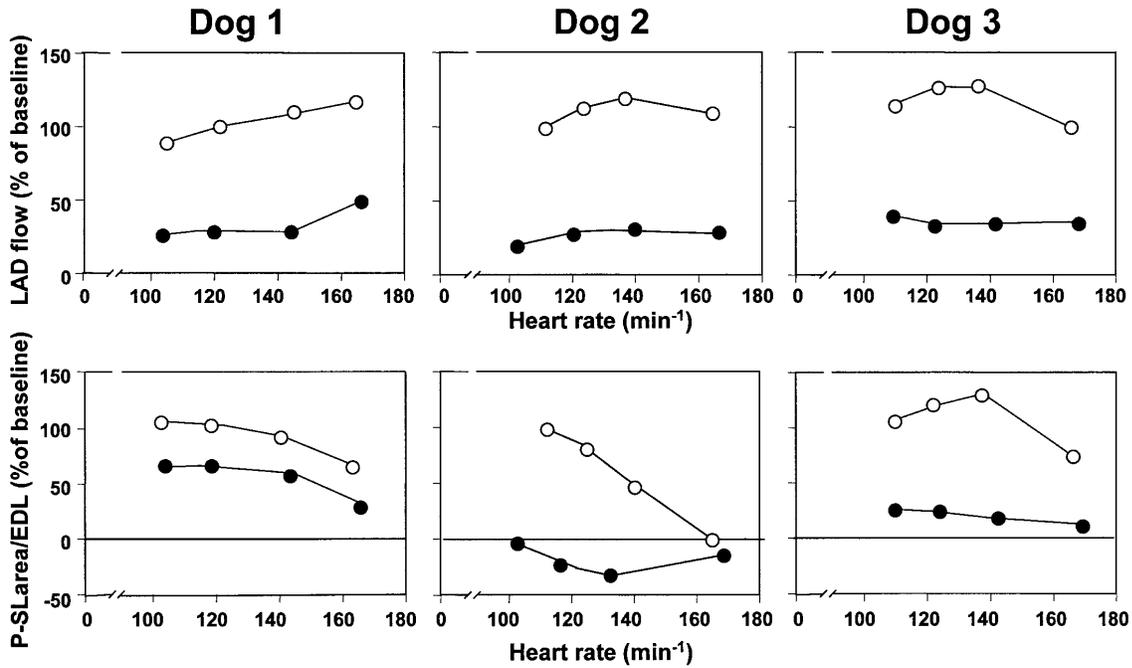


Fig 8. Changes of mean LAD flow and P-SL/EDSL during atrial pacing in 3 dogs before (*upper panels*) and after (*bottom panels*) placement of the LV-LAD shunt. P-SL area/EDL, Pressure-segment length area/end-diastolic length; LAD, left anterior descending coronary artery.

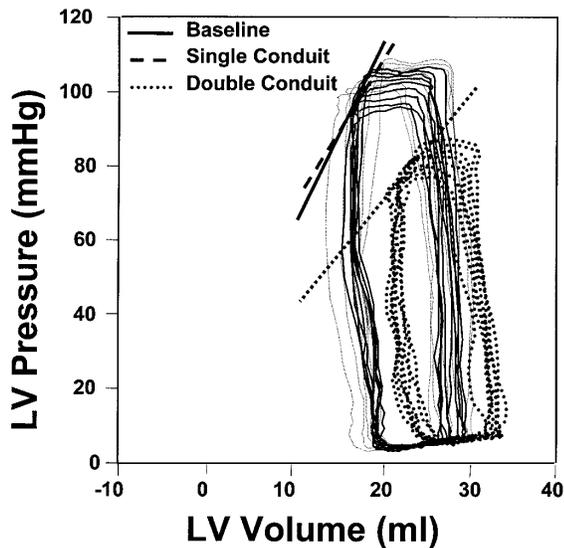


Fig 9. Example of pressure-volume loops and ESPVRs obtained in the double conduit experiments. Relations and loops obtained in this example are similar under baseline and single conduit conditions. With addition of the second conduit, global function is maintained at about half of the baseline condition. See Table III for details of how double hemodynamics are influenced in the double conduit experiment. LV, Left ventricular.

Furthermore, as in the current study, the procedure can be carried out without cardiopulmonary bypass with myocardial ischemic times less than 2 minutes. Another possible application could be for circumstances requiring rapid intraoperative restoration of flow such as emergency procedures after coronary occlusions following interventional procedures (angioplasty) or, as sometimes may occur, with intraoperative compromise of the coronary circulation.

Although normal flow was not achieved with the nonoptimized conduit used in the present study, it markedly improved myocardial perfusion and regional function compared with those of a vessel that was totally occluded. As reported by numerous investigators, revascularization of totally occluded vessels in regions of retained myocardial viability improves regional/global function and symptoms.^{12,13} Thus, especially in the patients with multivessel disease who lack available grafts, more complete revascularization could be achieved through the use of LV-coronary conduits.

Limitations

Dogs have significant pre-existent collateral coronary circulation.¹⁴⁻¹⁶ Therefore, it could be argued that collateral flow could have accounted for a significant amount

of regional flow and function observed after LV-LAD conduit implantation. Experimentally, any visible epicardial collateral vessels were ligated. Furthermore, three pieces of data ensure that this was not the case. First, on occlusion of the LV-LAD conduit, regional function deteriorated profoundly (eg, Figs 2 to 4), indicating that any existing collateral flow was unable to maintain regional function. Second, direct measurement of regional blood flow by means of microspheres (Fig 5) showed that both epicardial and endocardial blood flow dropped to near 0 on conduit occlusion. Third, in the double conduit experiment, all vascular supply is directly from the LV, and the fact that global function could be maintained to approximately 50% of normal (essentially the same as regional function in the single conduit experiment) indicates the lack of reliance on collateral flow. These findings indicate that the results were not influenced by the presence of collateral flow. On the other hand, because collateral vessels were ligated, we could not address whether an LV-LAD shunt used in the presence of significant collateral flow could create a steal phenomenon and induce ischemia in the source bed of the collateral blood. This is an important issue because many patients undergoing bypass surgery have collateral flow to ischemic beds.

Conclusions

An LV–coronary artery conduit can provide about 45% of normal regional coronary flow and function. Incorporation of a flow regulation to minimize backward flow during diastole significantly increases both parameters. Because this LV–coronary conduit significantly improves coronary flow compared with that of a totally or subtotally occluded vessel, this approach could be useful clinically for achieving a more complete degree of revascularization in cases of complicated coronary bypass if a clinically applicable device that maintains patency long term could be developed. It is also possible that a better engineered conduit with more favorable forward than backward flow characteristics could be designed to provide greater net forward flow.

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