Influence of the ischemic coronary bed on collateral blood flow *)

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Summary

The purpose of this study was to determine the influence of the resistance of the terminal vascular bed of an occluded coronary artery on collateral blood flow and collateral resistance. In 6 anesthetized dogs, left anterior descending coronary artery (LAD) was ligated, cannulated, and the terminal vascular bed was occluded by latex microspheres (diameter: 25 μ). Retrograde flow was measured using a new technique, which allowed control of outflow pressure of retrograde flow (PRF) at the LAD cannula. When retrograde flow was interrupted, pressure in the occluded vessel represented collateral perfusion pressure (CPP) within the border zone of the ischemic vessel. Collateral resistance was determined dividing the pressure difference across the collateral bed (CPP-PRF) by retrograde flow. Variation of PRF was used as a model for changes in resistance of the ischemic bed. Retrograde flow fell when PRF was increased from 11.0 ± 3.0 ml × min⁻¹ × 100 g⁻¹ (PRF = 0) to 8.3 ± 2.4 (p < 0.01) (PRF = 24.6 ± 6 mm Hg). For the same PRF range, collateral resistance fell from 9.68 ± 2.96 to 8.30 ± 2.50 mmHg × ml⁻¹ × min × 100 g (p < 0.01). These results indicate that the vascular resistance of the terminal ischemic bed may considerably influence collateral blood flow and resistance.

Key words: coronary collateral circulation, vascular resistance, coronary blood flowmeter, dogs

Introduction

Recent studies from this laboratory have shown that coronary collateral blood flow is dependent on the pressure at the origin of the collaterals from the unimpaired vessel, the collateral resistance, and the vascular bed of the ischemic vessel (6). It might be expected that the ischemic vascular bed is maximally dilated and thus represents a minimal part of the resistance to collateral blood flow (19). However, mean pressure in a coronary artery beyond the site of occlusion (PPC) can be as high as 11 to 32 mm Hg (11, 19, 20, 21, 24, 26). These high pressures imply that the resistance of the ischemic vascular bed may not be insignificant. Studies of Russel et al. confirm the importance of the influence of the terminal vascular bed of the ischemic zone on collateral blood flow (18). Downey and Kirk demonstrated a vascular waterfall mechanism in the coronary circulation of the beating heart (5), suggesting that if the pressure to a given coronary vessel drops below a certain critical level, blood flow

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ceases. At this critical pressure, the resistance of the vascular bed would become very large. However, it has not been shown if a critical pressure also exists for collateral vessels. Since collaterals are vessels with a minimal density of smooth muscle, they could be considered "collapsible tubes", and a waterfall phenomenon could also apply to the collateral vasculature. Thus collateral blood flow would not depend on PPC in the ischemic bed, since: Collateral Blood Flow = (CPP - Surrounding Tissue Pressure) / Collateral Resistance (17). One question to be answered by this study was therefore whether collateral blood flow is influenced by PPC and thus by the resistance of the terminal vascular bed of an occluded coronary artery. For this purpose, PPC had to be controlled, and collateral blood flow had to be measured for a large range of PPC. This was achieved as follows: perfusion of the ischemic myocardium was abolished by occluding the ischemic bed with microspheres. Thus retrograde flow represented total collateral flow and outflow pressure of retrograde flow (PRF) could be varied without draining blood into the ischemic bed (4, 6, 21, 24, 25). In addition, this technique may allow the determination of collateral perfusion pressure (CPP) and collateral resistance. When retrograde flow is interrupted, pressure in the occluded coronary artery represents the pressure in the ischemic borderzone, i.e., the collateral perfusion pressure (CPP) (4, 6, 21, 24, 25). Collateral resistance can thus be determined from the pressure across the collateral bed and retrograde flow (4, 6, 21, 24, 25). It has been shown that collateral resistance is dependent on collateral perfusion pressure (CPP) due to elastic properties of the collateral vessels (21). Assuming that intravascular pressure of the collateral bed is determined by collateral perfusion pressure CPP and by the pressure in the ischemic bed PPC, variations of PPC may induce changes in collateral resistance.

The present study had the following goals: 1) to develop a method which allows the continuous and exact measurement of retrograde flow and variation of PRF; 2) to determine the effects of PRF on collateral blood flow; and 3) to examine whether collateral resistance is pressure-dependent.

**Method**

Six mongrel dogs, weighing 22-34 kg, were anesthetized with chloralose (40 mg/kg i.v.), urethan (250 mg/kg i.v.), and morphine (3 mg/kg s.c.). Dogs were intubated and ventilated using a Starling respirator pump. Expiratory pressure was adjusted to 5 cm H\textsubscript{2}O. Aortic and left ventricular pressures were measured using a double tip manometer catheter (PC 780, Millar, Houston, Texas) inserted via the left femoral artery. Heart rate and dP/dt were computed continuously. Short catheters were inserted into the right femoral artery and vein for blood sampling and injections. Hematocrit, arterial P\textsubscript{0\textsubscript{2}}, P\textsubscript{CO\textsubscript{2}}, and P\textsubscript{H} were measured half-hourly (BMS 2, Radiometer, Copenhagen, Denmark), and respiration was adjusted to maintain normal arterial blood gases. Sodium bicarbonate was infused when needed to compensate for acidosis.

The chest was opened at the fifth left intercostal space, and the pericardium was dissected. The left anterior descending coronary artery (LAD) was isolated for about 3-4 cm distal to its origin, ligated, cannulated and perfused from the right carotid artery by a tubing system (fig. 1). Pressure in the LAD was measured by a