Oxygen Delivery and Consumption in the Perioperative Period of Coronary Artery Bypass Grafting without Blood Transfusion

Akio MIZUSHIMA and Yoshinari NIIMI

The perioperative changes in relationship between oxygen delivery ($\dot{D}_{O_2}$) and oxygen consumption ($\dot{V}_{O_2}$) were examined in forty patients who underwent coronary artery bypass grafting (CABG) without blood transfusion. Hemodilution was performed to maintain hematocrit of 19.2 ± 1.8% during cardiopulmonary bypass (CPB). Hemodynamic and metabolic parameters were measured in four stages; before CPB (stage I), after CPB (stage II), after ICU arrival (stage III), and the following day (stage IV). In each stage, there was a strong positive correlation between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$. In stage I, a decrease in $\dot{D}_{O_2}$ was met with low $\dot{V}_{O_2}$, and there was no imbalance between them ($r = 0.67$, $P < 0.01$). $\dot{V}_{O_2}$ increased significantly in stage II, and this increased $\dot{V}_{O_2}$ was compensated by an increase in $\dot{D}_{O_2}$ sufficiently to meet tissue oxygen demand ($r = 0.59$, $P < 0.01$). In stage III and IV, the increases in tissue oxygen requirements were met by increases in oxygen extraction ratio ($r = 0.81$, $P < 0.01$, $r = 0.60$, $P < 0.01$, respectively) reflected in lowered mixed venous oxygen tension and saturation. From these results, it is assumed that the adequate relationship between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ can be maintained in the perioperative period of CABG without blood transfusion. (Key words: oxygen consumption, oxygen delivery, coronary artery bypass grafting)

(Mizushima A, Niimi Y: Oxygen Delivery and consumption in the perioperative period of coronary artery bypass grafting without blood Transfusion. J Anesth 4: 313–318, 1990)

Cellular function is depending upon adequate supply of oxygen to meet metabolic requirement, and in the perioperative period, it is extremely important to maintain the balance of oxygen demand and supply in the whole body[1–3]. In patients under surgical stress, the oxygen consumption ($\dot{V}_{O_2}$) is maintained properly by appropriate oxygen delivery ($\dot{D}_{O_2}$), which is regarded as the determinant factor for the patient's survival[4]. Although a few studies have reported the relationship between $\dot{D}_{O_2}$ and $\dot{V}_{O_2}$ during anesthesia in open heart surgery, the results are contradictory[5–8]. Furthermore, no reports have discussed the safety of open heart surgery without blood transfusion from the aspects of oxygen delivery and consumption. Accordingly, we investigated the perioperative changes in relationship between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ in patients who underwent coronary artery bypass grafting (CABG) without blood transfusion.

Department of Anesthesiology, Juntendo University School of Medicine, Tokyo, Japan

Address reprint requests to Dr. Mizushima: Department of Anesthesiology, Juntendo University School of Medicine, 2-1-1, Hongo, Bunkyo-ku, Tokyo, 113 Japan

Materials and Methods

This study was performed on forty patients who underwent CABG for treating angina pectoris and/or old myocardial infarction (table 1). Blood transfusion was not

Table 1. Patient characteristics

Sex	(male : female)	38 : 2
Age	(yr)	58.1 ± 8.7
Body height	(cm)	162.9 ± 6.2
Body weight	(kg)	64.8 ± 8.9
Body surface area	(m^2)	1.70 ± 0.14
Initial dose of fentanyl	($\mu g \cdot kg^{-1}$)	35.2 ± 6.1
Total dose of fentanyl	($\mu g \cdot kg^{-1}$)	52.9 ± 7.9
Duration of operation	(min)	373.8 ± 71.5
Duration of anesthesia	(min)	445.9 ± 69.5
Duration of aortic cross clamping	(min)	82.8 ± 30.2
Duration of cardiopulmonary bypass	(min)	144.3 ± 46.9
Number of grafts		1.9 ± 0.7
Cases of inotropic support		14

(n=40, mean ± SD)

performed throughout the perioperative period. All patients were evaluated as NYHA Classification II, and patients with serious coexisting diseases and with poor cardiac function (less than 35% of ejection fraction) were excluded from the study.

Preanesthetic medication consisted of diazepam 0.1 mg p.o. (max; 5 mg) and morphine 0.2 mg i.m. (max; 10 mg). Before anesthesia, all patients received their customary dose of cardiac medications, including nitrate, beta-adrenergic blockers, and calcium channel antagonists.

Upon arrival in the operating room, venous and arterial (radial artery) catheters were inserted under local anesthesia. Anesthesia was induced with diazepam 0.1 ~ 0.2 $mg \cdot kg^{-1}$ and fentanyl 25 ~ 50 $\mu g \cdot kg^{-1}$ and maintained with nitrous oxide (0 ~ 3 $l \cdot min^{-1}$) - oxygen (6 ~ 3 $l \cdot min^{-1}$) - enflurane (0 ~ 2.0%), and additional doses of fentanyl were administered as needed. Endotracheal intubation was facilitated with pancuronium 0.1 ~ 0.15 $mg \cdot kg^{-1}$. A Swan-Ganz catheter (Edwards Laboratories, 93A-131-7F) was placed in the pulmonary artery via the internal jugular vein and connected with the cardiac output computer (Edwards Division Co., COM-1). Patients were mechanically ventilated to maintain arterial carbon dioxide tension (Pa_{CO_2}) between 30 and 40 mmHg. In order to prevent intraoperative myocardial ischemia, nitroglycerin 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was administered continuously after induction of anesthesia in all patients.

During CPB, membrane oxygenator with a non-pulsatile pump flow of 2.2 $l \cdot m^{-2} \cdot min^{-1}$ was used. Hemodilution was performed to maintain the minimum hematocrit (Ht) of 20 (19.2 ± 1.8)% and the minimum rectal temperature was maintained between 25 and 30°C according to the duration of aortic cross clamping. The myocardium was protected by using cardioplegic solution and topical cooling with ice slush.

After the termination of CPB, all diluted blood remaining in the circuit was concentrated to increase Ht to 42.8 ± 4.4% by using a hemofilter (Asahi Medical Co., Plasmaflo AP-08H), and returned to the systemic circulation[9]. Dobutamine 2 ~ 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was administered for a low cardiac output state.

Postoperatively, the patients were transfered to the intensive care unit (ICU) and their ventilation was controlled mechanically with diazepam and/or morphine as required. On the first postoperative day, after cofirming adequate spontaneous respiration, endotracheal tubes were extubated and subsequently administered 35 ~ 50% oxygen by a Venturi mask.

Heart rate (HR), mean arterial pressure (mAP), mean pulmonary arterial pressure (mPAP), pulmonary capillary wedge

Table 2. Changes in hemodynamics and oxygen transport

		Stage I	Stage II	Stage III	Stage IV
HR	(beat·min^{-1})	56.9 ± 12.0	83.1 ± 11.8*	86.2 ± 12.2*	81.3 ± 10.5*
mAP	(mmHg)	78.2 ± 10.1	73.2 ± 10.4	83.6 ± 10.6#	82.8 ± 11.8#
mPAP	(mmHg)	12.3 ± 3.1	15.3 ± 4.0*	12.2 ± 4.2#	12.7 ± 5.2#
PCWP	(mmHg)	9.4 ± 2.6	12.0 ± 3.7*	6.4 ± 3.7*#	8.8 ± 5.1#$
CVP	(mmHg)	5.2 ± 1.7	7.2 ± 1.8*	5.3 ± 2.0#	6.3 ± 2.4
CI	(l·min^{-1}·m^{-2})	1.66 ± 0.31	2.81 ± 0.50*	2.28 ± 0.51*#	2.51 ± 0.53*$
SVI	(ml·beat^{-1}·m^{-2})	29.9 ± 5.5	33.5 ± 6.5*	27.0 ± 6.2#	31.1 ± 6.4$
LVSWI	(g·m·m^{-2})	27.9 ± 6.8	27.7 ± 6.1	28.0 ± 6.8	31.8 ± 9.4
SVRI	(dyne·sec·cm^{-5}·m^2)	3610 ± 715	1942 ± 499*	2908 ± 878*#	2562 ± 633*#$
PVRI	(dyne·sec·cm^{-5}·m^2)	149 ± 91	108 ± 77	217 ± 119*#	137 ± 94$
Hb	(g·dl^{-1})	12.6 ± 1.4	8.2 ± 1.2*	10.9 ± 1.3*#	9.8 ± 1.4*#
Ht	(%)	39.1 ± 3.6	19.9 ± 2.0*	32.6 ± 4.4*#	30.8 ± 4.2*#
Rectal temperature	(°C)	36.4 ± 0.6	36.0 ± 0.7	37.0 ± 0.9#	37.5 ± 0.7*#
$P\bar{v}_{O_2}$	(mmHg)	49.1 ± 5.1	50.8 ± 7.5	39.2 ± 4.7*#	36.1 ± 3.9*#$
$S\bar{v}_{O_2}$	(%)	83.2 ± 5.1	82.9 ± 5.1	72.1 ± 4.5*#	66.9 ± 6.4*#$
$\dot{D}_{O_2}$	(ml·min^{-1}·m^{-2})	297.5 ± 68.0	336.9 ± 70.1*	353.1 ± 94.5*	324.5 ± 75.7*
$\dot{V}_{O_2}$	(ml·min^{-1}·m^{-2})	59.7 ± 12.6	76.9 ± 16.7*	100.3 ± 25.5*#	101.6 ± 20.8*#
OER	(%)	20.7 ± 4.6	23.4 ± 4.9*	28.9 ± 4.8*#	32.1 ± 6.7*#$

(mean ± SD)

Stage I: 30 minutes after induction of anesthesia and before initiation of CPB,
Stage II: 30 minutes after termination of CPB,
Stage III: 3 hours after arrival in ICU,
Stage IV: 6 hours after extubation on the first postoperative day
Paired t-tests: *; $P < 0.01$ versus stage I, #; $P < 0.01$ versus stage II, $; $P < 0.01$ versus stage III.

pressure (PCWP), central venous pressure (CVP) and cardiac output (CO) were measured. Cardiac index (CI), stroke volume index (SVI), left ventricular stroke work index (LVSWI), systemic vascular resistance index (SVRI) and pulmonary vascular resistance index (PVRI) were calculated by the standard formulae[10]. Hemoglobin content (Hb), Ht and rectal temperature were measured. Blood gas analysis was carried out on arterial and mixed venous blood samples (Radiometer Co., ABL2 Acid-Base Laboratory). $\dot{D}_{O_2}$, $\dot{V}_{O_2}$ and the oxygen extraction ratio (OER) were calculated from the standard formulae[8,10].

These measurements were performed at the following four stages; 1) thirty minutes after induction of anesthesia and before the initiation of CPB (stage I), 2) thirty minutes after termination of CPB (stage II), 3) three hours after arrival in ICU (stage III), and 4) six hours after extubation on the first postoperative day (stage IV). The correlation between $\dot{D}_{O_2}$ and $\dot{V}_{O_2}$ was analyzed with the regression line obtained by the least square method. These measured values were compared in each group. Student's t-test was utilized for a statistical significance , and P-value less than 0.01 was considered significant.

Results

The results are summarized in table 2.

HR and CI were in low level in stage I, but increased to an almost stable value in stage II, III, and IV. CVP, PCWP, and mPAP were increased significantly in stage II, but these parameters returned to the level of stage I in stage III and IV. A significant decrease in SVRI was observed in stage II. Other hemodynamic valiables were not changed significantly in any stage.

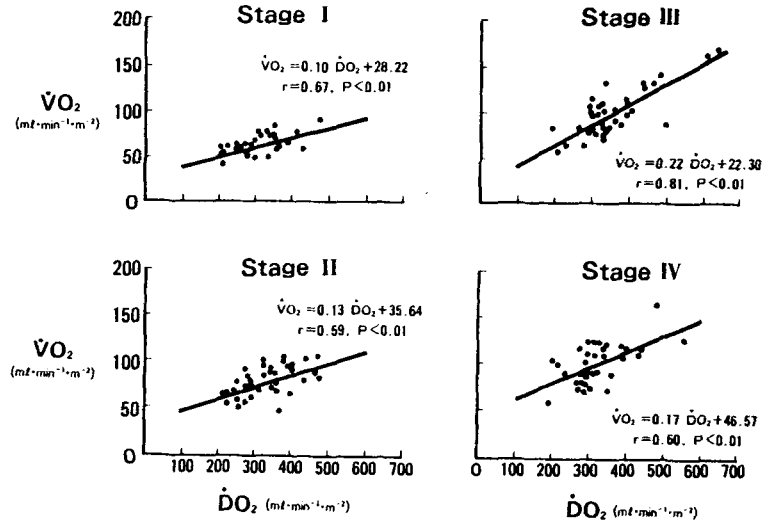

Fig. 1. Relationship between $\dot{D}_{O_2}$ and $\dot{V}_{O_2}$ at each stage.

Hb and Ht decreased significantly in stage II and recovered partially after autotransfusion.

$\dot{D}_{O_2}$ showed the lowest value in stage I and increased markedly in stage II, and then maintained the same level in stage III and IV. $\dot{V}_{O_2}$ and OER also showed the lowest values in stage I, and there was a tendency to increase gradually from stage I to stage II, III, and IV.

The correlations between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ in the four stages are shown in figure 1. Regression equations, correlation coefficients, and *P*-values in four stages were as follows: stage I; $\dot{V}_{O_2} = 0.10\ \dot{D}_{O_2} + 28.23$, r = 0.67, $P < 0.01$, stage II; $\dot{V}_{O_2} = 0.13\ \dot{D}_{O_2} + 35.64$, r = 0.59, $P < 0.01$, stage III; $\dot{V}_{O_2} = 0.22\ \dot{D}_{O_2} + 22.30$, r = 0.81, $P < 0.01$, and stage IV; $\dot{V}_{O_2} = 0.17\ \dot{D}_{O_2} + 46.57$, r = 0.60, $P < 0.01$. The range of $\dot{D}_{O_2}$ values were essentially the same in these four stages, but $\dot{V}_{O_2}$ showed the lowest in stage I and apparently higher values in stage II, III, and IV.

Discussion

In the past, open heart surgery usually required a large quantity of blood transfusion. However, since 1962, when Cooley et al.[11] succeeded in open heart surgery without blood transfusion in a Jehovah's Witness patient, the progress of the hemodilution method and re-use of blood remaining in the CPB circuit made it possible to perform CPB without blood transfusion safely. It is well known that surgery without blood transfusion has various advantages. However, it is also known that the method have complications such as pulmonary edema, hypoxia, postoperative anemia, and a decrease in $\dot{D}_{O_2}$[12,13]. Therefore, in open heart surgery without blood transfusion, it is extremely important to maintain a stable hemodynamics and the balance between oxygen demand and supply in vital organs, including the heart.

Gump et al.[1] pointed out that serial measurements of caloric expenditure and $\dot{V}_{O_2}$ were useful in the evaluation of therapeutic effects on circulatory failure. Seki et al.[2] also reported that the goal of the treatment for low output syndrome (LOS) was not to increase in CO by inotropic agents, but to maintain or improve the tissue metabolism judging from $\dot{V}_{O_2}$.

Although the normal values of $\dot{D}_{O_2}$, $\dot{V}_{O_2}$, and OER vary with individual condition, those in a healthy person at rest are reported to be 500 ~ 650 ml·min^{-1}·m^{-2}, 100 ~ 150 ml·min^{-1}·m^{-2}, and 20 ~ 25%, respectively[1,2,10,14]. When $\dot{D}_{O_2}$ is decreased by anemia, hypoxemia, or LOS, $\dot{V}_{O_2}$ is main-

tained within normal range by many compensatory mechanisms such as an increase in OER, a selective increase in blood flow to the vital organs, peripheral vasodilation, and right-shift of the oxgen-Hb dissociation curve. However, in critical conditions such as in adult respiratory distress syndrome[3,15], septic shock[3,16], and brain death[3], $\dot{V}_{O_2}$ decreases along with $\dot{D}_{O_2}$, showing that a human body behaves as the "oxygen conformer"[3]. Therefore, the aim of treatments in those patients is to maintain an adequate level of $\dot{D}_{O_2}$.

In general, $\dot{V}_{O_2}$ during general anesthesia seems to be decreased by 10 ~ 25% from that while wake and rest[6]. However, a very few studies have been reported concerning the relationship between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ during open heart surgery, and those results are controversial.

Shibutani et al.[6,7] reported in their study of anesthetized man that critical values of $\dot{D}_{O_2}$ were identified to be 330 $ml \cdot min^{-1} \cdot m^{-2}$ before CPB and 330 $ml \cdot min^{-1} \cdot m^{-2}$ after CPB respectively, and $\dot{V}_{O_2}$ reached a plateau, the values of which were 109 ± 16 $ml \cdot min^{-1} \cdot m^{-2}$ before CPB and 106 ± 13 $ml \cdot min^{-1} \cdot m^{-2}$ after CPB. However, in this study, significant positive correlations were found between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$. Although $\dot{D}_{O_2}$ ranged from approximately 200 to 500 $ml \cdot min^{-1} \cdot m^{-2}$ at each stage, there were no distinct critical values of $\dot{D}_{O_2}$. The reasons of discrepancy between this study and their one are not clear, but could be attributing to the differences in anesthesia, CPB technique, surgical procedures, and patient conditions.

$\dot{D}_{O_2}$ in stage I in this study showed a markedly lowered value (297.5 ± 68.0 $ml \cdot min^{-1} \cdot m^{-2}$) compared to the normal value. The value of $\dot{V}_{O_2}$ was also the lowest in stage I. In this situation, there was no patient showing a marked decrease in urine volume or the development of metabolic acidosis. Moreover, OER was low and the regression line showed the slightest gradient. These results suggest that the balance between oxygen demand and supply was sufficiently maintained in spite of the lowered value of $\dot{D}_{O_2}$.

There was no significant changes in mixed venous oxygen tension ($P\bar{v}_{O_2}$) and mixed venous oxygen saturation ($S\bar{v}_{O_2}$) between stage I and II, and the regression line in stage II showed a parallel shift upward with almost the same gradient as that in stage I. $\dot{V}_{O_2}$ significantly increased in the stage II and this increase in $\dot{V}_{O_2}$ was compensated by an increase in $\dot{D}_{O_2}$ sufficiently to meet tissue oxygen demand. This compensation could mainly attribute to a significant increase in CO with a marked decrease in SVRI. These results clearly show that the normal relationship between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ was maintained in the period immediately after CPB with hemodilution method.

In stage III, $\dot{D}_{O_2}$ was increased significantly mainly by an increase in Hb, in spite of relative hypovolemia due to rapid diuresis and peripheral vasodilation. However, a more marked increase in $\dot{V}_{O_2}$ resulted in a significant decrease in $P\bar{v}_{O_2}$ and $S\bar{v}_{O_2}$, and an increase in OER. The regression line showed the steepest gradient at this stage. From these results, it is assumed that, in stage III, the increased tissue oxygen requirements were met by a compensatory increase in oxygen extraction which was reflected in lowered $S\bar{v}_{O_2}$.

The pattern of regression line in stage IV was similar to that of stage III, but OER was further increased. The fact that $\dot{V}_{O_2}$ under spontaneous ventilation was increased by 4 ~ 10% compared to that during artificial ventilation[6,17] was probably one of the causes of further increasing OER at this stage. Forty percent of OER is believed to be the compensatory limit of oxygen utilization[18] and the value of OER in stage IV was barely within the safe limit, but a great caution have to be paied to maintain an adequate $\dot{D}_{O_2}$ at this stage. We did not continue this study after stage IV, but since a moderate anemia remained for several postoperative days, the same caution as in stage IV should be taken during the following ICU period.

In conclusion, in the perioperative period of CABG without blood transfusion, the relationship between $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ showed

significant positive correlations and no critical values in $\dot{D}_{O_2}$ were seen. Post-CPB management without blood transfusion could reasonably maintain the oxygen delivery to meet tissue oxygen demand. Because $\dot{V}_{O_2}$ and $\dot{D}_{O_2}$ are supposed to be modified with many factors such as anesthesia techniques, administeration of catecholamines, and body temperature, the more detailed studies are needed for establishing a rational approach in management of CABG without blood transfusion.

(Received May 30, 1989, accepted for publication Nov. 28, 1989)

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