Determinants of Systolic and Diastolic Flow in Coronary Bypass Grafts with Inotropic Stimulation

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Using implanted pulsed Doppler microprobes sutured on saphenous bypass grafts in ten patients we studied, 6 h after cardiac surgery, the effects of 5 and 10 μg·kg⁻¹·min⁻¹ of dobutamine on mean (Q₀), systolic (Qₘ), and diastolic (Q₄) coronary bypass graft flows, as well as on coronary systolic (Qₘ) and diastolic (Q₄) blood volumes entering the myocardium per cardiac beat. Q₀ increased during the inotropic stimulation from 61.8 ± 19.2 to 81.1 ± 21.8 ml·min⁻¹ (P < 0.001) and resulted from an unchanged Qₘ and from a large increase in Q₄ (P < 0.01). Q₄ increased more than did diastolic arterial pressure and was related to rate pressure product taken as an index of myocardial oxygen consumption (r = 0.76, P < 0.001). Despite the dobutamine-induced increase in heart rate (P < 0.01), Qₘ and Q₄, the systolic and diastolic inflow volumes per cardiac beat were unchanged. We conclude that increased myocardial blood supply through the saphenous vein bypass graft during inotropic stimulation by dobutamine resulted from different systolic and diastolic events. The oxygenated blood volume entering the coronary vascular bed per beat was unchanged despite tachycardia. (Key words: Artery, Coronary; blood flow. Sympathetic nervous system, catecholamines; dobutamine. Measurement techniques: Pulsed Doppler. Surgery, Cardiac: CABG.)

After coronary artery bypass graft surgery, coronary vascular tone may be reduced by rewarming, recent reperfusion and post-cardiopulmonary bypass vasodilation. In this situation, maintenance of adequate flow may depend on an increased perfusion pressure, particularly after inotropic stimulation. Because of technical limitations, few measurements of coronary flow are available in such patients and are usually performed using coronary sinus thermodilution.¹ However, this global technique measures only mean coronary blood flow (Q₄) from both reperfused and nonreperfused territories² and is difficult to perform after cardiac surgery. Furthermore, this method has a long response time and does not allow differentiation of systolic (Qₘ) and diastolic (Q₄) components of flow.² Measurements of bypass Q₈ and Q₄ may be important,³ especially during inotropic stimulation, since an increased heart rate may limit Q₄ via a reduction in diastolic time, whereas the enhanced contractile status may increase myocardial extravascular forces, and thus reduce Q₈. In other words, analysis limited to changes in Q₄ cannot estimate the respective changes occurring in phasic (systolic and diastolic) flows (Qₘ and Q₄).

Recent developments in Doppler technology allow measurements of bypass graft diameter and of phasic velocities on a beat-to-beat basis.⁵ We hypothesized that separate analyses of Q₈ and Q₄ rather than analysis of Q₄ would provide better insight into the coronary response to inotropic stimulation after coronary artery bypass graft surgery. Dobutamine was the inotropic drug chosen because of its usually pure inotropic action⁶⁻⁷ without marked tachycardia or important effects on arterial blood pressure.⁵,⁶⁻¹¹

Materials and Methods

Patients

Ten patients (57.8 ± 6 yr, mean ± SD) undergoing aortocoronary bypass graft surgery with saphenous vein grafts were selected for the study. Written informed consent was obtained for each patient after detailed description of the procedure and protocol was approved by the Human Use Committee of the University Paris–Val de Marne, the Fédération Française de Cardiologie, and the Institut National de la Santé et de la Recherche Médicale.

Clinical characteristics and operative procedures are summarized in table 1. Criteria for inclusion were an angiographically proven stenosis of the proximal left anterior descending coronary artery of over 80%, a preoperative left ventricular ejection fraction higher than 50%, a postoperative hemodynamic stability without inotropic or vasodilating drugs, and the absence of electrocardiographic or enzymatic evidence of myocardial ischemia.

Saphenous graft bypass, anesthesia (high-dose fentanyl), and cardiopulmonary bypass procedure (bubble oxygenator primed with Ringer’s lactate) were similar for all patients and included monitoring heart rate (HR, by ECG lead): systolic (SAP), diastolic (DAP), and mean (MAP)
Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Preoperative Treatment</th>
<th>LVEF (%)</th>
<th>Prior MI</th>
<th>Angiographic Findings (%)</th>
<th>Procedure</th>
</tr>
</thead>
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<td>62</td>
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<td>ACBG × 4</td>
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<tr>
<td>3</td>
<td>46</td>
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<td>60</td>
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<td>55</td>
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<td>ACBG × 3</td>
</tr>
<tr>
<td>5</td>
<td>64</td>
<td>Nitrates</td>
<td>60</td>
<td>Post</td>
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<tr>
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<td>LAD (90)</td>
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</tbody>
</table>

LVEF = left ventricular ejection fraction; MI = myocardial infarction; LAD = left anterior descending coronary artery; Diag = diagonal branch; LCx = left circumflex artery; RCA = right coronary artery; LMCT = left main coronary trunk; ACBG = aortocoronary bypass graft; $\beta^-$ = $\beta$ adrenergic receptor blockers; Ca$^+$ = calcium channel blockers.

systemic arterial pressures (mmHg, by radial catheter); and right atrial (RAP), pulmonary artery (PAP), and pulmonary capillary wedge (PCWP) pressures (mmHg). Cardiac index (CI, 1·min$^{-1}$·m$^{-2}$, by thermodilution) was measured as the average of at least three determinations performed in random order during the respiratory cycle.

**CORONARY BYPASS GRAFT FLOW MEASUREMENTS**

After separation from cardiopulmonary bypass, when hemodynamic status was stable, a single-use 8-MHz Doppler microprobe was sutured to the adventitia of the saphenous graft bypassing the left anterior descending coronary artery. Since the signal from Doppler apparatus does not drift and since zero flow is electronically determined, mechanical zero determination was not necessary. Dimensions of the probe used were 4 mm in width and 5 mm in length. The Doppler crystal is glued on a silicone prism in such a way to obtain an incidence angle of 60° between the ultrasonic beam and the red blood cells (fig. 1). Before implantation, the linearity response of each probe for flow velocities ranging from 5 to 80 cm/s was verified. To facilitate implantation and proper alignment of the probe along the vessel axis, the base of the silicone prism was cut in the shape of a concave gauge adapted to vessel curvilinearity. Then, the probe was fixed 2 or 3 cm downstream from the aortic anastomosis so that its removal could not affect graft position and angulation. Four 7-0 sutures (one anterior, two lateral, and one posterior) passed through the adventitia of the saphenous vein ensured close contact between silicone and vessel wall, with a proper alignment in relation to the vessel axis.

The probe was then linked to the Doppler flowmeter via connecting electrodes that emerged from the thorax through the skin. Gentle traction 6 days later removed the probe without any damage to the patient. Eighty-three implantations have been carried out using this material on various coronary vessels without any bleeding or infectious complications (personal data).

The zero-crossing pulsed Doppler blood flowmeter used in this study has been described and validated previously. The dominant feature is the range-gated time system of reception, which allows one to choose the sample volume size and to move it across the vessel lumen. Diameter (D) was determined according to the echographic equation: $D = C/2 \cdot (t_2 - t_1) \cdot \cos 60°$, where C is the
DOBUTAMINE AND CORONARY BYPASS GRAFT FLOW

Data Analysis

ECG leads II, V1, and V5 were monitored on the oscilloscope. Tracings were done at each step of the protocol for later reviewing by an independent investigator unaware of the patient's condition. Criteria for diagnosis of ischemia was the appearance of 1 mm ST-segment depression or elevation 60 ms after the J point, or a T-wave inversion.

Hemodynamic parameters and coronary bypass graft velocities were recorded on a multichannel recorder (Gould S 1000) and analyzed from high-paper-speed recordings (100 mm/s). Figure 2 shows a typical tracing of coronary artery bypass graft velocity. The maximal velocity is diastolic, as in normal coronary arteries. During systole, an early dip corresponds to the initiation of isovolemic contraction, followed by a positive forward flow during the rapid phase of aortic ejection. During the late

Protocol

Dobutamine was given intravenously via a central venous catheter used for no other purpose. Measurements were performed before (control) and after infusion of two successive doses of 5 and 10 \( \mu \)g \( \cdot \) kg\(^{-1} \cdot \) min\(^{-1} \) dobutamine, each step lasting 30 min. No inotropic or vasodilating drug was given prior to the study. Data were obtained in the Cardiac Surgical Intensive Care Unit, 6–8 h after the end of surgery, at a time when normothermia (central temperature higher than 37\(^\circ\) C, by pulmonary catheter), sinus rhythm, and stable hemodynamic status with adequate ventricular filling pressures were present. The lungs of all patients at this time were mechanically ventilated (CPU 1, Ohmeda, Maurepas, France) at a tidal volume of 10–12 ml/kg and a fractional inspired oxygen concentration (\( F_{1}\)\( \text{O}_{2} \)) of 35 ± 10% to achieve normal arterial blood gases.

FIG. 1. The pulsed Doppler microprobe. The ultrasonic incidence angle of the Doppler crystal and the vessel's red blood cells (\( \alpha \)) was exactly of 60\(^\circ\).

ultrasound speed in biologic tissues: \( t_{2} - t_{1} \), the difference between distal and proximal reception time corresponding to the vessel walls; and 60\(^\circ\), the incidence angle of the ultrasonic beam.\(^{13}\) Cross-sectional blood flow velocity (\( V_{m} \)) then can be measured within this diameter, and coronary bypass graft flow (\( Q_{m} \), ml/min) calculated according to the formula: \( Q_{m} = \pi \cdot \frac{D^{2}}{4} \cdot V_{m} \).

The accuracy of diameter determination was previously assessed by the comparison between actual diameters of calibrated latex tubes and apparent Doppler diameters.\(^{13}\) The error in diameter measurement with this technique is less than 8%, and the reproducibility averaged 5 ± 3%.\(^{13}\) For each patient, diameter measurements were performed in duplicate by two separate investigators. The difference between the values recorded by these investigators did not exceed 5%. The \( V_{m} \) recorded from the range-gated pulsed Doppler was the velocity of all the blood column, integrating the \( V_{m} \) profile. The linearity of the response for flow velocities ranging from 5 to 80 cm/s was systematically verified for each probe using an in vitro system.\(^{12}\)

FIG. 2. Data Analysis. From top to bottom: ECG; coronary bypass graft velocity (CBGV, cm/s); and systemic arterial pressure (SAP, mmHg). Data are recorded at low (5 mm/s) and then high (100 mm/s) paper speed. Systolic (\( T_{s} \), ms) and diastolic time intervals (\( T_{d} \), ms) defined with the ECG were used to measure systolic and diastolic CBGV's (see text for explanations). On the left part of the tracing (low paper speed), the modifications of CBGV's secondary to the systemic arterial pressure changes induced by the mechanical ventilation can be noted, illustrating the sensitivity of the method used.
were performed by two investigators. The intraobserver and interobserver measurement errors were less than 4 and 6%, respectively.

Using the area under the velocity curves and the cardiac intervals, we obtained cross-sectional systolic ($V_s$) and diastolic ($V_d$) coronary bypass graft velocities, and then $Q_s$ and $Q_d$ flows according to the formula: $Q_s = \pi \cdot D^2/4 \cdot V_s$ and $Q_d = \pi \cdot D^2/4 \cdot V_d$ in ml/min. Systolic ($f_Q_s$) and diastolic ($f_Q_d$) blood volumes entering the bypass during one cardiac cycle were calculated using phasic flow values and $T_s$ and $T_d$ respectively: $f_Q_s = Q_s \cdot T_s/60$ and $f_Q_d = Q_d \cdot T_d/60$, in milliliters.

Calculated hemodynamic parameters were: systemic vascular resistance index (SVRI, IU) = (MAP - RAP)/CI; stroke index (SI, ml x m$^{-2}$) = CI/HR; left ventricular stroke work index (LVSWI, g x m$^{-2}$) = 0.0136 x (MAP - PCWP) x SI; and rate-pressure product (RPP) = HR x SAP.

**Statistical Analysis**

Statistical analysis was by two way analysis variance and paired Newman-Keuls test for repetitive measurements in a same patient. A $P$ value of less than 0.05 was accepted as being statistically significant. Regression analysis studies were performed using the least squares method.

**Results**

No adverse effects were observed during dobutamine infusion and the protocol was successfully conducted in all patients. No arrhythmia or ST segment changes were observed, suggesting that significant myocardial ischemia did not occur.

**Systemic Hemodynamic Data**

No significant changes were observed following the first dose of 5 µg x kg$^{-1}$ x min$^{-1}$ of dobutamine (table 2).
From 5 to 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) of dobutamine, a 26\% significant increase in CI was found, secondary to a concomitant increase in HR and SI. These changes were obtained without any modification in PAP, PCWP or RAP which remained in a normal range. MAP increase resulted from a significant increase in DAP (+14\%) and SAP (+34\%), leading to an increased pulse pressure. As a consequence of these hemodynamic variations, SVRI decreased and RPP and LVSWI increased.

**Coronary Bypass Graft Hemodynamic Data**

Like the general hemodynamic data, no significant difference was found following the first dose of dobutamine. Table 3 shows the individual coronary flow data observed. The diameter of the coronary bypass graft (2.7 \pm 0.2 mm) did not change before or after inotropic stimulation. Despite the diversity in flows observed at the beginning of the study (this variability being mainly due to one patient with a high resting flow value), the increase in \( \dot{Q}_a \), following an increase of dobutamine from 5 to 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) was predominantly related to an increase in \( \dot{Q}_d \), whereas \( \dot{Q}_m \) remained unchanged (fig. 3). Although the increased HR reduced diastolic time, \( \dot{Q}_d \) and \( \dot{Q}_d \) supplied to the myocardium during one cardiac cycle were maintained (fig. 4).

A positive linear correlation was found between \( \dot{Q}_d \) and RPP (expressed as per cent of control values): \( r = 0.76, P < 0.001 \) (fig. 5). In order to ensure that this flow–RPP relationship was evidence of a physiologic variability induced by the inotropic stimulation, rather than the consequence of a biologic variability among a group of patients with different \( \dot{Q}_d \) and RPPs, the analysis given in figure 6 shows that the evolution of \( \dot{Q}_d \) and RPP during the inotropic stimulation followed a similar trend for all the patients. No correlation could be found between coronary bypass graft velocity and SI.

Finally, we determined the pressure–flow relationships during diastole between diastolic arterial pressure and coronary bypass graft flow for each situation (control, 5 and 10 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \) dobutamine). This direct comparison of pressure and flow revealed that a marked increase in \( \dot{Q}_d \) was observed despite a relatively small increase in end DAP (fig. 7).

**Discussion**

\( \dot{Q}_a \) and \( \dot{Q}_d \) of aortocoronary bypass have not been previously reported in the early postoperative period after an inotropic stimulation. After dobutamine, the increase in \( \dot{Q}_a \) resulted from both the maintenance of a significant bypass \( \dot{Q}_a \) and from a large increase in bypass \( \dot{Q}_d \). \( \dot{Q}_d \) was determined essentially by myocardial oxygen demand increase evaluated by the RPP. It is noteworthy that the oxygenated blood volume per beat entering the coronary vascular bed remained constant during dobutamine infusion, despite tachycardia. Within the limits of this study, we conclude that the aortocoronary saphenous bypass...
technique permits a volemic flow increase during the inotropic stimulation with dobutamine, which seems to be adapted to the increase in myocardial oxygen demand.

**CRITIQUE OF THE STUDY**

The technique that we used permits measurements of internal vessel diameter and instantaneous $V_m$. It is unable, however, to measure instantaneous variations in systolic–diastolic diameter. The diameter of the graft was assumed constant between systole and diastole. This assumption is in accordance with the results of Kajiyama et al. and with the fact that the saphenous graft is submitted to an arterial pressure that maximally stretches the vein during systole and diastole. At this pressure, the saphenous vein compliance would be very small, and systolic–diastolic variations of diameter negligible.

Fixation of the transducer on the vessel has at least three advantages in comparison with previous studies performed in open chest situations with a hand-held probe: 1) the probe follows the displacements of the graft during the cardiac cycle; 2) the ultrasonic incidence angle with the flow axis remains constant; and 3) postoperative measurements can be obtained. Flow was measured at the level of the bypass. The contribution of native coronary artery flow in our study was probably negligible since the left anterior descending coronary artery stenosis was more than 80%. Then, blood preferentially flowed through the lower resistive circuit, i.e., the saphenous graft. Assuming that the coronary bypass was not flow-limiting, we speculated that $Q_m$ represented the main blood supply of the dependent revascularized myocardium.

The delay between the end of surgery and the first measurements was chosen to minimize the influence of the surgery, since it has been shown that immediately after cardiopulmonary bypass, coronary vascular tone may be decreased, resulting in a linear relationship between pressure and flow. As a consequence, the effects of the inotropic stimulation on coronary flows might be misinterpreted since the autoregulatory properties of the coronary circulation are not recovered. Preoperative treatment also may affect the response to inotropic stimulation, since 5 of 10 patients were treated with β-adrenergic blocking agents. Although the time interval between the last oral dose (given with the preanesthetic medication) and the beginning of the study averaged 16 h, a significant response to the β-adrenergic stimulation was observed only after 10 μg·kg$^{-1}$·min$^{-1}$ dobutamine, a result suggesting that these patients might still have been slightly β-blocked.

Coronary vascular resistance was not calculated because it is difficult to know the exact back-pressure (the coronary critical closing pressure) and thus the true coronary driving pressure in clinical situations. This limitation also applies for the interpretation of the slopes of the coronary pressure–flow relationships. Since “none of the present formulations for computing coronary vascular resistance is perfect,” we directly compared the changes in pressure and flow. The relative status of the coronary vasculature was estimated by analysis of the overall effective input impedance of the coronary circulation before and after the inotropic stimulation.

Concerning metabolic aspects, myocardial oxygen consumption was not calculated since coronary sinus oxygen content ($C_{SO_2}$) was not measured. $C_{SO_2}$ content represents the results of global and not regional myocardial oxygen extraction. It is therefore impossible to differ-

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**FIG. 7. Schematic representation of dobutamine effects.** The mean values of pressure with the corresponding mean values of flow are represented during the control situation (A) and during 5 (B) and 10 μg·kg$^{-1}$·min$^{-1}$ of dobutamine (C). The significant increase in diastolic flow (QD) despite the modest increase (NS) in end diastolic arterial pressure (DAP) suggests an increase of metabolic demand with an upward shift of the coronary autoregulation curve.
entiate the revascularized territory from other myocardial areas not supplied by the graft. Finally, $\text{CSO}_2$ has been found unchanged during dobutamine infusion after cardiac surgery, suggesting that the induced myocardial oxygen demand increase was essentially supplied by a flow increase and not by higher oxygen extraction.

**Hemodynamic Effects of Dobutamine**

Inotropic stimulation was confirmed by the modifications in systemic hemodynamics observed during the two successive doses of dobutamine. These modifications were consistent with previously published data after cardiac surgery. Our patients had no evidence of heart failure, and CI increased, whereas RAP, PAP, and PCWP did not change significantly. This resulted from adequate ventricular filling pressures, which were controlled before the infusion of dobutamine by a prior fluid infusion when needed to avoid hypovolemia.

Considering coronary hemodynamics during dobutamine infusion, the increase in $Q_m$ confirmed previous reports on global coronary blood flow measured with thermodilution technique. The principal interest of this study, however, concerned the systolic-diastolic components of flow. Because dobutamine has primarily an inotropic effect, one could have predicted an increase in the extravascular intramyocardial forces during systole, compressing intramyocardial vessels and inducing a reduction in $Q_s$. The $Q_s$ impairment has been related to an increased intramyocardial pressure and to an increase in stiffness of the cardiac muscle surrounding the intramyocardial vessels. After the inotropic stimulation, the increase in systolic arterial pressure observed may account for a constant perfusion pressure during systole. One may hypothesize that the increase in systolic coronary inflow pressure helped in charging the intramyocardial capacitance of the coronary vessels, thus maintaining $Q_s$ despite a proportional increase in systolic intramyocardial resistance.

This relationship between pressure and $Q_s$ was also quantitatively important for $Q_m$, since $Q_s$ represented nearly 50% of $Q_m$, although considering inflow volumes instead of flows, $fQ_s$ was one fifth the volume of blood entering the coronary circulation per cardiac beat because of differences in systolic and diastolic intervals. For the clinician, it should be noted that during dobutamine infusion, the addition of a vasodilating drug that would not change the myocardial oxygen requirements must be monitored carefully in order to avoid an important decrease in blood pressure, which might induce a decrease in $Q_s$ that could affect $Q_m$.

Since the saphenous bypass is not directly submitted to the extravascular intramyocardial compressive forces, it may function as a systolic capacitor, as described by Chilian et al. for large epicardial arteries. A volume of blood may be stored during systole, and discharged in diastole, thus influencing the pattern of flow between the recipient coronary artery and the bypass graft.

Although differences have been found between the flow pattern characteristics of an internal mammary artery and a saphenous bypass graft, to our knowledge, the specific impact of the compliance of a graft on its velocity waveform has not been studied after an inotropic stimulation. In our patients, we considered that systolic-diastolic variations in bypass graft diameter were negligible, according to the observations of Kajiyama et al. Whereas the elastic and smooth muscle structures of the venous bypass graft are tiny compared to arterial vessels, one may suppose that the collagen structures of the saphenous graft were already maximally stretched by a SAP before the infusion of dobutamine, with a consequent negligible impact of the graft's compliance.

Like Vatner et al., who found in conscious dogs that "changes in late diastolic coronary flow and resistance paralleled the mean responses," in our study, $Q_d$ variations determined $Q_m$ and were related to the $\beta$ stimulation. After the inotropic stimulation, $Q_d$ increased almost three times more than did SAP. This suggests that the inotropic stimulation induced an upward shift of the coronary pressure-flow autoregulation curve. Our data do not permit differentiation of the exact mechanism of this action of dobutamine (increase in myocardial oxygen demand or direct pharmacologic coronary vasodilating effect). However, a metabolic explanation to the increased $Q_d$ may be suggested by the observed correlation between RPP and $Q_d$, and the well-known effect of dobutamine on myocardial oxygen demand. The three coronary pressure-flow points (fig. 7) confirm that the increase in myocardial oxygen consumption was followed by an increase in oxygen delivery. It is difficult, however, to state that this increase was sufficient, even if we found a positive correlation with RPP.

Even if dobutamine was chosen because of its usually relatively small effect on HR, it produced in our patients a substantial increase in HR that may affect the results observed. Since myocardial oxygen demand is ensured mainly by coronary flow-dependent oxygen supply and is determined phasically by each contraction, oxygen supply is also phasic and should pay the oxygen debt of the previous beat. The balance between myocardial oxygen supply and myocardial metabolic demand therefore could have been impaired by tachycardia, which increases oxygen demand and simultaneously decreases oxygen supply by decreasing the duration of diastole.

**Shirsiki H, Lee S, Hong YW, Jo YN, Strom JA, Goliner PL, Oka Y. Diagnosis of myocardial ischemia by the pressure-rate quotient and diastolic time interval during coronary artery bypass surgery. Journal of Cardiothoracic Anesthesia. 3: 592–596, 1989**

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since this parameter takes into account phasic energy requirement, \( Q_d \), and \( T_d \). We observed that despite tachycardia, this coronary "stroke volume" was maintained during the inotropic stimulation. This result suggests that after coronary revascularization, dobutamine did not affect the oxygen supply per beat, and thereby allowed the myocardium to contract without an energy deficit. Further studies are needed, however, to evaluate the impact of a decrease in the coronary blood volume per beat on the myocardial contraction.

We conclude that 6 h after coronary bypass graft surgery in humans, inotropic stimulation induced by 10 \( \mu g \cdot kg^{-1} \cdot min^{-1} \) dobutamine increased \( Q_m \). \( Q_m \) was held constant because the increase in SAP contributed to maintain coronary perfusion pressure in systole. \( Q_m \) increased in these patients with preserved global left ventricular function. This increase in \( Q_m \) seemed linked to the increase in myocardial oxygen demand. \( \int Q_m \) as well as \( \int Q_m \) entering the coronary bypass per cardiac beat were unmodified. Further studies are needed to quantitate the physiologic importance of this last phenomenon. Finally, the coronary systolic–diastolic blood supply was maintained in these patients during inotropic stimulation.

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