Cerebral Oxygen Desaturation in Patients with Totally Thoracoscopic Ablation for Atrial Fibrillation

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Abstract

Background: Epicardial radiofrequency ablation for atrial fibrillation under total video-assisted thoracoscopy causes severe cardiopulmonary disturbances and affects cerebral perfusion and oxygenation. The aim of the present study was to investigate the changes in regional cerebral oxygen saturation (rSO2) during the surgery and their correlations with hemodynamic or blood gas parameters.

Methods: A total of 45 patients scheduled for selective totally thoracoscopic ablation for stand-alone atrial fibrillation were enrolled in this study. The rSO2 was monitored at baseline (T0), 15 min after anesthesia induction (T1), 15 min after one-lung ventilation (T2), after right pulmonary vein ablation (T3), after left pulmonary vein ablation (T4) and 15 min after two-lung ventilation (T5) using a near-infrared reflectance spectroscopy (NIRS)-based cerebral oximeter. A Swan-Ganz catheter was placed in the pulmonary artery to obtain hemodynamic parameters. Arterial blood gas was analyzed using an ABL 825 hemoximeter. Associations between changes in rSO2 (△rSO2) and hemodynamic or blood gas parameters were determined with univariate and multivariate linear regression analyses.

Results: The rSO2 decreased greatly from baseline 65.3% to 57.5% at T3 ($P < 0.001$). Univariate analysis showed that △rSO2 correlated significantly with △pH (r = -0.371, $P = 0.012$), △PaCO2 (r = -0.276, $P = 0.066$), △PaO2/FiO2 (r = 0.332, $P = 0.026$), △HR (r = 0.27, $P = 0.073$), △CI (r = -0.228, $P = 0.132$) and △PVRI/SVRI (r = -0.216, $P = 0.153$). Multivariate linear regression analysis further showed that △rSO2 was only influenced by △PaO2/FiO2 (β = 0.026, $P = 0.025$).

Conclusion: The rSO2 decreased significantly during the totally thoracoscopic ablation for atrial fibrillation. There was a correlation between rSO2 reduction and PaO2/FiO2 changes. Greater attention should be paid to cerebral oxygen desaturation during the surgery.

Introduction

Atrial fibrillation occurs in approximately 1-2% of population and is associated with high incidence of stroke and heart failure [1, 2]. Pharmacological anti-arrhythmic and antithrombotic agents are first-line treatments for atrial fibrillation. However, these medications are often ineffective or poorly tolerated. In this case, ablation strategies including catheter ablation and surgical epicardial ablation have been developed as an alternative to medical management [3]. Compared to traditional catheter ablation, surgical ablation has gained greater interests in recent years. Two recent systematic reviews have demonstrated that surgical ablation brings higher freedom from atrial fibrillation and lower recurrence [4, 5]. These advantages are more prominent for those patients with refractory atrial fibrillation and prior failed catheter intervention.

Cox-Maze III procedure constitutes the basis of surgical ablation [6]. With the progressive advances in ablation energy sources, this procedure has evolved from the traditional ‘cut-and-sew’ method to ablation

with radiofrequency or cryothermy [7, 8]. Furthermore, total video-assisted thoracoscopy replaces the original bilateral thoracotomy, which makes surgery minimally invasive [9, 10]. The progress in ablation energy and surgical technique contributes to the popularity of epicardial ablation. However, surgical manipulations on the beating heart would cause severe hemodynamic instability and affect perfusion in peripheral tissues. In addition, the thoracoscopic ablation procedure is usually performed under one-lung ventilation. Interferences from both cardiovascular and pulmonary systems could result in cerebral hypoperfusion and hypoxemia. Therefore, during the surgery, the issues of determining brain perfusion and avoiding oxygenation insufficiency should be seriously addressed.

Regional cerebral oxygen saturation (rSO2) is widely used for monitoring cerebral tissue oxygenation during cardiovascular and thoracic surgeries [11]. It is reported that a decline in rSO2 is associated with cerebral tissue hypoxemia, cognitive dysfunction and higher mortality [12-14]. Maintenance of intraoperative rSO2 at normal levels helps reduce incidence of cognitive impairment after surgery and total mortality in the hospital. For better anesthesia management, the present study was to investigate rSO2 changes and their influencing factors during totally thoracoscopic ablation for atrial fibrillation.

Materials And Methods

Study population

Between July 2016 and June 2017, 45 adult patients scheduled for selective totally thoracoscopic ablation for stand-alone atrial fibrillation were enrolled in this study. Patients with concomitant severe valvular heart disease, history of cardiac or lung surgery, left ventricular ejection fraction lower than 30%, left atrium more than 70 mm, left atrial appendage (LAA) thrombi, and contraindications to pulmonary artery catheterization were excluded. This study was approved by the institutional review board of Xinhua hospital. All patients voluntarily agreed to participate in this study and written informed consent was received from each patient the day before the surgery.

Anesthesia management

After peripheral vein cannulation, a dose of 2 mg of midazolam was intravenously injected to sedate the patients. A right-sided radial artery cannula was then implanted for monitoring arterial blood pressure and analyzing baseline blood gas condition. General anesthesia was induced with midazolam (0.05 mg/kg), etomidate (0.3 mg/kg), fentanyl (3 μg/kg) and rocuronium (0.6 to 0.9 mg/kg), followed by the insertion of a left-sided double-lumen endotracheal tube (32, 35 or 37 French, Covidien, Mansfield, MA, USA). Subsequently, a Swan-Ganz catheter (#744F75, Edwards Lifescience, Irvine, CA, USA) was placed in the pulmonary artery to measure central venous pressure (CVP), mean pulmonary arterial pressure (mPAP), pulmonary artery wedge pressure (PAWP), cardiac index (CI), etc. Anesthesia was maintained with a continuous infusion of propofol (4 to 6 mg·kg−1·h−1), remifentanil (0.15 to 0.3 μg·kg−1·min−1) and rocuronium (0.6 mg·kg−1·h−1). All patients received positive controlled ventilation with a tidal volume of 5 to 8 ml/kg and a fraction of inspired oxygen of 0.6 to 1.0, at a respiratory rate of 10 to 12 breaths/min. The thoracoscopic surgical ablation was totally conducted on the left side of chest wall. Therefore,

patients were placed in the right lateral decubitus position and left lung collapse was requested. Various strategies including increasing FiO2, endotracheal suction, adjusting position of double-lumen tube and two-lung ventilation were used if SpO2 was lower than 90% during one-lung ventilation. The procedure was performed in alignment with a previous study [15]. Briefly, bipolar radiofrequency ablation was conducted across 3 circles and 2 lines on the left atrium. Ablation of three circles included lesions of the right pulmonary vein (including right superior and inferior pulmonary veins), the left pulmonary vein (including right superior and inferior pulmonary veins) and the circle crossing over the left inferior pulmonary vein and the right superior pulmonary vein. Two linear ablations were referred to linear lesions from the left pulmonary vein to the left atrial appendage and from the left inferior pulmonary vein to the mitral valve annulus. Subsequently, the ganglionic plexus on the epicardium was ablated and the left atrial appendage was removed using a stapler. When the operation ended, the patients were transferred to intensive care unit for extubation and recovery.

Cerebral rSO2 monitoring

Cerebral rSO2 was monitored using a near-infrared reflectance spectroscopy (NIRS)-based cerebral oximeter (EGOS-600A series, Suzhou Engin Biomedical Electronics Co., Ltd, Jiangsu, China). After cleaning the patient's skin surface with alcohol, two sensors were placed bilaterally on the forehead. To reduce light contamination, a black belt was used to cover the sensors during the surgery. The left and right rSO2 were simultaneously detected at baseline (T0), 15 min after anesthesia induction (T1), 15 min after one-lung ventilation (T2), after right pulmonary vein ablation (T3), after left pulmonary vein ablation (T4), and 15 min after two-lung ventilation (T5). The average rSO2 value was calculated at each time point.

Hemodynamic evaluation and blood gas analysis

Hemodynamic changes during the surgery were assessed at the same time points as the rSO2. The hemodynamic measurements included heart rate (HR), systolic arterial pressure (SAP), diastolic blood pressure (DAP), mean arterial pressure (MAP), central venous pressure (CVP), mean pulmonary arterial pressure (mPAP), pulmonary artery wedge pressure (PAWP), cardiac index (CI), pulmonary vascular resistance index (PVRI) and systemic vascular resistance index (SVRI). Concurrently, arterial blood gas was analyzed by measuring pH, arterial carbon dioxide tension (PaCO2), ratio of arterial oxygen pressure to fractional inspired oxygen (PaO2/FiO2), arterial oxygen saturation (SpO2), hemoglobin (Hb), hematocrit (Hct), base excess (BE) and lactate (Lac) using an ABL 825 hemoximeter (Radiometer Copenhagen, Denmark).

Statistical Analysis

Continuous data are presented as the mean ± standard deviation or median with interquartile range as appropriate. Categorical data are expressed as the number (percentage) of patients. The normality of continuous data was tested with Shapiro–Wilk method. Comparisons of rSO2, hemodynamic and laboratory parameters at different time points were performed using a one-way repeated-measures

ANOVA followed by *post hoc* Bonferroni analysis if the data met the assumption of normality, or nonparametric Friedman test would be applied. If rSO2 showed significant difference at a specific time point, the changes of rSO2 (△rSO2), hemodynamic and blood gas parameters from the baseline value were calculated. Then, a univariate analysis was first performed to illustrate possible influencing factors of △rSO2 with Pearson or Spearman correlations. The variables with *P* value less than 0.2 were further incorporated into a multivariate linear regression analysis (stepwise method). Statistical significance was considered if *P* value was less than 0.05. All the statistical analyses were conducted using IBM SPSS software version 20 (SPSS Inc., Chicago, IL, USA).

Results

Table 1 illustrated demographic and clinical characteristics of patients. A total of 45 patients consisting of 25 male and 20 female patients were included in this study. Among these patients, the average age was 62 ± 8 years. Thirty-four patients (76%) were diagnosed as paroxysmal atrial fibrillation, seven patients (15%) had persistent atrial fibrillation, and four patients (9%) had longstanding persistent atrial fibrillation. The mean (SD) left atrial dimension and left ventricular ejection fraction were 41.9 ± 4.8 mm and 60.9% ± 4.7%, respectively. The majorities of patients had various comorbidities and took different types of medicines.

Regarding rSO2, there were significant differences among the six time points ($P < 0.001$, one-way repeated-measures ANOVA) (Table 2). The rSO2 at T3 was lower than that at T0 ($P < 0.001$, *post hoc* Bonferroni test), which meant that right pulmonary vein ablation caused a significant decrease in rSO2. A one-way repeated-measures ANOVA or Friedman test revealed that significant differences were observed among six time points in term of hemodynamic parameters, including HR ($P < 0.001$), SBP ($P < 0.001$), DBP ($P < 0.001$), MAP ($P < 0.001$), CVP ($P < 0.001$), mPAP ($P < 0.001$), PAWP ($P < 0.001$), CI ($P = 0.026$), PVRI ($P < 0.001$), SVRI ($P < 0.001$) and PVRI/SVRI ($P < 0.001$). The blood gas analysis showed similar characteristics. There were significant differences among six time points in pH ($P < 0.001$), PaCO2 ($P < 0.001$), PaO2/FiO2 ($P < 0.001$), SpO2 ($P < 0.001$), Hb ($P < 0.001$), Lac ($P = 0.001$), BE ($P < 0.001$) and SvO2 ($P < 0.001$) (Table 3). These results suggested that surgical ablation of atrial fibrillation led to dramatic hemodynamic instabilities and disturbances in blood gas.

Univariate analysis showed that △rSO2 correlated significantly with △pH (r = -0.371, $P = 0.012$), △PaCO2 (r = -0.276, $P = 0.066$), △PaO2/FiO2 (r = 0.332, $P = 0.026$), △HR (r = 0.27, $P = 0.073$), △CI (r = -0.228, $P = 0.132$) and △PVRI/SVRI (r = -0.216, $P = 0.153$) (Table 4). However, there were no correlations between △rSO2 and other variables including △SBP, △DBP, △MAP, △CVP, etc. The variables screened in the univariate analysis were further included for multivariate linear regression analysis. We found that △PaO2/FiO2 was the only impact factor of △rSO2 (β = 0.026, $P = 0.025$).

Discussion

The present study was to determine whether surgical ablation of atrial fibrillation caused rSO2 changes and their influencing factors. Our study revealed that rSO2 decreased significantly during the surgery. Furthermore, we found that the decrease in rSO2 was associated with changes in PaO2/FiO2. However, no significant correlations were observed between the decrease in rSO2 and changes in other hemodynamic and blood gas variables including HR, MAP, CVP, mPAP, PAWP, pH, PaCO2, etc.

We found that rSO2 decreased mainly at two time points: after right pulmonary vein ablation and after left pulmonary vein ablation. However, a significant decrease in rSO2 was only seen after right pulmonary vein ablation. This result could be partly explained by surgical disturbances. The operation was performed through the left thoracic cavity. Most of pulmonary blood flow was gravitationally redistributed into right lung when the patients were placed in the right lateral position [16]. Hypoxic pulmonary vasoconstriction of the nonventilated left lung further increased right lung perfusion [17]. Consistent with previous studies [18, 19], the right-lung ventilation during the surgery enabled patients to maintain adequate ventilation/perfusion ratio and tissue oxygenation. Once the right pulmonary vein was clamped, oxygenated forward blood flow was totally stopped. Perfusion and oxygenation in peripheral organs, especially the brain, decreased dramatically and cerebral rSO2 showed a remarkable reduction in value. However, significant rSO2 changes were not observed at the step of clamping the left pulmonary vein, which is partly in alignment with a previous literature reporting that left pneumonectomy did not induce hypoxemia during the surgery [20].This observation was probably illustrated by the fact that the left lung was not ventilated. Therefore, blood flow blockade in the left pulmonary vein did not cause severe decreases in tissue oxygen supplies.

Our study demonstrated that the average baseline rSO2 of patients was 65.3%. This value is comparable to those measured by NIRS-based INVOS 5100 Cerebral Oxymeter System in previous studies [21-23], but lower than those detected by FORE-SIGHT absolute oximeter in the studies by Kazan [14] and Tang [24]. Differences in technological algorithms and extracranial contamination regarding the two instruments may contribute to inconsistent cerebral oxygen saturation values [22, 25, 26]. Similar to a previous study [27], we found that rSO2 increased slightly after anesthesia induction. Double-lung ventilation with inhalation of 100% oxygen in supine position indeed improves cerebral oximetry. In addition, we did not ascertain a significant decrease in rSO2 at 15 min after one-lung ventilation compared to baseline level. This result is consistent with previous studies [27-29] reporting that cerebral oxygen saturation remained relatively stable during one-lung ventilation. However, Hemmerling [30] described that one-lung ventilation caused at least 15% decrease in rSO2 during thoracic surgery. In fact, the baseline rSO2 (80%) in this study is higher than those majorities of studies reported. Furthermore, the one-lung ventilation in our study specifically referred to right-lung ventilation. Previous studies indicated that right-lung ventilation led to less incidence of hypoxemia than left-lung ventilation [18, 19]. The inconformity of research background may explain why rSO2 changes are different.

It has been demonstrated that the ventilation strategy is a key factor to influence cerebral oxygen saturation [31]. Our study showed that the decrease in rSO2 at the time point of right pulmonary vein ablation was associated with significant changes in PaO2/FiO2, which is consistent with previous

studies stating that augmenting PaO2 by raising FiO2 could increase rSO2 [22, 32]. However, we did not find a significant correlation between the decrease in rSO2 and changes in PaCO2. This is contradictory to previous studies asserting that increased PaCO2 improved brain blood flow and rSO2 [31-33]. Picton [31] reported in detail that rSO2 gained 2% additional increases when PETCO2 rose from the range of 30-35 mmHg to that of 40-45 mmHg, on the premise of FiO2 set at 100%. However, the increase in rSO2 is very small. In our study, average PaCO2 increased from baseline 39 mmHg to maximal 47 mmHg at the step of right pulmonary vein ablation. However, the effect of PaCO2 increase on rSO2 may be offset by the reduction in PaO2/FiO2. In addition, our study did not support the evidence of significant correlations between decrease in rSO2 and other variables of blood gas such as SpO2, SvO2, Hct, Hb, BE and Lac. Finally, no correlations between decrease in rSO2 and changes in CI, MAP and other hemodynamic variables were observed in the present study, which is consistent with the findings from Brinkman [28]. The reason for no correlation between rSO2 and CI may be from the imprecise measurement of cardiac index due to irregular heart rate in patients with atrial fibrillation [34]. A recent study further proved that blood pressure elevation was not necessary to alleviate cerebral oxygen desaturation [35]. Therefore, the strategy of rSO2 improvement by increasing blood pressure should be re-examined during the surgery.

There were limitations in this study. First, accuracy of rSO2 measurement was influenced by various factors during the surgery. It is reported that the rSO2 value at the upper forehead is smaller than that at the lower forehead [36]. Furthermore, in the lateral position, the rSO2 of the upper hemisphere is higher than that of the lower hemisphere [37]. Movements of sensors and patients' position may result in inaccurate measurement of rSO2. However, we used a black belt to fix the sensors during the surgery. In addition, the average rSO2 was calculated from rSO2 in upper and lower hemispheres. As a result, the rSO2 at baseline and after placement of lateral position was similar to those described in previous studies [22, 28]. Second, transcranial Doppler ultrasound was not applied to measure cerebral blood flow in the study. Comprehensive measurements combining rSO2 and transcranial Doppler ultrasound or imaging techniques would be used to better reflect brain perfusion and oxygenation. Finally, limited number of included patients may bias the precision of results in the study. More multicenter clinical trials with large samples should be performed in future.

Conclusion

Our study showed for the first time that the rSO2 decreased dramatically during totally thoracoscopic ablation for atrial fibrillation. The decrease in rSO2 was positively influenced by changes in PaO2/FiO2. Measurements that increase PaO2/FiO2 probably improve the rSO2 performance during the surgery.

Declarations

Not Applicable

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Availability of data and materials

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

GL designed the study, conducted the study and drafted the manuscript. SS designed the study, conducted the study, and collected the data. LY conducted the study and collected the data. YS performed the data analysis. XS revised the manuscript and approved the final version. All authors read and approved the final manuscript.

Ethics approval and consent to participate

This study was approved by the institutional review board of Xinhua hospital and written informed consent was received from each patient the day before the surgery.

Consent for publication

Not applicable.

Competing interests

The authors declare that there are no conflicts of interest.

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Table 1. Demographic And Clinical Characteristics Of Patients

Gender, male/female, No.	25/20
Age (y)	62 ± 8
Weight (kg)	66 ± 9
Height (cm)	163 ± 6
BMI (kg/m^2)	25 ± 2
Classification of atrial fibrillation (AF), No. (%)	
Paroxysmal AF	34 (76)
Persistent AF	7 (15)
Longstanding persistent AF	4 (9)
LAD (mm)	41.9 ± 4.8
LVEF (%)	60.9 ± 4.7
NYHA class, No. (%)	
I	3 (7)
II	34 (76)
III	8 (17)
ASA physical status, No. (%)	
I - II	5 (11)
III	37 (82)
IV	3 (7)
Comorbidies, No. (%)	
Hypertension	21 (47)
Coronary heart disease	6 (13)
Congestive heart failure	1 (2)
Obstructive sleep apnea	3 (7)
Cerebral infarction	6 (13)
Diabetes mellitus	9 (20)
History of catheter ablation	10 (22)
Medications, No. (%)	
Anti-arrhythmic drugs	
Amiodarone	13 (29)
Digoxin	4 (9)
β-Blockers	16 (36)
Calcium channel blockers	9 (20)
Anticoagulants, No. (%)	
Warfarin	6 (13)
Aspirin	10 (22)
Clopidogrel	4 (9)
ACEI or ARB, No. (%)	15 (33)
Diuretics, No. (%)	10 (22)

Data were presented as mean ± standard deviation or the number (percentage) of patients. BMI = body mass index; LAD = left atrial diameter; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; ASA physical status = American Society of Anesthesiologists *physical status*; ACEI = angiotensin-converting enzyme inhibitors; ARB = angiotensin receptor blocker.

Table 2. Rso2 And Hemodynamic Changes At Six Time Points Of Surgery

	T0	T1	T2	T3	T4	T5
$_2$ (%)	65.3 ± 3.9	65.6 ± 4.0	65. 0 ± 3.7	57.5 ± 6.5$^{***, ###}$	63.5 ± 4.0	64.6 ± 3.7
	79 (69, 95)	65 (56,78) ***	70 (63, 82)	74 (63, 91)	79 (66, 90)$^{##}$	75 (70, 84) $^{#}$
m)						
	139 (126, 152)	96 (88, 111) ***	108 (97, 125) **	52 (45, 62) ***, ###	87 (79, 95) ***	103 (97, 118) ***
ı Hg)						
	74 ± 12	60 ± 9***	64 ± 10***	40 ± 8$^{***, ###}$	56 ± 9***	65 ± 8**
ı Hg)						
P	97 ± 15	74 ± 12***	80 ± 12***	44 ± 8$^{***, ###}$	67 ± 9$^{***, #}$	79 ± 9***
ı Hg)						
	-	8 (7, 11)	12 (11, 15) ###	8 (7, 10)	16 (12, 18) ###	13 (12, 14) ###
H_2O)						
AP	-	18 (16, 21)	24 (21, 28)$^{###}$	14 (13, 17)	27 (26, 31)$^{###}$	22 (19, 24)$^{##}$
ı Hg)						
VP	-	11 (9, 12)	16 (13, 19) ###	8 (6, 9)	19 (18, 22) ###	14 (12, 16) ##
ı Hg)						
	-	2.1 ± 0.4	2.1 ± 0.4	2.2 ± 0.3	2.2 ± 0.4	2.3 ± 0.3
nin/m^2)						
I	-	103 (80, 131)	113 (95, 136)	84 (69, 108) $^{#}$	97 (80, 113)	96 (80, 111)
ı/s/m^2/cm^{-5})						
I	-	896 ± 254	924 ± 243	479 ± 118$^{###}$	668 ± 132$^{###}$	827 ± 196
ı/s/m^2/cm^{-5})						
I/SVRI	-	0.12 (0.09, 0.16)	0.12 (0.10, 0.15)	0.18 (0.14, 0.23) ###	0.15 (0.12, 0.19)	0.11 (0.10, 0.14)

Data were presented as mean ± standard deviation or median with interquartile range. rSO_2 = regional cerebral oxygen saturation; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean blood pressure; CVP = central venous pressure; mPAP = mean pulmonary artery pressure; PAWP = pulmonary artery wedge pressure; CI = cardiac index; PVRI = pulmonary vascular resistance index; SVRI = systemic vascular resistance index. T0 = at baseline; T1 = 15 min after anesthesia induction; T2 = 15 min after one-lung ventilation; T3 = after right pulmonary vein ablation; T4 = after left pulmonary vein ablation; T5 = 15 min after two-lung ventilation. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ *versus* T0, # $P < 0.05$, ## $P < 0.01$, ### $P < 0.001$ *versus* T1.

Table 3. Blood Gas Changes At Six Time Points Of Surgery

	T0	T1	T2	T3	T4	T5
H	7.42 ± 0.02	7.40 ± 0.04***	7.38 ± 0.06***	7.34 ± 0.06***, ###	7.35 ± 0.06***, ###	7.37 ± 0.06***, ##
$PaCO_2$	39 ± 4	40 ± 5	43 ± 6*	47 ± 8***, ###	46 ± 7***, ###	42 ± 5
mm Hg)						
PaO_2/FiO_2	380 (363, 429)	388 (341, 424)	76 (71, 92)***, ###	75 (62, 90)***, ###	128 (90, 155)***, ###	329 (258, 389)
SpO_2 (%)	96 (96, 97)	100 (99, 100)***	97 (96, 99)###	93 (88, 96)###	99 (97, 99)**, ##	99 (99, 100)***
b (g/L)	15.1 ± 1.8	14.2 ± 1.7***	14.0 ± 1.6***	13.9 ± 1.7***	13.8 ± 1.6***, #	13.3 ± 1.5***, ###
Lac	1.8 (1.4, 2.4)	1.8 (1.2, 2.2)	1.7 (1.3, 2.1)	1.9 (1.7, 2.6)	2.1 (1.6, 2.7)	2.5 (1.5, 2.9) #
mmol/L)						
E	-0.6 (-1.25, 1.1)	-0.3(-1.4, 1.2)	-0.2 (-1.3, 0.9)	-1.1 (-2.2, 0.7)**	-0.7 (-2.0, 0.1)*	-1.7 (-2.5, -0.8)***, ###
mmol/L)						
SvO_2 (%)	-	77 (71, 81)	73 (69, 77)	61 (57, 64)###	76 (72, 80)	79 (75, 82)

Data were presented as mean ± standard deviation or median with interquartile range. $PaCO_2$ = arterial carbon dioxide tension; PaO_2/FiO_2 = ratio of arterial oxygen pressure to fractional inspired oxygen; SpO_2 = arterial oxygen saturation; Hb = hemoglobin; Lac = lactate; BE = base excess; SvO_2 = mixed venous oxygen saturation. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ *versus* T0, # $P < 0.05$, ## $P < 0.01$, ### $P < 0.001$ *versus* T1.

Table 4. Associations Between △rso2 And Changes In Hemodynamic And Laboratory Parameters

	Univariate Regression		Multivariate Regression	
	r	*P*	β	*P*
△pH	-0.371	0.012	-0.171	0.238
△$PaCO_2$	0.276	0.066	0.186	1.301
△PaO_2/FiO_2	0.332	0.026	0.026	0.025
△HR	0.27	0.073	-0.009	0.951
△CI	-0.228	0.132	-0.054	0.713
△PVRI/SVRI	-0.216	0.153	-0.052	0.724
△SBP	0.051	0.74		
△DBP	0.128	0.409		
△MAP	0.107	0.483		
△CVP	0.08	0.6		
△mPAP	-0.119	0.438		
△PAWP	0.014	0.925		
△PVRI	-0.032	0.833		
△SVRI	0.143	0.350		
△SaO_2	-0.149	0.328		
△SvO_2	-0.045	0.771		
△Hct	0.017	0.909		
△Hb	0.041	0.787		
△BE	0.131	0.389		
△Lac	0.046	0.765		