Original

EFFECTS OF THE COMBINATION OF HYPERVOLEMIC HEMODILUTION AND HYPOTENSIVE ANESTHESIA WITH INHALED ANESTHETICS ON GASTRIC INTRAMUCOSAL PERFUSION

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Abstract

This prospective study was carried out to estimate the effects of acute hypervolemic hemodilution (HHD) and hypotensive anesthesia with isoflurane or sevoflurane on gastrc intramucosal perfusion in patients undergoing hip surgery. Forty patients were allocated randomly to control groups (isoflurane [Group A; n=10] or sevoflurane [Group B; n=10] anesthesia without hypotension) and hypotension groups (isoflurane-[Group C; n=10] or sevoflurane-[Group D; n=10] induced hypotension). After induction of anesthesia, HHD was produced by preoperative infusion of 1,000 mL of 6% hydroxyethylstarch without removal of blood in all groups. Final hematocrit value was 24 to 25% in any group. Controlled hypotension was induced by increasing the inspired concentration of isoflurane or sevoflurane, and mean arterial blood pressure was maintained at approximately 55 mmHg for 80 minutes in hypotension groups. Gastric intramucosal pH (pHi) were measured using tonometry for estimating gastric intramucosal perfusion. The mean pHi values showed no change after hemodilution in any group. In control groups, the pHi values showed no change throughout the time course. In hypotension groups, the pHi value decreased significantly at 80 minutes after starting hypotension and 60 minutes after recovery from hypotension, while they recovered on the first post-operative day.

We conclude that the combination of HHD and hypotensive anesthesia with isoflurane or sevoflurane causes gastric intramucosal hypoperfusion.

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—Key words—
Hemodilution, Inhaled anesthetics, Gastric intramucosal perfusion

Introduction

Preoperative acute hemodilution can achieved in two ways. First, by withdrawal of blood and simultaneous infusion of crystalloid or colloid solutions (acute normovolemic hemodilution, ANH[1]). Second, by rapid infusion of the solutions without blood withdrawal (acute hypervolemic hemodilution, HHD[2]). HHD has shown to be a simple and safe procedure for saving autologous blood in the patients undergoing hip surgery with a predicted blood loss of about 1000 mL[3]. HHD with colloid substitutes can cause and keep hyperdynamic change because of a large intravascular volume for approximately 3 to 4 hours[4][5]. Controlled hypotension with HHD may be more effective for avoiding allogenic transfusion, however, it may impair regional perfusion because of the reduction in the blood oxygen carrying-capacity and in the perfusion pressure[6].

Major reduction of splanchnic blood volume and flow can be vital in defending the perfusion of the important organs, i.e., the brain and the heart, in acute hypovolemia[7]. The gastrointestinal mucosa may be particularly vulnerable to even mild degrees of hemorrahgic shock[8]. Gastric intramucosal carbon dioxide partial pressure ($Pico_2$) and calculated pH (pHi) using gastric tonometry have provided the indicators of systemic hypovolemia[9] and/or gastrointestinal perfusion under clinical conditions[10][11].

Sevoflurane is similar to isoflurane in its effect on systemic and regional hemodynamics[12], and a higher concentration of their anesthetics may suppress those hemodynamics[13]. So hypotensive anesthesia with inhaled anesthetics during HHD may cause the change of systemic and regional hemodynamics resulting in gastrointestinal hypoperfusion. However, no previous studies have evaluated gastrointestinal perfusion during isoflurane or sevoflurane-induced hypotension with HHD in humans.

This study was carried out to evaluate the effects of hypotensive anesthesia with isoflurane or sevoflurane combined with HHD on gastric intramucosal perfusion by measuring gastric pHi using tonometry.

Methods

The subjects of this investigation were 40 ASA physical status I or II total hip arthroplasty patients, aged 48 to 71 years, weighing 44 to 71 kg, without hypertension, ischemic heart disease, cerebral infarction, hepatic or renal dysfunction, and anemia (hemoglobin < 11 g dL^{-1}). The protocol was approved by the Nagasaki Rosai Hospital Institutional Human Committee and written informed consent was obtained from each patient.

Patients were premedicated with intravenous ranitidine 50 mg approximately 2 hours before anesthesia induction, intramuscular atropine sulphate 0.5 mg, and hydroxyzine hydrochloride 1 mg kg^{-1}, approximately 1 hour before anesthesia induction. Ranitidine was used as a histamine (H_2)-blocker in premedication for preventing gastric intraluminal production of CO_2[14]. Patients were continuously monitored with pulse oximetry (Oxypal OLV-1200, Nihon Kohden Co., Ltd., Tokyo, Japan) and three lead electrocardiogram. A radial arterial catheter was inserted for continuous monitoring of arterial blood pressure (ABP) and for obtaining blood samples. ABP and heart rate (HR) were automatically recorded (Bed side monitor BSM-8500, Life Scope 12, Nihon Kohden Co., Ltd., Tokyo, Japan). Anesthesia was induced with intravenously thiamylal 5 mg kg^{-1}, and fentanyl 2 μ g kg^{-1}. Tracheal intubation was facilitated with intravenously vecuronium bromide 0.1 mg kg^{-1}. After induction of anesthesia, patients were allocated randomly to control groups (isoflurane [Group A; n=10] or sevoflurane [Group B; n=10] anesthesia without hypotension) and hypotension groups (isoflurane-induced hypotension [Group C; n=10] or sevoflurane-induced hypotension [Group D; n=10]). The patients were divided by sealed envelope assignment into each group. Anesthesia was maintained with either isoflurane (Groups A and C) or sevoflurane (Groups B and D) supplemented with 60% nitrous oxide (N_2O) in oxygen (O_2) at a total gas flow of 5 L min^{-1}using a Drager Narcomed Model 4 anesthesia machine (North American Drager, Telford, PA) with a semiclosed circle system using a soda lime canister. Intravenous fentanl, 1 to 2 μg kg^{-1} and vecuronium, 0.05 mg kg^{-1} were injected during surgery as required. Ventilation was controlled to maintain end-tidal carbon dioxide tension ($ETCO_2$) at approximately 4.8 kPa. $ETCO_2$ and end-expiratory concentration of isoflurane (1.2 to 1.6%) or sevoflurane (1.4 to 2.0%) were continuously monitored and recorded by anesthetic gas monitor (Capnomac; Datex Intsrumentarium, Helsinki, Finland). Acetated Ringer's solution was infused to the amount of 10 mL kg^{-1} before surgery during a 4-hour period. The infusion was continued at a rate of 6 mL kg^{-1} hr^{-1} during surgery. Additional acetated Ringer's solution was infused at three times the amount of blood loss. Rectal temperature was maintained at 36.0 to 36.5°C using a circulating water blanket and ajusting temperature to 25°C and humidity to 50% in the operating room. After induction of anesthesia, HHD was produced by preoperative infusion of 1,000 mL of 6% hydroxyethylstarch solution (HES; molecular weight=70,000) without removing blood. 6% HES was infused at a rate of approximately 50 mL min^{-1} using a rapid infusion pump. Acetated Ringer's solution and HES were infused at body temperature (36.5°C) after warming by medical warmer (NIKO Electric Medical Co., Ltd., Tokyo, Japan). Controlled hypotension was induced by increasing the inspired concentration of isoflurane (2.0 to 3.0%) in Group C or sevoflurane (2.4 to 3.6%) in Group D, and maen arterial blood pressure (MAP) was maintained at 55 to 60 mmHg for approximately 80 minutes during surgery. In groups A and B, autologous blood of 200 mL was obtained from patients on the 21st day prior to surgery and 200 mL was obtained on the 14th day before surgery, and stored at 4°C in a blood refrigerator. The volume of blood loss was estimated during operation by weighing swabs and measuring suction drainage, and after operation by measuring blood collected from the wound drainage. In all groups, autologous blood was stored by a cell saver (Haemolite, Haemonetics Corp., Boston, MA) during and after surgery and retransfused after surgery, In groups A and B, preoperative autologous blood donation also was retransfused after surgery.

Gastric $Pico_2$ was indirectly measured by tonometry. A balloon-tipped nasogastric tube for gastric tonometry

(TRIP NGS Catheter, Tonometrics, Inc., Worcester MA, USA) was inserted into the stomach and the correct position checked by roentgenography and by ausculation. Two-half mL of a saline solution was injected into the balloon. Thirty minutes later, 1 mL of the solution was aspirated (resembling the dead space of the tube) and discarded. The remaining 1.5 mL was then aspirated and the Pco_2 in the solution was determined with a blood gas analyzer. At the same time, samples of arterial blood were obtained for calculation of sodium bicarbonate (HCO_3^-) and measurement of serum lactate.

Measurements, including hematocrit (Hct), hemodynamics (MAP and HR), arterial blood gas, gastric $Pico_2$ and concentration of serum lactate were made before hemodilution (T0), after hemodilution (T1), 80 minutes after starting hypotension (T2), 60 minutes after recovery from hypotension (T3), and on the first post-operative day (T4).

Arterial blood gas was analyzed by a blood gas analyzer (ABL-4, Radiometer Corp., Copenhagen, Denmark). Arterial lactate was measured by enzymatic analysis (enzyme immunoassay kit, Determiner LA, Kyowa Medix, Tokyo, Japan). Hct value was determined by centrifugation. The blood samples were analyzed immediately after collection in the operating room. In order to obtain gastric $Pico_2$, the measured value of Pco_2 in the saline solution was calculated using a time-dependent Pco_2 correction for equilibration period between intraluminal Pco_2 and Pco_2 in the saline of the tonometer. The equilibration time of saline was set as 30 minutes at T0 and T1, 80 minutes at T2, 60 minutes at T3 according to surgical procedure and 90 minutes at T4. Gastric pHi was calculated with a modification of the Henderson-Hasselbalch equation using $Pico_2$ measured and arterial HCO_3^- values according to the recommendation by Fiddian Green et al[15].

Data are expressed as means (SD). Statistical analysis was performed using analysis of variance and a Bonferroni's correction. A p-value less than 0.05 was considered statistically significant.

Results

The groups were similar in demographic characteristics, operative period, hypotensive period, blood loss, urinary output, and infusion volume (Table 1). No patinet had homologous transfusion during and after surgery in all groups.

The changes of blood gas variables in all groups are shown in Table 2. No differences were observed between the four groups throughout the time course. There was no apparent acidemia or alkalemia in any group. The changes of Hct and MAP are shown in Figure 1. After hemodilution, final Hct value was 24 to 25% in any group. MAP was maintained at approximately 95 mmHg in control groups (Group A and B) and approximately 55 mmHg during controlled hypotension in hypotenision groups (Groups C and D).

The changes of lactate, gastric pHi in all groups are shown in Figure 2. No differences in lactate values were

Table 1 Patient Group Characteristics

	Group A (n = 10)	Group B (n = 10)	Group C (n = 10)	Group D (n = 10)
Age (yr)	61 (50 ～ 68)	63 (51 ～ 70)	60 (52 ～ 71)	61 (48 ～ 69)
Gender (female/male)	9/1	9/1	8/2	8/2
Weight (kg)	55 (6)	53 (7)	54 (6)	56 (8)
Operative time (min)	108 (10)	106 (9)	113 (12)	109 (11)
Hypotensive period (min)	—	—	81 (7)	80 (5)
Intraoperative infusion volume (mL)	2,380 (211)	2,312 (201)	2,211 (192)	2,190 (208)
Intraoperative blood loss (mL)	608 (197)	614 (189)	487 (99) †	492 (103) #
Intraoperative urinary output (mL)	234 (78)	288 (63)	187 (81)	217 (97)
Postoperative infusion volume (mL)	1,365 (225)	1,384 (168)	1,323 (208)	1,406 (185)
Postoperative blood loss (mL)	524 (261)	478 (236)	497 (211)	511 (192)
Postoperative urinary output (mL)	492 (183)	470 (182)	462 (276)	505 (213)
MAC-h	1.4 (0.2)	1.8 (0.3)	2.8 (0.3) †	3.2 (0.4) #

Results are means (SD). †$P < 0.05$ vs. Group A, #$P < 0.05$ vs. Group B. Intraoperative infusion volume does not include the volume of 6% hydroxyethylstarch for hemodilution in all groups. Group A, isoflurane anesthesia without hypotension ; Group B, sevoflurane anesthesia without hypotension ; Group C, hypotensive anesthesia with isoflurane ; Group D, hypotensive anesthesia with sevoflurane ; MAC-h, minimum alveolar concentration per hour.

Table 2 Changes in Arterial Blood Gas Variables

Variable	Group	T0	T1	T2	T3	T4
pHa	A	7.461 (0.036)	7.468 (0.034)	7.464 (0.038)	7.458 (0.033)	7.452 (0.046)
	B	7.465 (0.032)	7.466 (0.023)	7.454 (0.034)	7.460 (0.029)	7.458 (0.041)
	C	7.458 (0.040)	7.459 (0.035)	7.453 (0.036)	7.455 (0.041)	7.449 (0.043)
	D	7.454 (0.028)	7.452 (0.031)	7.448 (0.033)	7.450 (0.034)	7.453 (0.044)
Pao_2 (mmHg)	A	180 (23)	182 (28)	172 (22)	178 (26)	168 (38)
	B	176 (19)	174 (24)	168 (26)	172 (21)	159 (41)
	C	172 (20)	180 (31)	166 (24)	176 (28)	132 (34)
	D	188 (29)	176 (22)	178 (30)	182 (27)	169 (44)
$Paco_2$ (mmHg)	A	38 (2)	37 (2)	37 (3)	39 (3)	42 (5)
	B	36 (3)	35 (3)	36 (2)	38 (3)	40 (5)
	C	38 (3)	36 (2)	36 (3)	37 (3)	41 (6)
	D	37 (3)	36 (3)	37 (3)	38 (3)	40 (4)

Results are means (SD). Group A, isoflurane anesthesia without hypotension ; Group B, sevoflurane anesthesia without hypotension ; Group C. hypotensive anesthesia with isoflurane ; Group D, hypotensive anesthesia with sevoflurane ; pHa arterial blood pH ; Pao_2, arterial oxygen partial pressure ; $Paco_2$, arterial carbon dioxide partial pressure ; T0, before hemodilution ; T1, after hemodilution ; T2, 80 minutes after starting hypotension ; T3, 60 minutes after recovery from hypotension ; T4, first post-operative day.

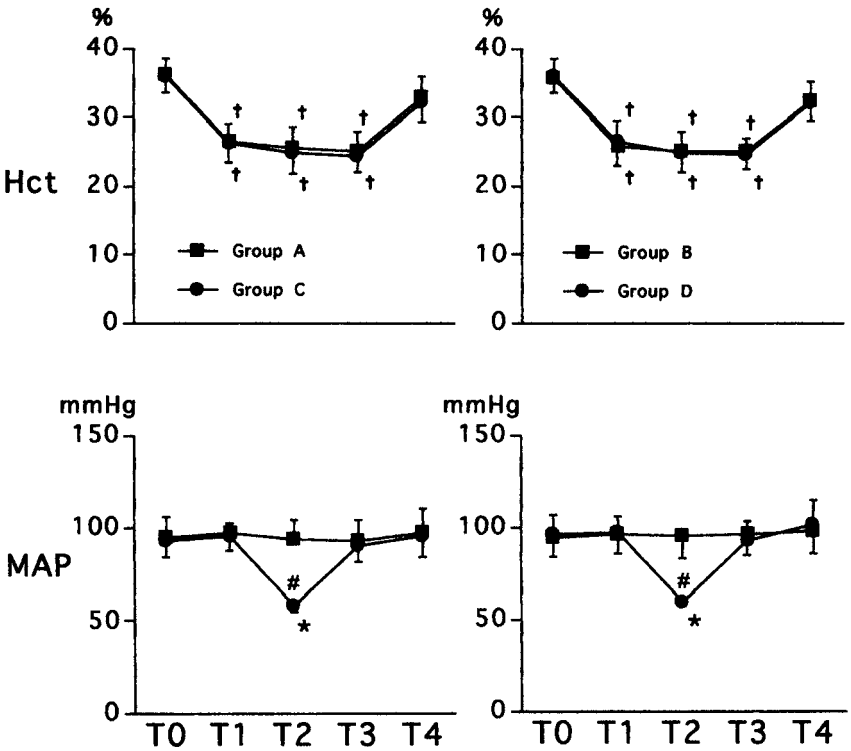

Figure 1. Time course of changes in hematocrit and mean arterial blood pressure (n = 10 for each point in all groups).
Group A, isoflurane anesthesia without hypotension; Group B, sevoflurane anesthesia without hypotension ; Group C, hypotensive anesthesia with isoflurane ; Group D, hypotensive anesthesia with sevoflurane ; Hct, hematocrit ; MAP, mean arterial blood pressure ; T0, before hemodilution ; T1, after hemodilution ; T2,80 minutes after starting hypotension ; T3,60 minutes after recovery from hypotension ; T4, the first post-operative day. Results are means ± SD. $^{\dagger}p < 0.05$, $^{\star}p < 0.01$, Significantly different from T0, # $p < 0.05$, Significantly different from Group A or Group B.

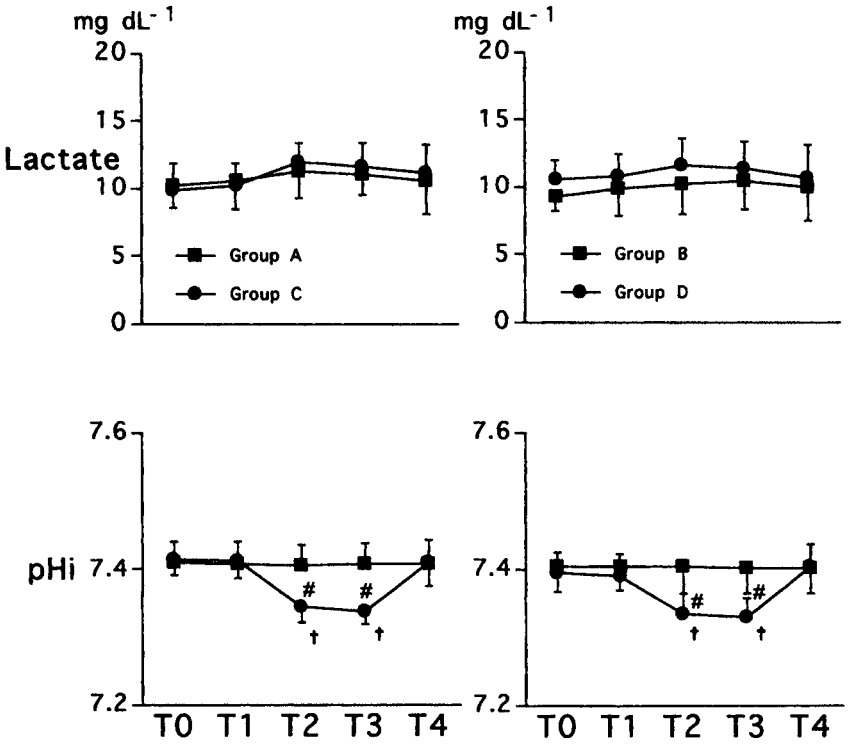

Figure 2. Time course of changes in serum lactate, gastric pHi (n = 10 for each point in all groups). Serum lactate value showed no change in any group throughout the study. The mean pHi values showed no change after hemodilution in any group. In hypotension groups, the mean pHi values showed significant decreases 80 minutes after starting hypotension and 60 minutes after recovery from hypotension, whereas they recovered on the first post-operative day.
Abbreviations as in Figure 1. pHi, gastric intramucosal pH. Results are means ± SD. † $p < 0.05$, Significantly different from T0, # $p < 0.05$, Significantly different from Group A or Group B.

observed throughout the study in any group. The mean pHi values showed no change after hemodilution in any group. In control groups, the pHi values showed no change at T2, T3 and T4. In hypotension groups, the pHi values decreased significantly at T2 and T3, while they recovered on the first post-operative day. The values in hypotension groups showed significant differences from control groups at T2 and T3.

There were no gastroenterologic problems after surgery in the patients.

Discussion

The present study suggests that the combination of HHD and hypotensive anesthesia with isoflurane or sevoflurane causes gastric intramucosal hypoperfusion.

A decrease in gastric pHi can indicate the insufficient oxygenation and/or the hypoperfusion of the gastric intramucosal mucosa. Suttner et al[16]. have evaluated splanchnic perfusion using gastric phi and the mucosal-arterial Pco_2 gradient.

Although the critical value of the low gastric pHi would be less than 7.32 or 8.35 in critically ill patients[17], perioperative studies have taken a pHi less than 7.32 as evidence of intramucosal acidosis[18].

Malan et al. [13] reported that the cardiovascular effects of sevoflurane at 1.0 minimum alveolar anesthetic concentration (MAC) were similar to those of isoflurane in volunteers.

Sevoflurane-N_2O or isoflurane at 1.0 MAC decreases in MAP, cardic output (CO) and mean poulmonary arterial pressure (MPAP). HHD under anesthesia may[2] or may not[4] increase in CO and MPAP.

In the present study, HHD under isoflurane or sevoflurane anesthesia did not show excessively an increase of

ABP and a decrease of arterial oxygen partial pressure (Pao_2) and did not cause gastric intramucosal acidosis. These results indicate that isoflurane or sevoflurane could attenuate cardiovascular responses to HHD for inducing vasodilation, besides HHD under isoflurane or sevoflurane anesthesia would maintain regional hemodynamics for a sufficient intravascular volume resulting in preservation of the adequate distribution to gastric intramucosal perfusion.

The combination of HHD and hypotensive anesthesia with isoflurane or sevoflurane caused a gastric intramucosal acidosis. Three patients of both groups showed pHi values of less than 7.32. The results suggest that the low pHi might be due to inadequate distribution of gastric intramucosal perfusion.

During isoflurane-induced hypotension in humans Co may[19] or may not[20] decrease and systemic vascular resistance (SVR) may decrease at a MAP of 40 mmHg. A high concentration of sevoflurane or isoflurane significantly may decrease CO, SVR and total hepatic and renal blood flows at a MAP of 50 or 60 mmHg in animals[12)21)22)]. In gastrointestinal perfusion, it has been reported that 1.5 MAC of sevoflurane or isoflurane with 50% N_2O decreased the blood flow in stomach and small intestine at a MAP of 67 mmHg in swine[23)], and 1.7 MAC of isoflurane or sevoflurane did not decrease it at a MAP of 50 mmHg in rats[12)].

It seems that in spite of HHD with a sufficient intravascular volume, isoflurane or sevoflurane-induced hypotension might cause systemic and regional hemodynamic changes resulting in blood flow redistribution within splanchnic circulation for preserving hepatic and renal perfusion. Splanchnic hypoperfusion may be easily caused by simpathomimetic and hormonal vasoactive stimulation.

In the present study, the impairment of gastric intramucosal perfusion in the combination would be mild from a clinical view point.

In conclusion, the combination of HHD and hypotensive anesthesia with isoflurane or sevoflurane causes gastric intramucosal hypoperfusion.

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高容量性血液希釈と吸入麻酔薬による低血圧麻酔併用が胃粘膜内血流に及ぼす影響

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—キーワード—
血液希釈，吸入麻酔，胃粘膜内血流

高容量性血液希釈とイソフルラン（Iso）またはセボフルラン（Sev)麻酔による低血圧麻酔併用が胃粘膜内血流に及ぼす影響を検討した．本研究の被験者として同意を得た40名の股関節予定手術をうける患者を対象とした．無作為にコントロールとして非低血圧群（A群＝Iso麻酔，10名およびB群＝Sev麻酔，10名）と低血圧群（C群＝Iso低血圧麻酔，10名およびD群＝Sev低血圧麻酔，10名）の4群に分けた．各々麻酔導入後，脱血せずにヒドロキシエチルスターチ1,000mlの急速輸液による高容量性血液希釈を行い最終ヘマトクリット値は24～25％であった．術中はIsoまたはSev麻酔で維持し，C群およびD群ではこれらの高濃度吸入にて平均血圧を約55mmHgに約80分間維持した．胃粘膜内血流の指標として胃トノミータを留置後，間接的に胃粘膜内Pco_2を測定し，胃粘膜内pH（pHi）を計算にて求めた．血中乳酸値とともに血液希釈前，血液希釈後，低血圧麻酔開始80分後，低血圧麻酔終了60分後，術後1日目に測定した．

非低血圧麻酔群ではpHiおよび乳酸値は全経過中変化を認めなかった．低血圧麻酔群において，pHiは低血圧麻酔開始80分後と低血圧麻酔終了60分後に血液希釈前および各々の非低血圧麻酔群に比し有意に減少したが，術後1日目には回復した．

乳酸値は全経過中変化を認めなかった．高容量性血液希釈およびイソフルランまたはセボフルラン麻酔による低血圧麻酔の併用は胃粘膜内血流を同等に減少させる．