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Left ventricular efficiency after ligation of patent ductus arteriosus for premature infants

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Abstract

Objective: The purpose of this study was to evaluate the hemodynamic changes in the left ventricular function before and after patent ductus arteriosus ligation in premature infants with regard to the energetic efficiency of left ventricular pumping.

Methods: Thirty-five premature infants who underwent patent ductus arteriosus ligation were enrolled in this study. The left ventricular efficiency was evaluated at four points: within 24 hours before patent ductus arteriosus ligation, within 24 hour after patent ductus arteriosus ligation, between postoperative days 2 and 4, and on postoperative day 7. The indices of contractility (Ees) and afterload (Ea) were approximated on the basis of the systemic blood pressure and systolic or diastolic left ventricular volume. The ratio of stroke work and pressure-volume area, representing the ventricular efficiency, was estimated using the theoretical formula: The ratio of stroke work and pressure-volume area = 1 / (1 + 0.5 Ea/Ees).

Results: The left ventricular efficiency was transiently deteriorated within 24 hours after patent ductus arteriosus ligation due to the marked increase of the afterload and the slight increase of contraction, and then recovered to the pre-operation levels by 2-4 days after patent ductus arteriosus ligation.

Conclusions: The analysis of indices representing the afterload, contractility and energetic efficiency of left ventricle could provide practical information for the management of premature infants during the postoperative period after patent ductus arteriosus ligation.

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Ultramini-Abstract

We investigated the hemodynamic changes in the left ventricular function before and after patent ductus arteriosus ligation in premature infants with regard to the energetic efficiency. The left ventricular efficiency was transiently deteriorated after surgery, and then recovered to the pre-operation levels by 2-4 days after surgery.

INTRODUCTION

Patent ductus arteriosus (PDA) is one of the most critical problems of the respiratory and circulatory system that can occur during the first few weeks after birth in premature infants.[1] The occurrence of PDA in premature infants increases with a lower gestational age and lower birth weight. In the EPICure cohort, the occurrence of PDA was about 65% for infants who were born at less than 28 weeks of gestational age, and was around 80% for infants whose birth weight was less than 800 g.[2,3] The first-line treatment or prophylaxis for PDA is administration of indomethacin, and the second-line treatment is surgical ligation. The surgical procedure has been safely carried out by pediatric cardiothoracic surgeons, nevertheless, the postoperative intensive management is sometimes complicated because the pulmonary vascular compliance, contraction power of the cardiac muscle and the renal function are physiologically changing during the few weeks after birth.[4]

For the evaluation of the hemodynamic change before and after PDA ligation, the previous reports investigated the left ventricular (LV) function by echocardiogram using the indices of ejection fraction (EF), shortening fraction, mean velocity of circumferential fiber shortening, end-systolic wall stress, or systemic vascular resistance,[5-8] or those by the tissue Doppler imaging.[9] These reports demonstrated that the increase of afterload after PDA ligation influenced LV function.[5-7] These reports, however, investigated only hemodynamic changes within 24 hours of PDA ligation, and dynamic changes in the LV efficiency after PDA ligation have not been sufficiently studied. The

other study using an animal model showed that PDA ligation for premature baboon neonates increased the afterload and decreased of LV function as evaluated by the index of shortening fraction.[10] As a new evaluation method of hemodynamics, the concept of energy efficiency has been recently applied for the patients who had cardiac surgery. The indices of end-systolic elastance (Ees), effective arterial elastance (Ea) and ventriculoarterial coupling (Ea/Ees) were simply calculated from blood pressure and measurements by echocardiogram, and provided a useful framework for investigating ventricular performance.[11] Using these indices, the previous reports studied the ventricular efficiency before and after aortic valve replacement,[12] mitral valve surgery[13] or for congenital heart diseases.[14-16]

The purpose of this study was to evaluate the hemodynamic changes in the LV function before and after PDA ligation in premature infants with regard to the energetic efficiency of LV pumping.

METHODS

Patient Information

Eight hundred forty-seven premature infants (gestational age less than 36 weeks) were admitted to Kyushu University Hospital from January 2003 to December 2010. The cases

accompanied with congenital heart diseases and chromosomal disorders, or in poor clinical condition, such as those with severe respiratory syndrome, bacterial sepsis or necrotic enterocolitis, were excluded from this study. All of the premature infants born at less than 25 weeks of gestational age without an apparently severe critical condition were routinely administrated prophylactic indomethacin after birth three times: 0.1 mg/kg/6h at 12–15 hours after birth, 0.1 mg/kg/6h 24 hours after the first administration, 0.1 mg/kg/6h 24 hours after the 2nd administration. The infants born at 25 weeks and more than 25 weeks of gestational age were treated with indomethacin only when clinical symptoms were observed such as tachypnea, tachycardia and a reduction of urine output due to PDA. The dose of indomethacin was 0.1mg/kg/6h; and if the PDA did not close, the same dose was administered 2nd and 3rd time, 24 hours after the first and the 2nd treatments, respectively. If the indomethacin treatment did not effectively close the PDA and symptomatic heart failure persisted due to the presence of PDA, surgical PDA ligation was carried out. Informed consent for the surgery was obtained from the parents of all patients. All retrospective data used in this study were obtained by means of charts and electronic database reviews.

Surgical Technique

PDA ligation was performed using the standard methods. Anesthesia was conducted in intubated infants by a standard technique, with intravenous infusions of fentanyl and the muscle

relaxant, pancuronium. The patients were placed in a right lateral position, and a standard left postero-lateral thoracotomy was performed through the 3rd intercostal space. A single ligation of the PDA was performed with a silk suture.

Analysis of Cardiovascular Function by Echocardiogram

The LV efficiency before and after PDA ligation was evaluated by transthoracic echocardiogram at four points: within 24 hours before PDA ligation, within 24 hours after PDA ligation, between postoperative days 2 and 4 (POD 2-4), and on POD 7. The LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV) were calculated by the Teichholz M-mode method based on the data about the LV end-diastolic dimension and the end-systolic dimension obtained from the echocardiogram.[17] The LV end-diastolic volume index (LVEDVI), end-systolic volume index (LVESVI) and EF were calculated as follows: LVEDVI (ml/m^2) = LVEDV/body surface area (BSA), LVESVI (ml/m^2) = LVESV/BSA , EF (%) = (1-LVESV/LVEDV) × 100. The arterial blood pressure was measured by the Korotkoff technique using the manchette method. The indices of contractility (Ees) and afterload (Ea) were calculated on the basis of the systemic blood pressure and cardiac volume data using the approximation method, as described previously.[18] The approximation of the Ees and Ea was performed as follows: Ees = mean blood pressure /minimal LV volume; Ea = maximal LV pressure / (maximal LV volume - minimal LV volume). The mean blood

pressure was calculated as: mean blood pressure = (systolic blood pressure + diastolic blood pressure×2) / 3. The maximal LV pressure was approximated by the systolic blood pressure. The maximal LV volume was defined as being equal to the LVEDV and the minimal LV volume was defined as being equal to the LVESV. The Ea/Ees indicates the ventriculoarterial coupling between the LV and the arterial system.[11] The ratio of stroke work and pressure-volume area (SW/PVA) represents the LV efficiency, which was estimated using the theoretical formula: SW/PVA = 1 / (1 + 0.5 Ea/Ees).[19]

Statistical Analysis

The values are presented as the mean values ± SD. An analysis of variance with repeated measures on one factor was used for the variables measured at the four points (within 24 hours before PDA ligation, within 24 hours after PDA ligation, between POD 2-4 and on POD 7). The Student-Newman-Keuls test was used as a post-hoc test.

RESULTS

Clinical Manifestations

Among the 847 premature infants, 98 infants were treated with indomethacin for the closure of or prophylaxis for PDA and 63 infants of these PDA cases were closed by 5.8 ± 4.1 days of age. There were 35 patients with persistent PDA even after indomethacin treatment, and surgical PDA ligation were carried out for all of these infants. The mean ± SD gestational age of these infants was 27.2 ± 4.4 weeks, and their birth weight was 934 ± 499 g. The narrowest PDA diameter was 2.43 ± 1.08 mm. The blood-flow speed in PDA was 2.16 ± 0.80 m/sec. Surgical PDA ligation was performed at 23.5 ± 11.6 days of age. The body weights of the infants at PDA ligation were 978 ± 504 g. The length of the operation was 37.2 ± 9.3 minutes. No critical complications developed in any of the patients during the operation (Table 1). Eighteen of 35 patients were treated with dopamine or dobutamine before surgery to assist cardiac function. After surgery, all patients including these 18 were given a dopamine or dobutamine infusion at a dose of 3-5 μg/kg/min for at least 24 hours; but vasodilating agents, such as phosphodiesterase 3 inhibitors, were not used. Eighteen patients were managed under mechanical ventilation before the surgery, and all of the patients were controlled under mechanical ventilation for at least for 3 days after the surgery. The detailed data on circulatory and respiratory supports for the patients before and after PDA ligation are shown in Table 2. No major complications occurred in any of the patients after surgery, such as pneumothorax, chylothorax, hemothorax, hypotension, vocal cord paralysis or infection. Clinical data such as blood pressures, heart rates, SpO_2, urine output or blood pH are shown in Table 3. Soon after the operation, the blood pressures were remarkably increased whereas other parameters, such as

heart rates or SpO_2, were not significantly changed.

Twenty-seven premature infants, whose ductus arteriosus was closed spontaneously, were also studied and compared with the 35 infants for whom surgical PDA ligation were carried out, and they are shown as Controls in Table 1. There were no significant differences in the clinical characteristics between them.

Hemodynamics

The points evaluated by echocardiogram were as follows: within 24 hours before PDA ligation (mean ± SD : 8.4 ± 5.8 h, range : 3-21 h), within 24 hours after PDA ligation (mean ± SD : 5.5 ± 4.0 h, range : 1-18 h), between POD 2-4 (mean ± SD : 70.6 ± 14.6 h, range : 49-92 h), on POD 7 (mean ± SD : 164 ± 2.6 h, range : 160-169 h). For the evaluation at the third point (POD 2-4), about two-third of the patients (25/35) were examined. As shown in the Table 4, the left ventricular EF was decreased significantly within 24 hours after the operation (from 74.1 ± 10.2 % to 63.5 ± 9.8 %, $p<0.05$), but the EF was recovered to the pre-operation level by POD 2-4 in most of the patients, and was gradually increased until POD 7 (75.6 ± 6.4 % and 79.5 ± 7.6 %, respectively). The Ea index was increased significantly within 24 hours after PDA ligation (from 1.25 ± 0.53 to 2.66 ± 0.96 $mmHg/ml/m^2$, $p<0.05$), but was gradually decreased following that time point (at POD 2-4: 2.26 ± 0.72 $mmHg/ml/m^2$, at POD 7: 2.01 ± 0.83 $mmHg/ml/m^2$). The Ees index was slightly increased

within 24 hours after PDA ligation, but the increase was not statistically significant (from 2.46 ± 1.88 to 2.92 ± 1.95 mmHg/ml/m^2), whereas the index was drastically increased at POD 2-4 (4.64 ± 1.22 mmHg/ml/m^2) and was maintained at the same level until POD 7 (4.45 ± 2.87 mmHg/ml/m^2). The SW/PVA ratio was decreased significantly within 24 hours after PDA ligation (from 76.3 ± 8.0 % to 66.6 ± 11.2 %), but was recovered to the pre-operation level by POD 2-4, and the ratio was maintained until POD 7 (78.7 ± 7.8 % and 78.6 ± 8.2 %, respectively). All of the hemodynamic data at POD 7 were almost the same as those of the Controls. Since the hemodynamic change within 24 hours might be variable, we re-evaluated the hemodynamic indices in all of the patients divided by the first evaluation time within 24 hours after PDA ligation. As a result, there were no significant changes in the indices among the groups (Supplemental Table 1). We assessed the circulatory and respiratory findings in between those under myocardial depression (Ea/Ees ≥1.0) or not (Ea/Ees <1.0) and found that conditions under circulatory and respiratory support did not have any significant differences between the groups within 24 hours after PDA ligation (Supplemental Table 2).

DISCUSSION

The presence of PDA in infants may play an important role in their adaptation to the hemodynamic changes after birth. The PDA can maintain adequate arterial oxygen tension[20] and protect the immature ventricle against afterload by maintaining low vascular resistance for a few

hours after delivery in premature infants.[10] On the other hand, a persistent PDA causes an inadequate blood flow balance in the pulmonary and systemic circulation, causing various clinical problems. PDA closure at an appropriate time is thus needed for premature infants. Some of them require surgical intervention, but postoperative LV hypofunction sometimes occurs after PDA ligation.[21] To improve the safe management of premature infants after PDA ligation, we herein investigated the time-course of hemodynamic changes before and after PDA ligation with regard to the energy efficiency of the LV.

The energetic efficiency of the heart is defined as the ratio of stroke work (SW)[24] to myocardial oxygen consumption. The oxygen consumption is linearly correlated with the pressure-volume are (PVA),[23] hence, the energetic efficiency can be estimated from the ratio of the SW to the PVA. The Ea/Ees represents the ventriculoarterial coupling between the left ventricle and arterial system, and indicates the balance between contractility and afterload. The energetic efficiency (SW/PVA) was simply calculated from the Ea/Ees.[19] Theoretical study and clinical data for the patients with normal heart function showed that the SW of ventricular contraction was maximized when Ea/ Ees = 1 under a constant level of myocardial oxygen consumption,[24] whereas the energetic efficiency of ventricular contraction was maximized when Ea/Ees = 0.5.[11] The present study showed that the Ea/Ees index was over 1.0 within 24 hours after PDA ligation, indicating that the LV efficiency was impaired soon after PDA ligation. The LV contraction (Ees) drastically improved by 2-4 days after PDA ligation, whereas the afterload (Ea) gradually recovered, but was still

at a higher level 7 days after ligation than that in the pre-operation state. In contrast, the transient deterioration of the LV energy efficiency after PDA ligation was recovered within 2-4 days after ligation. Hence, these parameters made us possible to assess the hemodynamic changes of LV contraction, afterload and energy efficiency of the LV, separately.

In the present study, none of 35 premature infants showed any respiratory complications or systemic hypotension after PDA ligation. If the patients have any pre- or post-operative complications which may affect their systemic hemodynamics, the present data would be helpful for determining the drug indications. For example, in cases of systemic hypotension within 24 hours after PDA ligation, inotropic agents such as dopamine or epinephrine should be used to increase LV contractility (Ees) in addition to phosphodiesterase inhibitors to decrease the afterload (Ea). Beginning 24 hours after PDA ligation, vasodilators to decrease the afterload would be the essential and first-line treatment.

Therefore, the findings in this study provide useful information for the critical management of the circulatory and respiratory conditions for premature infants after PDA ligation. However, there were several limitations to this study. First, the accuracy of the approximation of the Ea and Ees on the basis of echocardiography in the premature infants has not been evaluated. Thus, a comparison of data obtained from a catheter system[25] and by echocardiography at each time point, if possible, would be useful in a future study. Second, we did not assess any premature infants without PDA, therefore, further study for those will be important to uncover the particular effect by existence

of PDA. Third, we did not take into consideration the possible modification of the indices by anesthesia. Since the anesthetizing agents, fentanyl and pancronium, could alter the LV contraction and afterload, further study to evaluate the effects of these drugs would be important. In addition, we did not discuss the effect of preload because the energy efficiency of the LV (Ea, Ees) is theoretically independent of the preload. Nevertheless, it was likely that preload could influence the LV contraction, then further comprehensive evaluation of the preload by the standard methods, such as the early and atrial mitral inflow velocities,[6] in combination with the energy efficiency would give us much information to reveal detailed cardiovascular status before and after PDA ligation. Finally, we did not have data from a conductance catheter technique; hence a comparison of data obtained from a catheter system and by echocardiography at each time point, if possible, would be useful in a future study.

In conclusion, we investigated the LV function in terms of the energetic efficiency before and after PDA ligation. This study showed that the LV efficiency was transiently deteriorated within 24 hours after PDA ligation due to a marked increase of the afterload and a slight increase in contractility, and then recovered to the pre-operation levels by 2-4 days after PDA ligation. The consecutive evaluations of the afterload, contractility and the energetic efficiency of LV function, may provide practical information for the management for premature infants during the post-operative period following PDA ligation.

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TABLE 1. The characteristics of the patients

	Total number N=847	Patients with PDA			Controls N=27
		Total N=98	Closed by indomethacin N=63	Closed by ligation N=35	
Male/Female	461/386	51/47	32/31	19/16	14/13
Gestational age (week)	31.4±3.2	26.9±4.2	26.8±4.3	27.2±4.4	27.3±4.6
Birth weight (g)	1588±518	938±441	940±423	934±499	980±503
RDS (%)	197 (23.2)	57 (59.1)	46 (74.1)	12 (36.3)	14 (51.8)
CLD (%)	89 (10.5)	38 (38.7)	27 (43.1)	11 (33.3)	10 (37.0)
ICH (%)	6 (0.69)	6 (7.1)	6 (10.3)	1 (3)	2 (7.4)
NEC (%)	4 (0.48)	2 (2.0)	2 (3.4)	0 (0)	1 (3.7)
ROP (%)	13 (15.7)	40 (40.8)	27 (43.1)	13 (39.3)	13 (48.1)
Death (%)	5 (5.4)	1 (1.0)	1 (1.7)	0 (0)	0 (0)
PDA closure					
age (days)			5.8±4.1	23.5±11.6	
body weight (g)			941±445	978±504	
Narrowest PDA diameter (mm)				2.43±1.08	
The blood-flow speed in PDA (m/s)				2.16±0.80	

Data are shown as the mean ± standard deviations (SD).

RDS, respiratory distress syndrome; *CLD*, chronic lung disease; *ICH*, intracerebral hemorrhage; *NEC*, necrotizing enterocolitis; *ROP*, retinopathy of prematurity.

TABLE 2. Circulatory and respiratory supports before and after PDA ligation

	within 24 hour before PDA ligation	within 24 after PDA ligation	Post-operative day 2-4	Post-operative day 7
Mean doses of catecholamines	1.66±1.77 (N=18)	3.30±0.98 * (N=35)	0.36±1.12 * (N=4)	0.08±0.50 * (N=1)
Dopamine (μg/kg/min)				
Dobutamine (μg/kg/min)	0.16±0.69 (N=2)	0.30±1.06 (N=4)	0.07±0.48 (N=1)	0.0 (N=0)
Mean doses of Intravenous fluid administration (ml/kg/hr)	4.53±1.71 (N=35)	4.61±1.71 (N=35)	4.80±1.22 (N=25)	5.76±1.22 * (N=35)
Mechanical ventilation	(N=18)	(N=35)	(N=17)	(N=17)
Mean FiO_2	0.24±0.05	0.25±0.08	0.22±0.02	0.22±0.03
SIMV	(N=8)	(N=24)	(N=8)	(N=6)
RR (/min)	35.5±9.1	33.7±7.9	31.2±8.7	26.7±8.7 *
PIP (mmHg)	20.1±3.60	18.9±4.33	15.5±4.65 *	16.6±5.3
PEEP (mmHg)	4.84±0.80	4.90±0.42	4.63±0.67	4.60±0.69
HFO	(N=10)	(N=11)	(N=9)	(N=11)
MAP (mmHg)	10.2±0.63	10.2±1.19	9.80±1.39	9.45±1.50
SV (ml)	10.5±1.58	10.0±1.00	9.50±1.71	9.36±1.91

Data are presented as mean±SD, N number of the treated patients.

* $p<0.05$ vs. before PDA ligation

FiO_2, fractional inspired oxygen concentration; *SIMV*, synchronized intermittent mandatory ventilation; *RR*, respiratory rate; *PIP*, peak inspiratory pressure; *PEEP*, positive end-expiratory pressure; *HFO*, high-frequency oscillation; *MAP*, mean airway pressure; *SV*, stroke volume.

TABLE 3. The clinical data before and after PDA ligation

	within 24 hour before PDAligation	within 24 after PDA ligation	Post-operative day 2-4‡	Post-operative day 7
Systolic BP (mmHg)	54.6±7.89	59.1±11.9	61.4±8.46 *	60.7±10.5 *
Diastolic BP (mmHg)	28.3±7.37	36.4±7.43 *	37.8±7.73 *	38.0±10.3 *
Mean BP (mmHg)	36.8±6.72	43.2±8.38 *	45.2±6.95 *	45.4±10.2 *
Heart rates (bpm)	152±8.53	153±8.79	148±8.75	148±8.50
SpO_2 (%)	96.6±2.88	96.7±2.86	94.1±16.4	97.4±1.71
Blood gas data				
pH	7.38±0.07	7.37±0.07	7.40±0.05	7.39±0.06
BE	1.95±4.28	1.00±4.32	3.47±3.48	1.80±2.24
Lactate (mmol/L)	1.72±0.80	1.68±0.91	1.44±0.47	0.71±1.71
Urine output (ml/kg/hr)	3.37±1.30	4.05±2.27	4.21±1.49	4.45±1.46 *

Data are presented as the mean±SD.

* $p<0.05$ vs. before PDA ligation

‡ Twenty-five in 35 patients were evaluated.

SpO_2, saturation of peripheral oxygen; *BE*, base excess.

TABLE 4. The hemodynamic data before and after PDA ligation

	within 24 hour before PDA ligation	within 24 hour after PDA ligation	Post-operative day 2-4‡	Post-operative day 7	Controls
LVEDVI (ml/m^2)	70.8±37.9	49.6±30.3 *	41.7±23.3 *	48.0±31.1 *	49.8±10.5 *
LVESVI (ml/m^2)	21.7±15.2	22.2±16.3	12.5±8.0 * †	14.2±10.2 * †	14.9±7.4 * †
EF (%)	74.1±10.2	63.5±9.8 *	75.6±6.4 †	79.5±7.6 †	80.1±10.0 †
Ea ($mmHg/ml/m^2$)	1.25±0.53	2.66±0.96 *	2.26±0.72 *	2.01±0.83 *	2.11±0.77 *
Ees ($mmHg/ml/m^2$)	2.46±1.88	2.92±1.95	4.64±1.22 * †	4.45±2.87 * †	4.52±2.73 * †
Ea/Ees	0.64±0.28	1.10±0.57 *	0.56±0.42 †	0.56±0.28 †	0.55±0.23 †
SW/PVA (%)	76.3±8.0	66.6±11.2 *	78.7±7.8 †	78.6±8.2 †	79.2±10.5 †

Data are presented as the mean±SD.

＊p<0.05 vs. before PDA ligation **† p<0.05 vs. within 24 hour after PDA ligation**

‡ Twenty-five in the 35 patients were evaluated.

SUPPLEMENTAL TABLE 1. The hemodynamic data within 24 hours after PDA ligation

	Within 2 hour (N=23)	At 2-6 hour (N=4)	At 6-9 hour (N=1)	At 12-18 hour (N=7)
Ea (mmHg/ml/m^2)	2.44±0.97	2.50±0.45	2.13	2.10±0.64
Ees (mmHg/ml/m^2)	2.79±1.46	2.32±1.03	2.18	1.86±1.07
Ea/Ees	1.03±0.46	1.12±0.29	0.98	1.16±0.55
SW/PVA (%)	67.6±10.0	64.4±6.21	67.2	64.8±10.8

Data are presented as the mean±SD.

SUPPLEMENTAL TABLE 2. Comparison of circulatory and respiratory supports within 24 hour after PDA ligation between the patients with Ea/Ees ≥1.0 and < 1.0.

	Ea/Ees ≥1.0 (N=18)	Ea/Ees < 1.0 (N=17)
Mean doses of catecholamines (N)	(18)	(17)
Domamine (μg/kg/min)	3.40±0.82	3.18±1.16
Dobutamine (μg/kg/min)	0.55±1.39	0.00±0.00
Mechanical ventilation (N)	(18)	(17)
FiO_2	0.25±0.06	0.26±0.10
SIMV (N)	(10)	(13)
RR (/min)	31.5±7.47	35.5±8.15
PIP (mmHg)	19.3±4.19	18.5±4.60
PEEP (mmHg)	4.91±0.31	4.91±0.51
HFO (N)	(8)	(4)
MAP (mmHg)	10.1±0.75	10.4±1.67
SV (ml)	10.5±1.04	9.40±0.54
Blood gas data		
pH	7.38±0.07	7.34±0.01
BE	2.69±3.72	0.08±4.29
Lactate (mmol/L)	1.85±1.07	1.50±0.71

Data are presented as the mean±SD

N, number of treated patients; *FiO_2*, fractional inspired oxygen concentration; *SIMV*, synchronized intermittent mandatory ventilation; *RR*, respiratory rate; *PIP*, peak inspiratory pressure; *PEEP*, positive end-expiratory pressure; *HFO*, high-frequency oscillation; *MAP*, mean airway pressure; *SV*, stroke volume; *BE*, base excess.

Abbreviation

PDA	patent ductus arteriosus
LV	left ventricule
EF	ejection fraction
Ees	end-systolic elastance
Ea	effective arterial elastance
Ea/Ees	ventriculoarterial coupling
POD	postoperative days
LVEDV	left ventricular end-diastolic volume
LVESV	left ventricular end-systolic volume
LVEDVI	left ventricular end-diastolic volume index
LVESVI	left ventricular end-systolic volume index
SW/PVA	the ratio of stroke work and pressure-volume area
SW	stroke work
PVA	pressure-volume area