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Whole body oxygen consumption during aortic cross clamping and declamping in dogs anesthetized with isoflurane

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Hemodynamic and metabolic changes after aortic cross clamping and declamping of the abdominal aorta were studied in dogs under isoflurane anesthesia. During aortic cross clamping, cardiac output, whole body oxygen consumption, and saphenous vein O_2 saturation decreased significantly ($p<0.05$) and arterial pressure, pulmonary arterial pressure, pulmonary arterial wedge pressure, and systemic vascular resistance increased ($p<0.05$). After aorta declamp arterial pressure, systemic vascural resistance and pulmonary arterial wedge pressure was decreased, although these changes were not significant to compare the pre-aortic clamping value. The correlation between percent change in cardiac output and oxygen consumption was poor after aorta clamping and declamping ($r=0.35$ $p>0.05$). The cause of the decrease in cardiac output which followed aortic clamping is not due to the changes of the oxygen consumption and partially explained by depression of myocardial performance. There was no relationship between the degree of reduction of arterial pressure after aortic declamping and O_2 debt of the ischemic tissue during aortic cross clamping ($r=0.40$ $p>0.05$). This means O_2 debt calculated from oxygen consumption can not reflect the degree of the reduction of arterial pressure after aortic declamping.

Key words: Anesthesia, vascular surgery, Complication, Oxygen consumption

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Introduction

Aortic cross clamping is sometimes applied during vascular surgery. Aortic cross clamping and declamping causes an increase and a decrease in mean arterial pressure and in systemic vascular resistance[1]. The magnitude of these hemodynamic changes depend on the position of the aortic clamp, the amount of collateral circulation to the proximal tissue[2] and the duration of the clamp. The hemodynamic changes which follow removal of the clamp may be due to the severity of the metabolic changes of ischemic tissue which is produced by aortic clamp[3]. It is difficult to predict the hemodynamic changes after aorta declamp during aorta clamp.

Whole body oxygen consumption depends on the oxygen demand of tissue. Cardiac output and oxygen consumption are matched in the normal state[4], but the relationship between hemodynamic changes and oxygen consumption during aortic clamping and declamping is not well documented.

We measured the hemodynamic and

metabolic changes of aortic clamping and declamping during isoflurane anesthesia to examine the relation between whole body oxygen consumption and the cardiac output and the change of oxygen consumption during aorta clamp reflects the hemodynamic changes after aorta declamp.

Methods and procedures

The study was approved by the subcommittee on animal care of the Utah university. Seven mongel dogs (21.1±3.3 kg) were anesthetized with 10 mg/kg thiopental and their tracheas intubated. Isoflurane and 28-30% oxygen and nitrogen were administered along with pancronium bromide (0.4 mg). The dogs were mechanically ventilated by a Servo 900 B ventilator (Siemens Elema, Sweden) nonrebreathing system. The ventilator was initially set to keep the $PaCO_2$ between 30 to 40 mmHg. The tidal volume and the rate remained unchanged through the study. The end-tidal isoflurane concentration was monitored by an infrared anesthetic gas analyzer (IRINA Dräger, Lubeck, W Germany) and the inspired concentration was adjusted to maintain an expired concentration of 1.4 volume %. A catheter was inserted in the carotid artery to measure the arterial pressure and obtain blood samples. A flow directed thermodilution catheter was placed in the pulmonary artery via a jugular vein to obtain mixed venous blood samples and measure pulmonary arterial and pulmonary capillary wedge pressure. A 16 gage catheter was advanced to a saphenous vein through a femoral vein to sample peripheral venous blood. An 18 gage catheter was placed in a forelimb vein to infuse the lactate Ringer solution (5 ml/kg/hr.), muscle relaxant, heparin, and sodium bicarbonate. Heparin (500 units) was injected before aortic cross clamping. Sodium bicarbonate was administered to correct an acid-base abnormality before starting the study. Cardiac output was measured by thermodilution methods (model 9520 A, Edwards Laboratory, U. S. A.). Oxygen consumption was measured by an Oxyconsummeter (Dräger, Lubeck, W. Germany). Blood temperature was monitored by a thermister on the thermodilution catheter.

Following abdominal incision and after waiting one hour for the stabilization, the abdominal aorta was clamped just above the celiac artery by arterial forceps. Measurements included arterial, mixed venous, and saphenous vein blood gases, Hb and saphenous vein serum sodium and potassium. Blood gases were measured using an ABL II analyzer (Radiometer, Copenhagen, Denmark). Sodium and potassium were measured by NOVA I analyzer (NOVA, U. S. A.). Systemic vascular resistance, stroke volume, and left ventricural stroke work was calculated[5]. Oxygen debt was calculated by multiplying the difference between VO_2 before applying the clamp and VO_2 during the clamp by the duration of the aorta clamp. The measurement was made before aortic calmping (control), 3, 6 and 15 minutes after aorta clamping and 3, 6, 15, and 30 minutes after aorta declamping. Student's paired t test was used and p values less than 0.05 was considered to be statistically significant.

Results

Hemodynamic changes following application and removal of the aortic cross clamp are presented in the Table 1. Mean arterial pressure and pulmonary arterial pressure increased with aorta clamp and continued to gradually increase druing the period of the clamp. Pulmonary arterial wedge pressure was elevated after aorta clamp and remained high until 6 minutes after aortic declamping ($p<0.05$). Heart rate decreased just after aorta clamp ($p<0.051$), but returned to control values before aortic declamping. Cardiac output decreased 3 minutes after aorta clamp ($p<0.05$), but soon increased to greater than

Table 1 Hemodynamic parameters

	Control	Clamp 3 min	Clamp 6 min	Clamp 15 min
MAP (mmHg)	99.6±27.1	152.3±21.8 ##	153.4±20.3 ##	155.0±22.0 ##
MPAP (mmHg)	21.0±3.6	22.4±5.1	23.7±4.4 #	26.4±4.6 ##
HR (/min)	148±22	133±18 ##	136±21 #	142±23
PAW (mmHg)	7.6±3.0	11.8±2.3 ##	13.6±3.9 #	13.7±2.0 ##
CO (l/min/kg)	0.16±0.04	0.12±0.03 #	0.13±0.03	0.20±0.07 ##
SVR (dynes·sec·cm^{-5})	2317±970	4927±1243 ##	4415±1186 ##	3387±1871
LVSW (g·m)	29.5±14.1	36.3±13.3	38.2±6.6	57.4±27.5 ##
	Declamp 3 min	Declamp 6 min	Declamp 15 min	Declamp 30 min
MAP (mmHg)	106.0±32.6	96.7±26.4	90.1±24.1	90.4±27.4
MPAP (mmHg)	29.4±9.2 ##	26.6±9.3 #	24.4±4.7 ##	23.1±4.7
HR (/min)	143±15	134±14	136±15	137±16
PAW (mmHg)	12.1±5.4 #	11.3±7.2	9.6±3.0	8.3±3.0
CO (l/min/kg)	0.25±0.11	0.21±0.09	0.20±0.08	0.19±0.05
SVR (dynes·sec·cm^{-5})	1696±755	1847±779	1786±630	1854±825
LVSW (g·m)	46.9±21.9 #	37.6±17.2	43.1±17.9	31.6±13.5

# $p \leqq 0.05$, ## $p \leqq 0.01$, vs. control value.
MAP=mean arterial pressure.
MPAP=mean pulmonary arterial pressure.
HR=heart rate.
PAW=pulmonary arterial wedge pressure.
CO=cardiac output.
SVR=systemic vascular resistance.
LVSW=left ventricular stroke work.

control values, although this change was not significant compared to control values except for the value 3 minutes after aorta clamp ($p<0.05$). Systemic vascular resistance was elevated 3 and 6 minutes after aorta clamp ($p<0.01$), but then gradually returned to control values. The cardiac function curve shows the depression of cardiac function 3 and 6 minutes after aorta clamp ($p<0.05$), although the return to control curve 15 minutes after aorta clamp (Fig. 1).

Table 2 shows the metabolic changes. Oxygen consumption, which was 6.04±0.95 ml/min./kg before the clamp fell to 3.79±0.68

Table 2 Metabolic parameters

	Control	Clamp 3 min	Clamp 6 min	Clamp 15 min
VO_2 (ml/min/kg)	6.02±0.91	3.79±0.68 ##	3.94±0.84 ##	4.13±0.99 ##
pH (a)	7.401±0.087	7.428±0.095	7.423±0.827	7.385±0.107
Bicarbo (mEq/l)	20.6±5.7	18.8±6.0	21.3±7.4	17.5±6.3
pH (V)	7.379±0.075	7.386±0.099	7.353±0.115	7.335±0.109
$SatO_2$ (%)	68.1±16.8	41.3±9.9 ##	23.8±12.6 ##	17.1±4.9 ##
K (mEq/l)	3.35±0.29	3.68±0.32 ##	3.79±0.29 ##	3.93±0.42 ##
Na (mEq/l)	145.5±4.3	146.2±3.1	145.5±3.3	145.3±2.5
O_2 debt (ml/kg)	0	6.70±1.92 ##	12.95±3.05 ##	29.91±6.41 ##
	Declamp 3 min	Declamp 6 min	Declamp 15 min	Declamp 30 min
VO_2 (ml/min/kg)	7.22±1.11	6.72±1.15	6.48±1.25	6.18±1.03
pH (a)	7.301±0.089	7.263±0.075 ##	7.263±0.045 ##	7.275±0.069 ##
Bicarbo (mEq/l)	16.1±5.8	16.4±3.8	14.1±2.2	15.2±2.9
pH (V)	7.259±0.056 ##	7.245±0.046 ##	7.242±0.061 ##	7.262±0.058 ##
$SatO_2$ (%)	59.3±16.9	63.6±11.8	62.7±14.1	58.5±21.9
K (mEq/l)	3.80±0.51 #	3.67±0.41 ##	3.74±0.23 ##	3.74±0.38 #
Na (mEq/l)	149.9±5.2	142.5±5.9	141.9±5.6	143.6±3.7

$p \leqq 0.05$, ## $p \leqq 0.01$, vs. control value.
VO_2=Oxygen consumption.
pH (a)=pH of arterial blood.
Bicarbo=bicarbonate of arterial blood.
pH (V)=pH of mixed venouse blood.
$SatO_2$=saphenouse vein oxygen satuation.
K=serum potassium of saphenouse vein blood.
Na=serum soium of saphenouse vein blood.
O_2 debt=O_2 debt during aortic clamping.

ml/min./kg during the clamp ($p<0.01$). Oxygen debt increased during the time of the clamp ($p<0.01$). Arterial blood pH, arterial blood bicarbonate, and mixed venous blood pH were lower than control after the aortic clamp was removed ($p<0.01$). Saphenous vein O_2 saturation decreased during the clamp ($p<0.01$). Serum potassium of saphenose vein were elevated after the aorta clamp ($p<0.01$).

Table 3, and Fig. 2-5 show the relationships among the changes of oxygen consumption and changes of arterial pressure, cardiac output, systemic vascular resistance and saphenous vein oxygen saturation. Relationships among

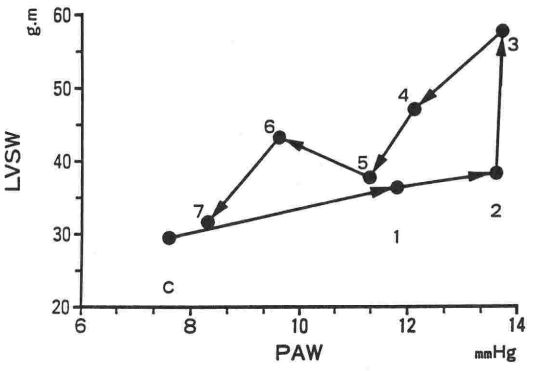

Fig. 1. Cardiac function curve before aorta clamp, after aorta clamp and after aorta declamp. Cardiac function curve moved to right lower part after 3 and 6 minutes after aorta clamp. These movement was significant to control ($p<0.05$)
LVSW means left ventricular stroke work.
PAW means pulmonary arterial wedge pressure.
C indicates control. 1 indicates 3 minutes after aortic clamping.
2 indicates 6 minutes after aortic clamping.
3 indicates 15 minutes after aortic clamping.
4 indicates 3 minutes after aortic declamping.
5 indicates 6 after aortic declamping.
6 indicates 15 minutes after aortic declamping.
7 indicates 30 minutes after aortic declamping.

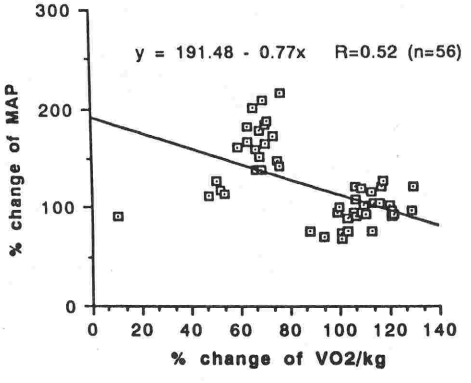

Fig. 2. Relationship between percent changes of mean arterial pressure and oxygen consumption.
% change of MAP means percent changes of mean arterial pressure to control value.
% change of VO_2/kg means percent changes of oxygen consumption per body weight to control value.

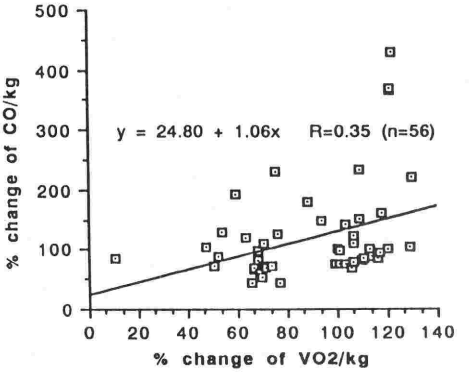

Fig. 3. Relationship between percent changes of cardiac output and oxygen consumption.
% change of CO/kg means percent changes of cardiac output per body weight to control value.
% change of VO_2/kg means percent changes of oxygen consumption per body weight to control value.

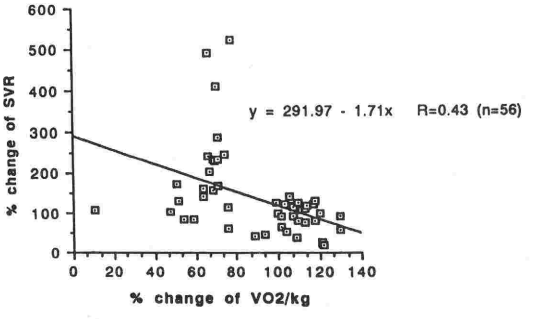

Fig. 4. Relationships between percent changes of systemic vascular resistance and percent changes of oxygen consumption.
% change of SVR means percent changes of systemic vascular resistance to control value.
% change of VO_2/kg means percent changes of oxygen consumption per body weight to control value.

changes in arterial pressure after aorta declamp, O_2 debt at 15 minutes after aorta clamp, and maximum percent changes in saphenous vein O_2 saturation during the course of aorta clamp were also shown in the Table 3.

Discussions

Sudden interruption of arterial blood flow to tissue causes oxygen debt in ischemic tissue.

Table 3

I. Relationships between percent changes in VO_2, and percent changes in MAP, CO, SVR, or saphenouse vein oxygen saturation.

Y(% of MAP)=191.48−0.77X (r=0.52 p<0.01 n=56)

Y(% of CO/kg)=24.80+1.06X (r=0.35 p<0.05 n=56)

Y(% of SVR)=291.97−1.71X (r=0.43 p<0.01 n=56)

Y(% of saphenouse vein O_2 saturation)=5.28+0.84X (r=0.45 p<0.01 n=56)

X means percent changes of oxygen consumption (VO_2) per kg to control value.

% of MAP means percent changes of mean arterial pressure to control value.

% of CO means percent changes of cardiac output (CO) to control value.

% of SVR means percent changes of systemic vascural resistance (SVR) to control value.

II. Relationships between percent changes of the reduction of arterial pressure after aorta declamp, and O_2 debt of 15 minuts after aorta clamp and percent changes of maximam reduction of saphenouse vein O_2 saturation during the time of clamp.

Y(O_2 debt)=36.7−0.22X (ml/kg) (r=0.72 p<0.05 n=6)

Y(% of saphenouse vein O_2 saturation)=28.8+14.4X (r=0.15 p>0.05 n=6)

X means the percent changes of mean arterial pressure of 3 minutes after aorta declamp to 15 minutes after aorta clamp.

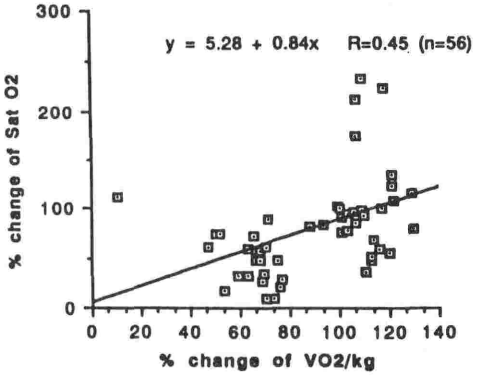

Fig. 5. Relationships between percent changes of saphenouse vein saturation O_2 and percent changes of oxygen consumption.
% change of sat O_2 means percent changes of saphenouse vein oxygen saturation to control value.
% change of VO_2/kg means percent changes of oxygen consumption per body weight to control value.

This oxygen debt may be reflected as a decrease in whole body oxygen consumption, if other organs or tissues do not consume more oxygen to compensate the failed function after interruption of blood flow.

In our study, an application of an aortic cross clamp was followed by a significant fall in oxygen consumption, ($p<0.01$), a significant fall in cardiac output ($p<0.05$), a significant fall in saphenous vein O_2 saturations ($p<0.01$) and by a significant rise in systemic vascular resistance ($p<0.01$). The decrease in cardiac output and the increase in systemic vascular resistance are probably related to a decrease in the overall volume of tissue of blood perfused rather than to deterioration of myocardial performance. But in our study, the aortic cross clamp also resulted in a significant increase in pulmonary arterial wedge pressure ($p<0.01$). The cardiac function curve moved to the right 3 and 6 minutes after the clamp, indicating the depression of myocardial performance[6]. The transient decrease of cardiac output after aortic cross clamping may be partially explained by this cardiac depression, which is in part caused by the stretching of left ventricular muscle fiber from the abrupt increase of outflow resistance[7].

Gelman S and et al[8] believe that cardiac output is reduced after aortic cross clamping, because of reduced oxygen consumption. We found no relationship between oxygen consumption and cardiac output, after aortic clamping and declamping ($r=0.35$ $p>0.05$).

The sudden reduction of arterial pressure which occurred just after aorta declamp may be related to the release of a cardiovascular depressant substance to the central circulation from ischemic tissue and a subsequent hyperemic reaction of blood vessels of reperfused tissue. The production of the cardiovascural depressant substance and the degree of hyperemic reaction of reperfused vessel may be depended on the time and the place of the cross clamp of the aorta. We examined the correlation between the reduction of arterial pressure or systemic vascular resistance after aortic declamping, and O_2 debt, but there were no statistically significant correlations among these variables ($r=0.45$ $p>0.05$). O_2 debt calculated from oxygen consumption does not seem to reflect the cardiovascural changes after aorta declamp. These changes may be affected not only by O_2 debt, but also by another factor which includes the blood sugar, Ca^{++}, catecholamine, and the production of free radical[9,10] in ischemic tissue. These results suggests it is difficult to predict the outcome of the hemodynamic changes after aorta declamp from the measurement of oxygen consumption during aorta clamp.

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