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Research article

Effect of static lung expansion on pulmonary function following cardiopulmonary bypass in children

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ABSTRACT

Objective: To observe the effect of the lung-protective ventilation strategy, static lung expansion, during cardiopulmonary bypass (CPB) on pulmonary function and tracheal intubation time following cardiac surgery in children.

Methods: A total of 48 child patients (aged 1–3) with ventricular septal defect (VSD) were enrolled, and all underwent CPB cardiac surgery for the first time. The patients were divided into two groups using the random number table method: the experimental group (Group A, $n = 30$) and the control group (Group B, $n = 18$). After terminating the mechanical ventilation during CPB, the adjustable pressure limiting valve of the anesthesia machine was adjusted in the experimental group to maintain the pressure of the breathing circuit at 5 cmH_2O, such that both lungs remained in a static expansion state. In the control group, routine mechanical ventilation was terminated as usual.

Results: When static lung expansion with a continuous positive airway pressure of 5 cmH_2O was employed in the VSD children during CPB, compared with termination of mechanical ventilation, the partial pressure of oxygen in the arterial blood increased, while the respiratory index decreased and the oxygenation index increased following the surgery.

Conclusion: In child patients undergoing VSD reparation under CPB, lung injury occurs following the procedure, and the pulmonary oxygenation function and pulmonary oxygen diffusion function decrease. When static lung expansion of 5 cmH_2O is performed during CPB, the improvement in lung function is better than that of apnea without lung expansion pressure.

1. Introduction

Cardiopulmonary bypass (CPB) is an important auxiliary method commonly used in pediatric ventricular septal defect (VSD) surgery. During the CPB procedure, the pulmonary artery stopped supplying blood, and the blood flow of the bronchial artery also decreased, which decreased the overall pulmonary flow perfusion. At the end of surgery, the pulmonary artery begins to supply blood to the lungs, which can lead to acute lung ischemia-reperfusion injury [1,2]. Pulmonary ischemia-reperfusion injury is characterized

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by endothelial dysfunction, capillary leakage and inflammation, and the damage of pulmonary capillary endothelial cells is the characteristic change of pulmonary ischemia-reperfusion injury [2]. It is common practice to cease ventilation during CPB since the pulmonary function is performed by an external gas exchanger [3]. However, terminating the ventilation during CPB will lead to atelectasis, decreased production of surfactants and increased secretion, all of which will merely aggravate any pulmonary complications [4].

There are several methods available in current clinical studies to reduce acute pulmonary ischemia-reperfusion injury in patients. Static lung dilation refers to the anesthesiologist continuously inflating the lungs with air after mechanical ventilation is terminated during CPB. By applying a certain pressure to the lungs, the anesthesiologist maintains the airway pressure at a specific level, ensuring that the alveoli are fully expanded. This helps improve gas exchange and oxygenation function. Maintaining ventilation during CPB

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Repeated Measures Analysis

Design Report

Term	Test	Power	n	N	Multiply Means By K	Std Dev of Effects (σm)	Standard Deviation (σ)	Effect Size (σm/σ)	Alpha	Beta
B1(2)	F	0.0931	24.0	48	1.0	0.06	0.63	0.089	0.05	0.91
	GG F	0.0931	24.0	48	1.0	0.06	0.63	0.089	0.05	0.91
	HF F	0.0931	24.0	48	1.0	0.06	0.63	0.089	0.05	0.91
	Box F	0.0931	24.0	48	1.0	0.06	0.63	0.089	0.05	0.91
W1(4)	F	0.8108	24.0	48	1.0	0.22	0.45	0.489	0.05	0.19
	GG F	0.8008	24.0	48	1.0	0.22	0.45	0.489	0.05	0.20
	HF F	0.8108	24.0	48	1.0	0.22	0.45	0.489	0.05	0.19
	Box F	0.5800	24.0	48	1.0	0.22	0.45	0.489	0.05	0.42
B1*W1	F	0.8108	24.0	48	1.0	0.22	0.45	0.489	0.05	0.19
	GG F	0.8008	24.0	48	1.0	0.22	0.45	0.489	0.05	0.20
	HF F	0.8108	24.0	48	1.0	0.22	0.45	0.489	0.05	0.19
	Box F	0.5800	24.0	48	1.0	0.22	0.45	0.489	0.05	0.42

n's: 32 16

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Report Definitions

Term: The identifying label for the factor or interaction. For factors, the number of levels is also given in parentheses.
Test: Identifies the test statistic for which the power is calculated.
Power: The computed power for the term.
n: The number of subjects per group.
N: The total number of subjects in the study.
K: The means were multiplied by this value.
Std Dev of Effects (σm): This value represents the magnitude of differences among the means for the term.
Standard Deviation (σ): The random variation against which σm is compared in the F test.
Effect Size (σm/σ): An index of the size of the mean differences relative to the standard deviation.
Alpha: The significance level of the test. The probability of rejecting the null hypothesis when the null hypothesis is true.
Beta: The probability of failing to reject the null hypothesis when the alternative hypothesis is true.

Fig. 1. The sample size was estimated using the "Repeat measures analysis" method.

can help prevent alveolar collapse, atelectasis, and hypoxemia [5]. In the CPB implementation guidelines included in the lung protection section, it is recommended to use ventilation (IIb/B) during the procedure [6].

Previous studies have shown that successful static lung dilation during CPB may have the following potential effects: 1. Potential improvement in postoperative recovery time: Static lung dilation can reduce postoperative lung complications by improving gas exchange and oxygenation function, thereby reducing postoperative recovery time. 2. Reduction of complications: Static lung dilation can prevent and manage postoperative lung complications, such as atelectasis and pulmonary edema, thereby reducing the incidence and severity of complications. 3. Broader implications for clinical practice guidelines: Successful static lung dilation practices can provide new evidence and recommendations for clinical practice guidelines to help physicians better manage and treat patients receiving CPBS [1,7,8]. At present, there are relatively few studies on lung protection strategies during CPB in pediatric populations [9]. Due to the differences between children's physiological characteristics and adults', specific lung protection strategies need to be developed for different age groups. The purpose of this study was to observe the effects of static lung dilation on arterial oxygenation, pulmonary ventilation function and postoperative intubation time in 1–3 year old CPB children, and to evaluate the effects of static lung dilation on pulmonary function in 1–3 year old CPB children.

2. Data and methods

2.1. Effect of static lung expansion on pulmonary function following cardiopulmonary bypass

2.1.1. Patient selection and grouping

A total of 48 child patients with a VSD undergoing reparation using CPB were enrolled. The patients were aged 1–3 years, were assessed as ASA grade III with uncorrected congenital heart malformations [10], and had a VSD of 0.6–1 cm and a body mass index of 13–18 kg/m^2. All patients underwent cardiac surgery under CPB for the first time. Prior to the operation, the patients had no anemia, fever, pulmonary infection, abnormal bleeding and coagulation dysfunction, infectious endocarditis, or liver and kidney dysfunction. The patients were divided into two groups using the random number table method: the experimental group (Group A, $n = 30$) and the control group (Group B, $n = 18$). After terminating the mechanical ventilation during CPB, the adjustable pressure limiting valve of the anesthesia machine was adjusted in the experimental group to maintain the pressure of the breathing circuit at 5 cmH_2O, such that both lungs remained in a static expansion state. In the control group, routine mechanical ventilation was terminated as usual. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Affiliated Hospital of Fujian Medical University. A written informed consent was obtained from legal guardians of all participants.

2.1.2. Sample size calculation

Using PASS16, the sample size was estimated using the "Repeat measures analysis" method. With a power of 0.8, alpha of 0.05, and a 2:1 ratio of experimental group to control group, the required number of subjects was calculated to be 48. (The calculation results of sample size are shown in Fig. 1)

2.1.3. Exclusion criteria

The exclusion criteria were as follows.

1) Patients with anemia prior to the operation, with a hemoglobin level of <10 g/dL
2) Patients with abnormal bleeding and coagulation function prior to the operation
3) Patients with a fever prior to the operation, where the axillary temperature was <37.5 °C one day before surgery
4) Patients with pulmonary infection or other infectious diseases prior to the operation
5) Patients with moderate pulmonary arterial hypertension, with a pulmonary systolic pressure of >50 mmHg.
6) Patients with pulmonary overcirculation in clinical.
7) Patients with a history of bronchial asthma or airway hyperresponsiveness
8) Patients with a history of allergy to anesthetic agents
9) Patients with combined abnormal liver function

2.1.4. Anesthesia protocol

2.1.4.1. Anesthesia induction. Peripheral venous access was established after the patients entered the operation room, and routine electrocardiogram (ECG) monitoring, pulse oxygen saturation (SpO_2), and end-expiratory partial pressure of carbon dioxide ($PECO_2$) was performed. Midazolam (0.2 mg/kg), sufentanil (0.5 μg/kg), propofol (2 mg/kg) and rocuronium (1 mg/kg) were then injected intravenously for induction. Following endotracheal intubation, the anesthesia machine was connected for mechanical ventilation, radial artery puncture and catheterization were performed. Additionally, a disposable pressure sensor device was used for invasive arterial pressure monitoring. The right internal jugular vein was punctured under ultrasound guidance to insert a venous catheter and to monitor the central venous pressure (CVP).

2.1.4.2. Maintenance of anesthesia. Propofol, midazolam, sufentanil, and rocuronium were injected intravenously to maintain the

bispectral index at between 40 and 60. Midazolam (0.05 mg/kg), sufentanil (0.5 μg/kg), rocuronium (0.3 mg/kg), and propofol (0.5–1 mg/kg) were injected intravenously at the initial stages of the sternum sawing and the CPB. After the sternum was sawn, heparin sodium (3 mg/kg) was administered via an internal jugular vein. After 5 min, 0.5 ml of venous blood was drawn for activated clotting time (ACT) monitoring.

2.1.4.3. Management of cardiopulmonary bypass. Once the ACT had reached 400s, catheterization was conducted using the ascending aorta and superior inferior vena cava, while after the ACT had reached 480s, CPB was commenced to repair the VSD. During the CPB procedure, the hematocrit was maintained at 25%–30 %, the nasopharyngeal temperature and rectal temperature were controlled at 32°C-34 °C, and the mean arterial pressure (MAP) was maintained at around 30–50 mmHg (1 mmHg = 0.133 kPa). The internal environment was adjusted according to the blood gas analysis results.

2.1.4.4. Respiratory management. Pressure-controlled volume-guaranteed ventilation was performed following endotracheal intubation. The ventilation parameters were as follows: inhalation oxygen concentration (FiO_2) = 60 %, tidal volume (VT) = 8 ml/kg, and respiratory rate = 20–35 times/min. A positive end-expiratory pressure (PEEP) of 4 cmH_2O was administered to maintain the end-tidal partial pressure of carbon dioxide ($PETCO_2$) within 35–45 mmHg. Once the CPB bypass reached full flow, the ventilator was stopped. Group A set the gas flow valve to 2L/min, adjust the exhaust valve of the ventilator, maintain the pressure of the respiratory circuit to 5 cmH_2O, and maintain the static expansion of the lung. In group B, the pressure is maintained at 0 cm H_2O. After opening the superior and inferior vena cava, the ventilator controlled the respiration and maintained the $PETCO_2$ at within 35–45 mmHg. Two hours after the patients were transferred to the intensive care unit (ICU), the FiO_2 was adjusted to 40 %, and the administration of PEEP of 4 cmH_2O was continued. The patients' ventilator adopted SIMV ventilation mode, and the tidal volume was set at 6–8 ml/kg according to the patient's specific conditions. On the second day after operation, if the patient's hemodynamics was stable, the bedside chest radiograph showed no inflammation and atelectasis, and the blood gas analysis was normal, the sedative drugs and muscle relaxants should be stopped. After the patient's spontaneous breathing recovered, the tracheal catheter was removed and non-invasive positive pressure ventilation (NIPPV) was used. The ICU doctors and nurses were blinded to the specific grouping of the study and made various clinical decisions independently.

2.1.5. Observation index and observation time point

2.1.5.1. Routine monitoring of indicators. The indicators included heart rate, invasive radial artery blood pressure (systolic blood pressure, diastolic blood pressure, MAP), CVP, ECG, SpO_2, and $PETCO_2$. The blood gas monitoring included the partial pressure of oxygen in the arterial blood (PaO_2) and partial pressure of carbon dioxide ($PaCO_2$). The Pulmonary oxygen exchange index included oxygenation index (OI), alveolar-arterial oxygen partial pressure difference (P [A-a]O_2) and the respiratory index (RI). Other indicators included ascending aorta clamping time, CPB time, postoperative tracheal intubation time, and ICU duration.

2.1.5.2. Monitoring time point. The monitoring time points were as follows.

- T0: before skin incision
- T1: 30 min after CPB was terminated
- T2: 2 h following transference to cardiac ICU (CICU)
- T3: 5 h following transference to CICU

2.1.5.3. Indicator monitoring and calculation. Arterial blood was collected at each monitoring time point for blood gas analysis, and the CVP, MAP, and PaO_2 levels at the corresponding time points were recorded. Following the operation, ascending aorta clamping time, CPB time, operation duration, postoperative tracheal intubation management time, and ICU duration were recorded.

2.1.5.4. Statistics processing. The PASS software (Power 0.9, $\alpha = 0.05$) was used to calculate the sample size. The data were analyzed using SPSS22.0 statistical software. The measurement data were expressed as mean ± standard deviation ($\bar{x} \pm SD$). An independent

Table 1
Comparison of general data, CPB time, tracheal intubation time and ICU duration between the two groups ($\bar{x} \pm SD$).

	Group A (30 cases)	Group B (18 cases)	t/X^2	P
Age (years)	1.77 ± 0.82	1.83 ± 0.92	−0.261	0.796
Height (cm)	87.93 ± 10.64	87.17 ± 11.93	0.231	0.818
Body weight (kg)	11.98 ± 2.16	11.63 ± 2.67	0.534	0.596
Gender (male/female)	19/11	11/7	0.024	0.878
Size of VSD (cm)	0.66 ± 0.06	0.65 ± 0.06	0.544	0.589
Ascending aorta clamping time (min)	34.87 ± 6.07	32.61 ± 5.15	1.317	0.194
CPB time (min)	62.73 ± 5.46	59.83 ± 5.98	1.719	0.092
Tracheal intubation time (h)	26.87 ± 4.50	25.06 ± 3.23	1.49	0.143
ICU duration (h)	53.73 ± 8.86	51.39. ± 6.36	0.98	0.332

sample *t*-test and one-way analysis of variance (ANOVA) were used to compare the patients' general conditions and various monitoring indicators at different time points between the two groups, with repeated measurement ANOVA used for intra-group comparisons of each monitoring index of the patients at different time points and the least significant difference test used for pairwise comparisons. The count data were evaluated using a X^2 test or Fisher's exact probability test. A *P*-value of <0.05 was considered statistically significant.

3. Results

1 General data

There were no significant differences in age, weight, height, gender, ascending aorta clamping time, CPB time, postoperative tracheal intubation time, and ICU duration between the two groups ($P > 0.05$) (Table 1).

2 Changes in hemodynamic indexes and partial pressure of oxygen and partial pressure of carbon dioxide

There were no significant differences in CVP, MAP and $PaCO_2$ between the two groups at each time point ($P > 0.05$) (Table 2). Compared with at T0, the PaO_2 decreased at T1, T2, and T3 in both groups, with the difference statistically significant ($P < 0.05$). The PaO_2 at T1, T2, and T3 were higher at each timepoint for Group A compared to Group B, and the difference was statistically significant ($P < 0.05$) (Table 2).

3 Pulmonary oxygen exchange index

Compared with T0, the RI increased and the OI decreased at T1, T2, and T3 in both groups, with the differences statistically significant ($P < 0.05$). The P (A-a)O_2 and RI being relatively lower and the OI being relatively higher at T1, T2, and T3 in Group A compared to Group B., with the differences statistically significant ($P < 0.05$) (Table 3).

4 Summary of results

1. In the child patients with VSD repaired under CPB, lung injury occurred following CPB, and the pulmonary oxygenation function and pulmonary oxygen diffusion function decreased compared with those before the operation, while the PaO_2 and OI decreased and the RI increased.
2. During CPB in VSD children, using static lung expansion with a continuous positive airway pressure of 5 cmH_2O resulted in higher PaO_2, lower P (A-a)O_2 and RI, and higher OI compared to terminating mechanical ventilation. This suggests that static lung expansion at 5 cmH_2O during CPB improves lung function better than apnea without lung expansion pressure.

4. Discussion

Following open heart surgery, there will inevitably be a certain degree of postoperative temporary pulmonary dysfunction. While there are still many unknown factors and mechanisms involved in this pulmonary dysfunction, a number of factors that can lead to this complication are known, including CPB, anesthesia, hypothermia, perioperative hemodynamic instability, pulmonary ischemia-

Table 2
Comparison of CVP, MAP, PaO_2 and $PaCO_2$ between the two groups at each time point (mean ± SD, 30 cases in the experimental group and 18 cases in the control group).

Group		T0	T1	T2	T3	F	P
CVP	Group A	8.90 ± 1.81	10.43 ± 1.41	10.20 ± 1.47	10.77 ± 1.36	7.126	0.001
	Group B	9.50 ± 1.69	11.12 ± 1.72	10.67 ± 1.19	10.44 ± 1.29	3.132	0.057
	F1	1.103	2.580	1.298	0.657	–	–
	P	0.260	0.115	0.260	0.422	–	–
MAP	Group A	73.73 ± 6.02	73.67 ± 5.54	75.57 ± 5.12	74.73 ± 4.93	0.862	0.473
	Group B	73.05 ± 5.92	74.22 ± 4.73	75.54 ± 4.99	75.17 ± 4.22	0.673	0.582
	F1	0.144	0.126	0.062	0.097	–	–
	P	0.706	0.724	0.804	0.757	–	–
PaO_2	Group A	253.93 ± 23.80	194.33 ± 34.48	211.20 ± 23.30	157.50 ± 19.56	246.108	<0.001
	Group B	262.56 ± 24.49	173.44 ± 32.58	194.72 ± 28.95	145.61 ± 17.77	415.402	<0.001
	F1	1.445	4.299	4.685	4.446	–	–
	P	0.236	0.044	0.036	0.040	–	–
$PaCO_2$	Group A	38.86 ± 2.96	39.41 ± 3.41	40.44 ± 3.58	39.42 ± 3.02	0.890	0.459
	Group B	38.73 ± 3.22	39.69 ± 2.88	40.34 ± 2.35	40.65 ± 1.64	1.537	0.246
	F1	0.020	0.082	0.011	2.535	–	–
	P	0.888	0.776	0.918	0.118	–	–

Notes: CVP: Central venous pressure, MAP: mean arterial pressure, HR: heart rate, PaO_2: arterial partial pressure of oxygen, $PaCO_2$: arterial partial pressure of carbon dioxide. "F" is the time effect statistic for repeated measurements, "F1" is the separate effect statistic for each time point of repeated measures,"P" is the P value obtained from their comparative analysis.

Table 3
Comparison of P (A-a)O_2, RI and OI of the two groups at each time point (mean ± SD, 30 cases in the experimental group and 18 cases in the control group).

Group		T0	T1	T2	T3	F	P
P(A-a)O_2	Group A	125.25 ± 26.54	190.34 ± 27.21	151.91 ± 21.45	93.38 ± 21.46	142.542	<0.001
	Group B	118.24 ± 31.30	206.78 ± 20.79	167.62 ± 32.15	107.03 ± 21.56	216.088	<0.001
	F1	0.685	4.858	4.130	4.535	–	–
	P	0.412	0.033	0.048	0.039	–	–
RI	Group A	0.50 ± 0.14	1.02 ± 0.26	0.73 ± 0.16	0.61 ± 0.17	76.684	<0.001
	Group B	0.46 ± 0.15	1.23 ± 0.25	0.87 ± 0.18	0.75 ± 0.20	134.703	<0.001
	F1	1.049	7.731	7.878	7.060	–	–
	P	0.31	0.008	0.007	0.011	–	–
OI	Group A	423.23 ± 39.77	323.93 ± 57.54	352.00 ± 38.88	394.00 ± 48.90	178.428	<0.001
	Group B	437.61 ± 40.83	289.00 ± 54.35	324.67 ± 48.14	364.28 ± 44.37	75.013	<0.001
	F1	1.442	4.318	4.645	4.447	–	–
	P	0.236	0.043	0.036	0.040	–	–

P(A-a)O_2: alveolar-arterial oxygen partial pressure difference, RI: respiratory index, OI: oxygenation index. "F" is the time effect statistic for repeated measurements, "F1" is the separate effect statistic for each time point of repeated measures, "P" is the P value obtained from their comparative analysis.

reperfusion process, and drugs and blood transfusion [11].

The lungs are isolated from artificial CPB, which results in blood supply to the lungs only from the bronchial arteries. The lungs have two independent circulatory systems for blood delivery. The bronchial artery contributes only 3%–5% to the pulmonary blood flow system. During CPB experiments, this flow may decrease to 1/10 of its original level, as observed in pig models [12]. In addition, alveolar collapse, changes in chest wall compliance, diaphragm dysfunction, lung and phrenic nerve injury, visceral hypoperfusion, lung ischemia/reperfusion, and protamine use are also the causes of ischemia [13]. At this stage, the mismatch between pulmonary oxygen requirement and oxygen supply may lead to oxidative stress, in which excessive reactive oxygen species are accumulated [9]. The inflammatory cascade reaction induced by CPB is also an acceptable mechanical hypothesis [14].

The patients in this study were children aged 1–3 years with VSD repaired under CPB, and all the patients underwent CPB and had the ascending aorta clamped. The results of this study revealed that all the patients had different degrees of pulmonary hypofunction following the operation. Compared with before the operation, the PaO_2 of the patients in both groups decreased at all time points following the operation, suggesting that the patients had a tendency toward hypoxia and that the pulmonary oxygen exchange function decreased.

The P (A-a)O_2 is a sensitive indicator of pulmonary ventilation insufficiency and diffusion dysfunction in the early stage, and this indicator increases in cases of ventilation/blood flow disorder, diffusion disorder, or intrapulmonary shunt increase. Meanwhile, the RI is a simple and practical index that can reflect whether the pulmonary ventilation function and oxygenation function are normal. The normal value of RI is 0.1–0.37. The larger the RI, the poorer the lung function. The OI (PaO_2/FiO_2) is an index that can reflect the efficiency of pulmonary oxygen exchange, with the attendant value easy to calculate and useful to understanding the progress of the disease and the treatment response. This study found that post-operation, both groups' RI increased and OI decreased compared to preoperative levels, indicating pulmonary oxygen exchange dysfunction, reduced efficiency, and lower diffusion function. With postoperative respiratory support and treatment, the postoperative OI gradually recovered and the respiratory function gradually improved.

The results also revealed that there was no significant difference in tracheal intubation time and ICU duration between the two groups after the patients were transferred to the ICU post-surgery. The improvement in postoperative pulmonary oxygenation function and pulmonary diffusion function via static lung expansion is relatively transient, only affecting the early postoperative pulmonary function. In this study, the static lung expansion during CPB did not significantly improve the clinical prognosis of the patients in the ICU following the operation.

Lung injury following CPB involves many factors, and the use of static lung expansion during CPB can improve the postoperative lung function to some extent, but it cannot reverse the lung injury. As the volume of the thoracic cavity of children is relatively small, slightly higher pressure may cause clear lung expansion and block the surgical field of vision, and a low static lung expansion pressure (5 cmH_2O) was thus adopted in this study. There are the following shortcomings in this study: 1. There is a lack of exploration on the optimal static pulmonary dilation pressure for pediatric cardiac surgery CPB to improve postoperative lung function, as well as the basis for determining the optimal pressure, including the factors based on which further research is needed. 2. The follow-up period of this study was relatively short, and the long-term postoperative effects were not explored. 3. This study only compared clinical phenomena and did not delve into the molecular and cellular mechanisms involved. In subsequent research, we will determine the appropriate standard range for static lung expansion based on physiological parameters and imaging evaluation of the patient, using biomechanical models for prediction and clinical trial results. Meanwhile, due to significant physiological differences among patients, it is necessary to study how to customize lung protection strategies based on their specific conditions (such as age, weight, underlying disease, CPB time, etc.). Exploring the effects of combining static lung expansion with other lung protection measures such as low tidal volume ventilation and restricted fluid management, as well as optimizing the combination of these strategies, will also be a direction for future research.

When discussing lung protection strategies during CPB, it is important to compare the effects of static lung dilation with other

strategies. For example, by comparing these strategies using lung retensioners such as surfactants, the effectiveness and suitability of static lung dilation in alleviating lung dysfunction after CPB can be determined. In addition, the risk-benefit ratio of different strategies can be evaluated to provide a basis for clinical decision-making. There are many other areas that warrant further study, such as the evaluation of long-term effects. Long-term studies can help us understand the impact of CPB on childhood growth and development and the risk of chronic lung disease in adulthood.

Any CPB-related lung injury presents a complex problem. While a ventilation strategy is not the only way to resolve this problem, it is a relatively simple procedure implemented during surgery, does not affect the operation, and does not increase the medical costs, meaning it could be popularized and applied in clinical practice.

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Data availability statement

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Ethics approval and consent to participate

This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Affiliated Hospital of Fujian Medical University. A written informed consent was obtained from legal guardians of all participants.

CRediT authorship contribution statement

Yu Huang: Writing – original draft, Resources, Formal analysis, Data curation, Conceptualization. **Guolin Lu:** Software, Resources, Data curation, Conceptualization. **Zengchun Wang:** Software, Resources. **Qing Zheng:** Software, Resources, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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