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Splanchnic oxygen transport after cardiac surgery: evidence for inadequate tissue perfusion after stabilization of hemodynamics

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Abstract *Objective:* To evaluate the adequacy of visceral oxygen transport and gastric pH_i after open heart surgery in patients with stable hemodynamics.
Design: Nonrandomized control trial.
Setting: A general intensive care unit in a tertiary care center.
Patients: Sixteen postoperative cardiac surgery patients were studied after stabilization of systemic hemodynamics.
Interventions: The effect of dobutamine infusion ($6\ \mu g\,kg^{-1}\,min^{-1}$) on systemic and regional oxygen transport was studied in ten patients, with six patients serving as controls. Systemic oxygen consumption was measured by indirect calorimetry and splanchnic and femoral blood flow, by continuous infusion of indocyanine green using regional catheters and gastric mucosal pH_i by gastric tonometer.
Measurements and results: Gastric mucosal acidosis was observed in half of the patients. Dobutamine increased cardiac output (3.2 ± 0.6 vs $4.4 \pm 0.7\ l \cdot min^{-1} \cdot m^{-2}$; $P < 0.05$), splanchnic blood flow (0.68 ± 0.28 vs $0.91 \pm 0.28\ l \cdot min^{-1} \cdot m^{-2}$; $p < 0.05$) and femoral blood flow (0.25 ± 0.08 vs $0.32 \pm 0.11\ l \cdot min^{-1} \cdot m^{-2}$; $p < 0.05$). Changes in splanchnic oxygen delivery and consumption were parallel in the two study groups. In response to dobutamine, gastric pH_i did not change (7.30 ± 0.08 vs 7.31 ± 0.06; NS), while in the control group, gastric pH_i tended to decrease (7.32 ± 0.04 vs 7.28 ± 0.06; NS). Systemic oxygen consumption increased in response to dobutamine (141 ± 11 vs $149 \pm 11\ ml \cdot min^{-1} \cdot m^{-2}$; $P < 0.05$) but did not change in the control group.
Conclusions: We conclude that a mismatch between splanchnic oxygen delivery and demand may be present despite stabilization of systemic hemodynamics after cardiac surgery. This is suggested by the parallel changes in splanchnic oxygen delivery and consumption. Dobutamine is likely to improve splanchnic tissue perfusion at this phase.

Key words Cardiac surgery · Dobutamine · Inotropes · Oxygen consumption · Regional blood flow · Splanchnic circulation

Introduction

Low cardiac output syndrome with tissue hypoperfusion is an infrequent but serious complication of coronary artery bypass surgery. Regional tissue hypoxia may develop despite apparently stable hemodynamics, as suggested by episodes of gastric mucosal acidosis in up to 50% of patients after cardiac surgery [1]. The increasing metabolic demand during the immediate postoperative period may increase the risk of tissue hypoxia [2]. We have recently demonstrated that immediately after coronary artery bypass surgery, the increasing metabolic demand is compensated for by a combination of increased oxygen extraction and blood flow, both in the whole body and in the splanchnic region, and that the regional oxygen extraction capability is well preserved [3]. The gastric mucosal pH ($=pH_i$) continues to decrease, reaching its nadir several hours postoperatively [4]. This suggests that a regional mismatch between oxygen delivery and demand may persist or develop after the stabilization of systemic hemodynamics.

To test this hypothesis, we evaluated the effects of dobutamine on systemic, splanchnic and femoral blood flow and oxygen consumption, and gastric pH_i postoperatively in coronary artery bypass patients after rewarming and stabilization of systemic hemodynamics had occurred in the intensive care unit. Dobutamine was selected because it increases total splanchnic blood flow and oxygen delivery after cardiac surgery [3].

Materials and methods

The study was approved by the Ethics Committee of the hospital and informed consent was obtained from each patient. Sixteen patients (13 male and 3 female) without preexisting hepatic disease or congestive heart failure were studied after coronary artery bypass grafting surgery (Table 1). The effects of dobutamine were studied in ten patients using data obtained before administration of dobutamine as control. A separate nonrandomized control group ($n = 6$) was included in order to evaluate spontaneous changes in hemodynamics and oxygen transport during the corresponding postoperative time period. The dobutamine group was studied before the control group. High-fentanyl anesthesia was used. Anesthesia was induced by means of 20 μg/kg of fentanyl, 0.07 mg/kg of midazolam and muscle relaxants, a mixture of alcuronium 0.125 mg/kg and pancuronium 0.15 mg/kg. It was subsequently maintained with continuous infusions of fentanyl (0.004 mg $kg^{-1}h^{-1}$), alcuronium (0.07 mg $kg^{-1}h^{-1}$) and midazolam (0.03 mg $kg^{-1}h^{-1}$), supplemented by thiopental and halothane. A membrane oxygenator (Sorin-Dideco, Compactflo DI703, Mirandola, Italy) with crystalloid priming and continuous flow was used for cardiopulmonary bypass. The pump flow rate was 2.25 l/m^2 at normothermia.

Table 1 Clinical data of patients receiving dobutamine ($n = 10$) and control patients ($n = 6$), presented as mean ± SD (NYHA class = New York Heart Association functional classification)

	Dobutamine	Control
Age (years)	54 ± 8	61 ± 4
NYHA class	2.8 ± 0.4	2.8 ± 0.4
Number of anastomosis	5.3 ± 1.0	4.8 ± 0.7
Time of cardiopulmonary bypass (min)	150 ± 32	114 ± 14*
Aortic occlusion (min)	111 ± 28	98 ± 12

*$p < 0.05$ for difference between the two groups (Mann-Whitney U-test)

In a separate study, five of the patients in the dobutamine group had received dobutamine for 90 min 3–5 h earlier. The baseline oxygen transport variables of these patients were compared with those of the rest of the patients and no significant differences were found (Mann-Whitney test). All studies were performed during controlled mechanical ventilation (Servo 900C, Siemens AB, Solna, Sweden) according to clinical protocol. The oxygen fraction of the inspiratory gas flow, FiO_2, was set at between 0.40 and 0.50 and a positive end-expiratory pressure of 5–6 cmH_2O was used in all of the patients studied. Oxygen consumption (VO_2) was measured continuously from the inspired and expired gases by open-circuit indirect calorimetry (Deltatrac™, Datex/Instrumentarium Corp., Helsinki, Finland). This device has been validated in this laboratory as well as by other researchers and it has a relative error of < 5% under the study conditions [5, 6]. Systemic oxygen consumption was calculated as the mean of 15 consecutive data points corresponding to the time of blood sampling during regional blood flow measurement. In this study, the coefficient of variation for the measurement of oxygen consumption calculated from the 15 repeated 1-min measurements was 2.3 ± 0.7%. Cardiac output was measured by the thermodilution technique in triplicate using 10 ml of room temperature saline; the mean value was used for calculations.

In addition to the radial and pulmonary artery catheters used for routine monitoring, catheters were inserted in the hepatic and femoral veins and the femoral artery. The hepatic vein was cannulated via the right internal jugular vein, and the correct position of the catheter was verified with fluoroscopy using a small amount of contrast dye. The splanchnic and leg blood flow was estimated by a primed (12-mg), continuous (1 mg/min), 30-min infusion of indocyanine green (Cardiogreen®, Becton-Dickinson Microbiology Systems, Cockeysville, Md., USA) [7, 8]. Indocyanine green was infused in the femoral artery. Blood was sampled for the measurement of femoral arterial and hepatic and femoral vein indocyanine concentrations after 20, 25, and 30 min of infusion. The indocyanine green concentrations were in steady state at each phase of measurement, as indicated by the coefficient of variation of 6.3 ± 5.6% for the three consecutive samples of each infusion. The indocyanine green extraction was 73.6 ± 14.4%, in each case exceeding the limit of 10% that is required for valid application of this method [9]. The coefficient of variation for the three consecutive blood flow measurements during each ICG infusion was 4.7 ± 4.0% and 5.9 ± 5.2% for the femoral and the splanchnic blood flow, respectively. Hemoglobin oxygen saturations were measured using a Cooximeter (IL 282, Instrumentation Laboratories, Lexington, Mass., USA). Oxygen delivery (DO_2) was measured as the product of thermodilution cardiac output and arterial oxygen content (CaO_2). CaO_2 was calculated as 1.39 × hemoglobin concentration × arterial oxygen saturation + dissolved oxygen. Regional DO_2 was calculated as the product of regional flow and CaO_2, and regional VO_2 as the product of the regional flow and the regional difference between CaO_2 and CvO_2.

Oxygen extraction was derived from the VO_2 obtained by indirect calorimetry and DO_2. Arterial plasma lactate concentrations were measured enzymatically (Boehringer Mannheim, Mannheim, Germany).

Gastric pH_i measurements were done using a gastric tonometer (Tonomitor, Tonometrics, Worcester, Mass., USA) [1]. The tonometer consists of a silicone balloon that is freely permeable to CO_2 and a sampling tube that is impermeable to CO_2. Following removal of air, the tonometer balloon was filled with 2.5 ml of normal saline at room temperature. The PCO_2 of the saline was determined and a simultaneous arterial blood sample was obtained to measure bicarbonate concentration. An IL-1302 blood gas analyzer (Instrumentation Laboratory, Lexington, Mass, USA) or an ABL-520 Blood Gas System (Radiometer A/S, Copenhagen, Denmark) was used to measure the PCO_2 of the saline. In the dobutamine group, the median PCO_2 of the saline was 5.22 kPa (range 4.04–6.42) and in the control group, 5.21 kPa (range 4.42–7.31). For each individual patient, all samples were analyzed using the same analyzer. For proper equilibration, a minimum time of 50 min was allowed before the saline sample was taken. During dobutamine infusion, the dose was kept constant for 50 min before samples for the pH_i measurement were taken. The Henderson-Hasselbalch equation was used to calculate the gastric mucosal pH_i, and a time-dependent correction factor for equilibration was used, as recommended by the manufacturer. The correct position of the tonometer was confirmed using fluoroscopy. H_2 receptor antagonists were not routinely given. In the present study, pH_i was also measured after induction of anesthesia in seven patients, none of whom received H_2 blockers, and it was found to be 7.45 ± 0.6 (range 7.38–7.55). These values are in agreement with observations in healthy subjects receiving ranitidine [10]. Based on the reproducibility of the saline PCO_2 measurements, a change in pH_i exceeding 0.05 pH-units was regarded as significant [11]. Gastric pH_i measurements were obtained from nine patients in the dobutamine group and from all six patients in the control group.

Protocol

The protocol is schematically shown in Fig. 1. After admission to the intensive care unit, crystalloids, blood and hydroxyethylstarch (mol. wt. 120,000, Plasmafusin, Leiras-Kabi, Helsinki, Finland) were infused to maintain the pulmonary artery occlusion pressure (PAOP) at the level that was judged clinically to be adequate for cardiac performance during the postperfusion period of the operation. When the patient's hemodynamics and peripheral circulation had stabilized (at the time when weaning from the ventilator normally would have been started), a 30-min measurement of hemodynamics, oxygen transport and regional blood flow was started. At the time of the baseline measurement, the mean arterial pressure, systemic vascular resistance index, pulmonary artery occlusion pressure and cardiac index were stable without any vasoactive medication or volume replacement and there was no evidence of peripheral vasoconstriction, as judged clinically and by measurement of peripheral skin temperature (Table 2). At the end of the

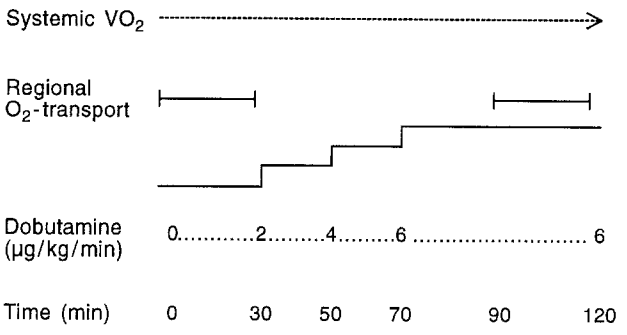

Fig. 1 Study protocol. In the control group, measurements were done at identical timepoints without vasoactive drug infusions

Table 2 Hemodynamics (mean ± SD) (*MAP* mean arterial pressure, *PAOP* pulmonary artery occlusion pressure, *SVRI* systemic vascular resistance index, *TC* central body temperature, *Tp* peripheral skin temperature)

		Baseline	Second measurement
Heart rate (beats · min^{-1})	Dobutamine	87 ± 6	111 ± 15**[bc]
	Control	83 ± 6	85 ± 8
MAP (mmHg)	Dobutamine	71 ± 11	73 ± 8
	Control	74 ± 13	72 ± 12
PAOP (mmHg)	Dobutamine	10 ± 2	7 ± 2
	Control	8 ± 3	8 ± 2
SVRI (dyne · s · cm^{-5} · m^{-2})	Dobutamine	1730 ± 480	1330 ± 320**[a]
	Control	1950 ± 430	1860 ± 350
Splanchnic vascular resistance (dyne · s · cm^{-5} · m^{-2})	Dobutamine	9130 ± 2300	7100 ± 2300*
	Control	8030 ± 3080	8150 ± 3780
Femoral vascular resistance (dyne · s · cm^{-5} · m^{-2})	Dobutamine	25340 ± 7820	19960 ± 6560**
	Control	26960 ± 10360	27210 ± 10150
CI (l · min^{-1} · m^{-2})	Dobutamine	3.2 ± 0.6	4.4 ± 0.7**[bc]
	Control	2.8 ± 0.4	2.9 ± 0.3
Tc (centigrade)	Dobutamine	38.5 ± 0.7	38.6 ± 0.5
	Control	38.4 ± 0.7	38.5 ± 0.6
Tp (centigrade)	Dobutamine	35.3 ± 2.0	36.6 ± 1.8**
	Control	34.8 ± 2.7	36.0 ± 2.5*

* $p < 0.05$; ** $p < 0.01$ for baseline vs second measurement
[a] $p < 0.05$ (group by time, analysis of variance)
[b] $p < 0.01$ (group by time, analysis of variance)
[c] $p < 0.01$ (difference between the groups, analysis of variance)

30-min period, a sample was obtained from the gastric tonometer and analyzed for the gastric pH_i while the tonometer balloon was again filled with saline. After the baseline measurement, dobutamine infusion was started and increased stepwise in increments of $2\ \mu g \cdot kg^{-1} \cdot min^{-1}$ at 20-min intervals to the maximum dose of $6\ \mu g \cdot kg^{-1} \cdot min^{-1}$. Six $\mu g \cdot kg^{-1} \cdot min^{-1}$ was chosen for the maximum dose based on our previous experience with the dosing of dobutamine after cardiac surgery [3]. When the maximum dose of dobutamine had been infused for 20 min, the measurements of the systemic and regional hemodynamics and oxygen transport were repeated. Saline was sampled from the tonometer and gastric mucosal pH_i measured at the end of the 30-min regional blood flow measurement period, i.e. after 50 min infusion of dobutamine with a constant dose. In the control group, measurements were made at identical timepoints without vasoactive drug infusions. Appropriate sedation and absence of shivering was confirmed by continuous monitoring at bed-side.

Statistical methods

Intragroup differences with respect to the two measurements were compared with the Wilcoxon matched pair test [12]. The effect of dobutamine and intergroup differences were compared by the analysis of variance [13] using one dependent variable (an oxygen transport or a hemodynamic variable), one grouping factor (use of dobutamine) and one within-subject factor (time). In order to test the presence of supply-dependence of oxygen consumption, the Wilcoxon matched pair test was used to compare the oxygen consumption corresponding to the minimum and the maximum oxygen delivery. Nonparametric tests were used because of the small size of the sample. Statistical significance was assumed at $p < 0.05$. All results are presented as mean ± SD.

Results

The recovery from the coronary artery bypass operation was uneventful except in one patient, who had mediastinitis and acalculous cholecystitis and needed prolonged ventilatory support and intensive care for 5 weeks.

Systemic hemodynamics and blood flow

In the dobutamine group, cardiac index, heart rate and peripheral skin temperature increased and systemic, splanchnic and femoral vascular resistance index decreased (Table 2, Figure 2). In patients receiving dobutamine, femoral blood flow increased by 35 ± 33% ($p < 0.05$) and splanchnic blood flow, by 44 ± 60% ($p < 0.05$; Fig. 2). The fractions of the femoral and splanchnic blood flow of the cardiac output did not change. In the control group, cardiac index did not change, and both the femoral and the splanchnic blood flow changes were variable (mean change in femoral flow 0 ± 16%, range −24% to +19% and in splanchnic flow 6 ± 36%, range −34% to +62% respectively).

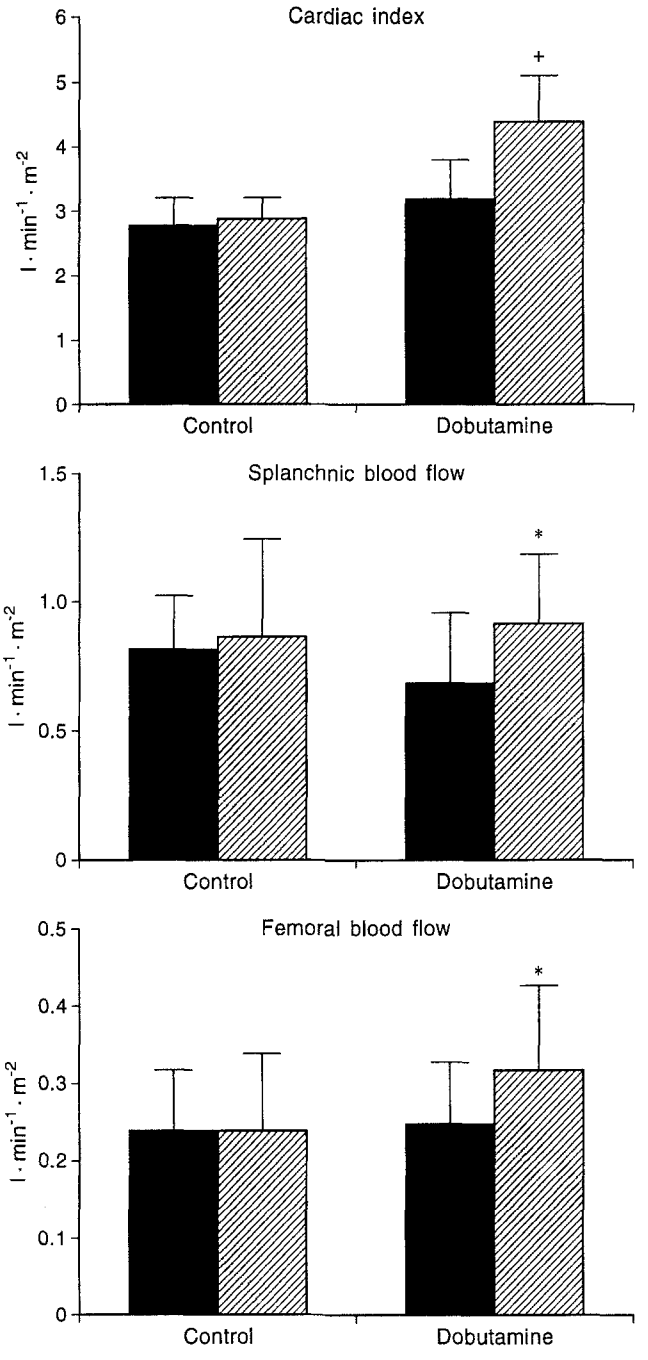

Fig. 2 Systemic and regional blood flow (mean ± SD). *Black column*, first measurement; *open column* second measurement. * $p < 0.05$, † $p < 0.01$ for difference from first measurement. Between the two groups, $P < 0.01$ for cardiac index and $p < 0.05$ for femoral blood flow (analysis of variance)

Systemic oxygen transport

Systemic oxygen delivery increased by 44 ± 17% ($p < 0.01$) and systemic oxygen consumption by 6 ± 5% ($p < 0.05$) in response to dobutamine, while

there were no significant changes in the control group (Table 3). The core temperature remained unchanged throughout the study period in both groups. The baseline oxygen consumption was lower in the control group ($p < 0.01$).

Femoral oxygen transport

Dobutamine increased femoral oxygen delivery in all except one patient, whereas femoral oxygen consumption did not change (Table 3). No significant changes were observed in either femoral oxygen delivery or consumption in the control group.

Splanchnic oxygen transport

In the dobutamine group, splanchnic oxygen delivery increased ($p < 0.05$) in all patients but one, in whom splanchnic blood flow decreased markedly with a concomitant major reduction in the splanchnic and whole body oxygen consumption (Table 3, Fig. 3). Changes in splanchnic oxygen consumption and delivery were parallel in nine of the ten patients receiving dobutamine (Fig. 3). Splanchnic oxygen consumption was higher at the higher splanchnic oxygen delivery ($p < 0.01$) in response to dobutamine. In the control group, changes in splanchnic oxygen delivery and consumption were parallel in all cases, and the actual direction of the spontaneous changes varied among individuals. In the control group, as well, splanchnic oxygen consumption was higher at the higher splanchnic oxygen delivery ($P < 0.05$). Whole body, splanchnic and femoral oxygen extraction decreased significantly in response to dobutamine, while in the control group, only systemic extraction decreased slightly.

At baseline, gastric mucosal acidosis, if defined as $pH_i < 7.32$, was present in half of all patients. In the dobutamine group, the mean gastric pH_i was 7.30 ± 0.08 at baseline measurement and 7.31 ± 0.06 during dobutamine infusion (NS) (Fig. 4). An increase in pH_i by more than 0.05 units was observed in two patients. In the control group, baseline gastric pH_i was 7.32 ± 0.04 and 7.28 ± 0.06 during the second measurement (NS) (Fig. 4). A decrease in pH_i by more than 0.05 pH units was observed in two patients.

Table 3 Oxygen transport responses (mean ± SD)

		Baseline	Second measurement
Systemic O_2 delivery ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	435 ± 74	$620 \pm 90^{**b}$
	Control	435 ± 70	465 ± 56
Systemic O_2 consumption ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	140 ± 10	$149 \pm 10^{*ac}$
	Control	126 ± 15	125 ± 17
Splanchnic O_2 delivery ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	99 ± 42	$135 \pm 35^{*}$
	Control	124 ± 28	139 ± 65
Splanchnic O_2 consumption ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	44 ± 14	51 ± 12
	Control	44 ± 10	48 ± 25
Femoral O_2 delivery ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	36 ± 12	$49 \pm 18^{*}$
	Control	38 ± 14	40 ± 21
Femoral O_2 consumption ($ml \cdot min^{-1} \cdot m^{-2}$)	Dobutamine	10 ± 2	10 ± 2
	Control	10 ± 2	11 ± 3
Systemic O_2 extraction (%)	Dobutamine	33 ± 5	$24 \pm 3^{**b}$
	Control	29 ± 5	$27 \pm 4^{*}$
Splanchnic O_2 extraction (%)	Dobutamine	46 ± 8	$38 \pm 6^{**ad}$
	Control	36 ± 5	35 ± 6
Femoral O_2 extraction (%)	Dobutamine	30 ± 8	$23 \pm 7^{*a}$
	Control	29 ± 12	29 ± 8
Arterial lactate (mmol/l)	Dobutamine	1.65 ± 0.63	1.62 ± 0.66
	Control	1.82 ± 0.29	1.89 ± 0.35

* $p < 0.05$; ** $p < 0.01$ (baseline vs second measurement)
[a] $p < 0.05$ (group by time, analysis of variance)
[b] $p < 0.01$ (group by time, analysis of variance)
[c] $p < 0.01$ (difference between the groups, analysis of variance)
[d] $p < 0.05$ (difference between the groups, analysis of variance)

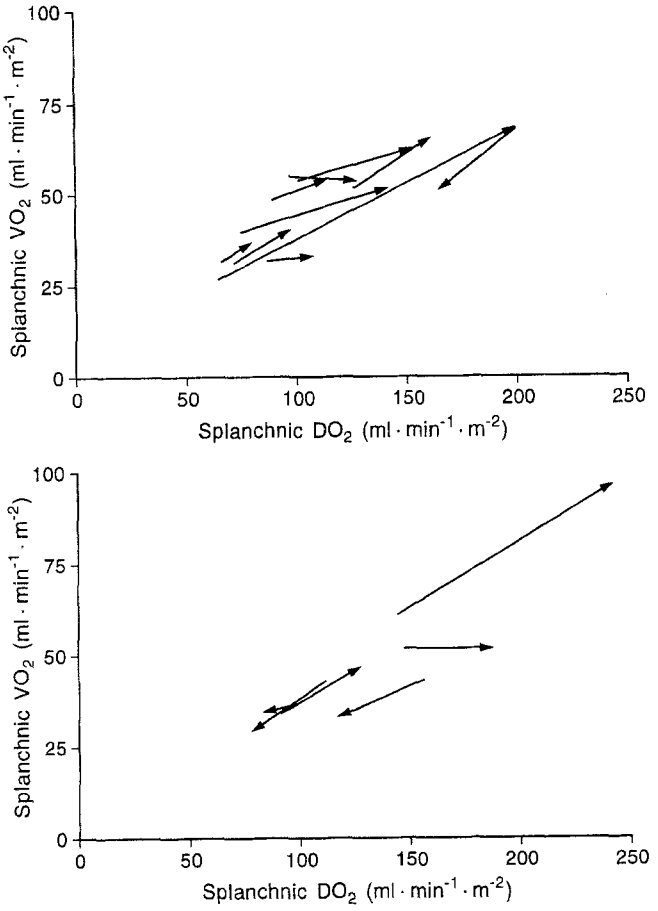

Fig. 3 Relationship between changes in splanchnic oxygen delivery and consumption in patients receiving dobutamine (*up*) and in controls (*down*)

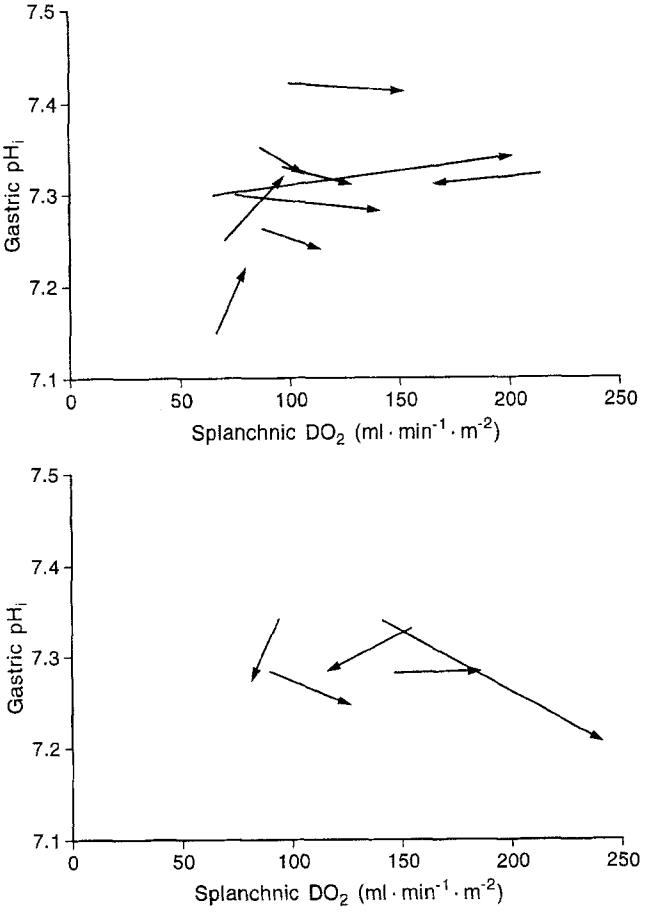

Fig. 4 Relationship between changes in splanchnic oxygen delivery and gastric mucosal pH_i in patients receiving dobutamine (*up*) and in the controls (*down*)

Discussion

There are two main findings in this study. First, changes in splanchnic oxygen delivery and oxygen consumption both in patients receiving dobutamine and in the controls were parallel. Second, depending on what is regarded as normal pH_i, gastric mucosal acidosis was present in one third or half of the patients at the baseline, despite stable systemic hemodynamics. The gastric mucosal acidosis and the apparent flow dependence of splanchnic oxygen consumption in both study groups supports the hypothesis that a mismatch between splanchnic oxygen delivery and demand was present.

The observed changes in the systemic and, in some cases, also in the regional oxygen consumption were small and therefore susceptible to methodological errors. The gas exchange monitor has been validated by our group and several other groups of researchers, and it has a bias of less than 5% when FiO_2 is 0.60 or less, as in this study [5, 6]. The detection of small changes in oxygen consumption necessitates slight variability between repeated measurements. In the present study, the coefficient of variation between repeated measurements of oxygen consumption was sufficiently small ($2.3 \pm 0.7\%$) to allow for the detection of the small changes induced by dobutamine. A steady-state dye concentration and sufficient hepatic extraction of the indocyanine green dye, both prerequisites for accurate measurement of splanchnic blood flow, were confirmed. The coefficient of variation for consecutive measurements of splanchnic blood flow was $5.9 \pm 5.2\%$, which is better than is usually obtained for cardiac output by the thermodilution method [14]. Factors which may influence whole-body oxygen consumption, such as shivering, levels of analgesia and sedation, changes in body temperature, and routine intensive care procedures, were carefully controlled during the study [15, 16]. The regional oxygen

transport measurements are susceptible to similar mathematical coupling to calculated oxygen consumption used in the assessment of global oxygen consumption/delivery [17]; therefore, small changes in regional oxygen transport should be interpreted with caution. In the present study, when minor ($< 15\%$) changes are excluded, both oxygen delivery and consumption in the splanchnic region changed in seven of the ten patients receiving dobutamine and in four of the six patients in the control group, and in each case the changes were parallel. In contrast, parallel changes in splanchnic oxygen delivery and consumption were not observed either in response to dobutamine or in controls when studied several hours earlier after cardiac surgery [3].

The changes in gastric mucosal pH_i were small in most patients and within the range of variability of the method of measurement. According to a study with healthy volunteers, detection of borderline changes in pH_i might have been improved if H_2 receptor antagonists had been used [10]. However, in two recent studies done in critically ill patients, H_2 antagonists had no influence on pH_i values and it was concluded that their use is not necessary in patients [18, 19].

The two alternatives to the findings in this study are the metabolic effects of dobutamine and possible spontaneous metabolic changes. Dobutamine is known to have a thermogenic effect, which in healthy subjects may increase the systemic oxygen consumption by 20–25% [20, 21]. It is unlikely that the thermogenic effect could explain the findings in this study, since the increase in oxygen consumption in the splanchnic region was the main component of the change in the whole body oxygen consumption. While the catecholamines presumably exert their thermogenic effect primarily in the skeletal muscle [20], no changes in femoral oxygen consumption were observed. We suggest that the thermogenic response to dobutamine may have been reduced, presumably due to the concomitant catecholamine response to the surgical trauma [21, 22]. Spontaneous metabolic changes are unlikely to explain the changes in the dobutamine group, since there were no changes in systemic oxygen consumption in the control group.

In the control group, gastric mucosal pH_i either decreased or remained stable, whereas in the dobutamine group, pH_i either increased (suggesting improved perfusion) or remained stable. In patients receiving dobutamine, there are at least two factors that may explain why low gastric mucosal pH_i was not completely corrected. First, it is possible that there was no splanchnic tissue hypoxia in these patients and the splanchnic oxygen delivery-consumption covariance is physiologic. However, if one assumes that gastric tonometry reflects gastric mucosal perfusion and that low gastric mucosal pH_i actually reflects mucosal hypoxia, this explanation is unlikely. Second, the increase in splanchnic oxygen delivery may have been too small or too brief to correct the splanchnic oxygen delivery-demand mismatch. In contrast to the findings in this study, we earlier found dobutamine to induce gastric mucosal acidosis, despite increasing splanchnic blood flow, when infused immediately after cardiac surgery [23]. In addition, dopexamine, when infused immediately after cardiac surgery, decreases gastric mucosal pH_i despite increased splanchnic blood flow [24]. In a recent article by Gutierrez et al., when dobutamine was infused to septic patients to increase systemic oxygen delivery, gastric mucosal pH_i increased, suggesting improvement of regional tissue hypoxia [25]. In the control group in this study, the tendency of the gastric mucosal pH_i to decrease further probably reflects an insufficient response of splanchnic oxygen delivery to increased splanchnic oxygen demand. As some of the patients in both study groups had relatively low pulmonary artery occlusion pressures, it is possible that more aggressive volume support would have improved gastric mucosal perfusion in those patients.

The lack of randomization is a limitation of this study. Although the study's main finding is the parallel changes in splanchnic oxygen delivery and consumption and the low gastric mucosal pH_i in both study groups, the difference in baseline systemic oxygen consumption and in cardiopulmonary bypass time between the groups may somewhat limit comparison between the groups. However, we did not find any correlation between bypass time and gastric mucosal pH_i in these patients.

Our results are in agreement with those of previous studies demonstrating episodes of gastric mucosal acidosis in up to 50% of patients after cardiac surgery and progressive decrease in gastric mucosal pH_i, until a minimum is reached 5 h after operation [1, 4]. Landow et al. found a correlation between gastric pH_i and hepatic venous oxygen saturation after elective cardiac surgery in the immediate postoperative period, suggesting an imbalance between splanchnic oxygen delivery and metabolic needs [26]. The present study demonstrates that the risk for splanchnic tissue hypoxia persists postoperatively even after hemodynamics and peripheral circulation have stabilized. The lack of increased systemic lactate levels does not contradict the hypothesis of the mismatch between splanchnic oxygen delivery and demand. If liver function is well preserved, as suggested by the high indocyanine green extraction, increased lactate production from the splanchnic organs does not necessarily induce systemic hyperlactatemia.

In most of the patients, the postoperative course was uneventful. The clinical relevance of a short-term mismatch between splanchnic oxygen delivery and demand may be small, although this cannot be concluded due to the small number of patients in the present study. Any deterioration of systemic hemodynamics at this phase will substantially increase the risk of severe splanchnic tissue hypoxia. Use of dobutamine in hemodynamic support is also likely to have favorable effects in splanchnic tissue perfusion at this phase of postoperative recovery.

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