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Electrical cardiometry changes in full-term neonates with persistent pulmonary hypertension on high-frequency mechanical ventilation versus conventional mechanical ventilation

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Abstract

Background Persistent pulmonary hypertension of the newborn (PPHN) is characterized by high pulmonary vascular resistance, causing right-to-left blood shunting and respiratory distress. High-frequency oscillatory ventilation (HFOV) is used to improve oxygenation in PPHN cases. This study aimed to evaluate the hemodynamic changes in neonates with PPHN on HFOV compared to conventional mechanical ventilation (CMV) using electrical cardiometry (EC).

Methods This prospective cohort study included 58 full-term neonates with PPHN, admitted to a neonatal intensive care unit (NICU). Neonates were divided into two groups; group 1: those receiving CMV, and group 2: those switched to HFOV after CMV failure. Measurement of EC were taken at day 0, 1, 3, and 7 of MV.

Results Cardiac output, index of contractility, and stroke volume were significantly lower in the HFOV group compared to the CMV at days 0, 1, 3, and 7. However, they significantly improved in both groups, but the improvement was less significant in the HFOV group. Systemic vascular resistance and thoracic fluid content were comparable between both groups at days 0, 1, 3, and 7.

Conclusions Both HFOV and CMV improve the hemodynamic status in full-term neonates with PPHN. However, hemodynamic improvement was more obvious in the CMV group than in the HFOV group. Hence, HFOV should be used in PPHN only in severe cases.

Keywords PPHN, HFOV, CMV, Electrical cardiometry

Introduction

Persistent pulmonary hypertension of neonates (PPHN) is characterized by marked elevation of pulmonary pressure due to increased pulmonary vascular resistance (PVR) [1]. PPHN is a common cause of respiratory distress in neonates with significant morbidity and mortality

[2, 3]. It affects two in 1000 live births and is associated mostly with term or near-term infants, and mortality accounts for 10–50% [3]. PPHN is associated with several diseases such as meconium aspiration syndrome, pneumonia, pulmonary hypoplasia, and congenital diaphragmatic hernia [4].

High-frequency oscillatory ventilation (HFOV) has been used as an adjuvant for the treatment of PPHN. HFOV improves oxygenation at lower mean airway pressures in infants with PPHN compared to conventional mechanical ventilation (CMV) [5].

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There are few studies about hemodynamic effects in neonates with PPH. A better understanding of the hemodynamic effects of HFOV would facilitate clinical decision-making [6]. The hemodynamic assessment in neonates with PPHN has traditionally been restricted to heart rate, blood pressure, and oxygen saturation monitoring, along with intermittent clinical and laboratory indices of perfusion. While invasive techniques like pulmonary artery catheters (PAC) were previously used, they are impractical for neonates. Noninvasive methods such as echocardiography and electrocardiometry are increasingly being utilized [7–9].

Electrocardiometry (EC) can monitor hemodynamics and cardiac output in neonates with the advantages of being continuous, non-invasive, and operator independent. The principle of its work is to use the changes in thoracic electrical bioimpedance, which are primarily impacted by erythrocyte orientation and peak flow velocity in the ascending aorta throughout the cardiac cycle [10].

No previous study has evaluated hemodynamics in neonates with PPHN on HFOV. Therefore, this study aimed to evaluate the hemodynamic changes in full-term neonates with PPHN supported by HFOV compared to those supported by CMV using EC.

Patients and methods

This prospective cohort study was conducted on 58 full-term neonates with PPHN who were admitted to the NICU and put on CMV or HFOV as a respiratory support between December 2020 to December 2022, following ethical approval from our faculty of medicine's local Ethical Committee with ethical approval code (34,297/11/20). The caregivers provided informed written consent.

The criteria for excluding candidates for the study included neonates with major congenital anomalies, dysrhythmias, chest deformities, congenital heart disease except small defects such as patent ductus arteriosus or atrial septal defect, sepsis, perinatal asphyxia with Apgar score <5 at 5 min, hydrops fetalis, intraventricular hemorrhage (grades 3 and 4), all surgical cases, inability to tolerate adhesive skin leads of EC, and maternal diabetes.

The neonates were classified into two groups:

Group 1: included neonates with PPHN who were on CMV.

Group 2: included neonates with PPHN who were on HFOV after the failure of CMV.

CMV was considered failed when a peak inflation pressure >28 and tidal volumes >6 ml/kg are required to

improve oxygenation and keep Pa_{CO_2} less than 60 mm Hg.

All neonates underwent a comprehensive review of their medical history, clinical examination, and laboratory and radiological testing.

Chest X-ray and blood gases were performed in all included neonates. Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded at four time points; 0, 1, 3, and 7th day after ventilation.

Ventilatory settings

HFOV: (Fabian, Acutronic Medical System and SLE 5000, UK) mean airway pressure (MAP) was optimized for each neonate according to the baseline observations (begin with 1–2 cm H_2O above the pressure on the previous CMV) using an open lung approach aiming to decrease FIO_2 to below 0.4 as soon as possible. We set oscillatory frequency between 10–15 Hertz according to the patient's weight. We adjusted inspiration/expiration ratio to 1:2. Moreover, we initially adjusted amplitude as double mean level then we changed it according to the level of PCO_2 . We also adjusted tidal volume between 1.5 and 2.5 ml/kg to keep PCO_2 between 45–50 mmHg and we adjusted oxygenation by increasing MAP and FIO_2 . We knew that optimum lung volume is reached when anteroposterior chest X-ray revealed an inflation close to eighth posterior ribs.

CMV was provided by ventilators with positive pressure ventilation or trigger modes. We adjusted MAP between 20 and 25 cm H_2O , FIO_2 between 60 and 80%, positive end-expiratory pressure (PEEP) = 5 cm H_2O , ventilator rate between 40 and 60 rpm. These initial parameters were changed according to the results of blood gases and the condition of every neonate.

Electrical cardiometry

Electrical cardiometry (EC) was performed using the ICON® hemodynamic monitor (ICON Cardiotronics, Inc., La Jolla, CA, USA), and assessments were made before and at the first, third, and seventh days of ventilation and treatment. We connected the monitoring system, which uses four standard surface electrocardiogram electrodes, to the sensors placed on the neonate's forehead, lower neck, mid-axillary line, and left thigh [7].

The hemodynamic parameters measured included stroke volume (SV), cardiac output (CO), systemic vascular resistance (SVR), and contractility (ICON index). The EC measurements were made in the supine position to avoid agitation, and phototherapy was temporarily discontinued during the readings. EC operators were blinded to the study groups. The data collection followed specific protocols, such as pausing for one minute during

measurements for neonates under HFOV to maintain stable mean arterial pressure (MAP). Non-invasive blood pressure measurements were taken prior to EC readings, ensuring consistency in the data collected. The EC system continuously displayed important parameters such as cardiac output (CO), index of contractility (ICON), systemic vascular resistance (SVR), thoracic fluid content (TFC), and stroke volume variation (SVV) providing valuable insights into the neonates' cardiovascular function as described by Hsu et al. [7].

Echocardiography was performed by an expert pediatric cardiologist to measure systolic pulmonary artery pressure to monitor the improvement of the included neonates at the same time points of EC. Sildenafil was given to all included neonates in a dose of 0.5–2 mg/kg/dose up to 4 doses per day [11].

The primary outcome of the study was to assess electrocardiometry changes in neonates with PPHN on HFOV versus those on CMV. The secondary outcomes were to assess the changes in blood gases and vital signs.

Sample size calculation

The calculation of sample size was conducted using G*Power 3.1.9.2 (Universitat Kiel, Germany). Sample size calculation determined that 26 neonates were required in each group to achieve a 95% power and an alpha error of 0.05. This calculation aimed to detect a difference of 56 ml/kg/minute in the mean cardiac output (CO) between the two groups.

Statistical analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing Student's *t* test. Qualitative variables were presented as frequency and percentage (%) and were compared in both groups utilizing the chi-square test. Comparison of quantitative data in

different days was performed utilizing repeated one-way analysis of variance (ANOVA) with post hoc test (Tukey). A two-tailed *P* value <0.05 was considered statistically significant.

Results

We included 32 neonates in HFOV group and 31 neonates in CMV groups, however three neonates died early in HFOV group and two neonates died in the CMV group and they were excluded from the study as their hemodynamic measurement data were incomplete. Only 29 neonates were included in each group. Sex, gestational age, weight, and new Ballard and Down scores were comparable in the two studied groups. However, Apgar scores at 1 and 5 min were significantly lower in the HFOV group in comparison to the CMV group. Need for inotropic support was significantly higher in HFOV group compared to CMV group. The inotropes used in our neonates were dopamine and milrinone according to the protocol of our unit (Table 1).

Systolic and diastolic blood pressures were significantly lower in the HFOV group in comparison to the CMV group at days 0, 1, 3, and 7. However, PCO₂ was significantly higher in the HFOV group in comparison to the CMV group at days 0, 1, and 3 with no significant difference at day 7. HR, pH, and HCO₃ levels did not show any significant differences between the HFOV and CMV groups at any of the time points (days 0, 1, 3, and 7). In both groups, HR was insignificantly different between days 0 and 1, but significantly decreased at days 3 and 7 after ventilation. In both groups, SBP and DBP were insignificantly different between days 0 and 1 but significantly increased at days 3 and 7 after ventilation. pH and HCO₃ were insignificantly different between days 0 and 1, but they significantly increased at days 3 and 7 after ventilation. PCO₂ was insignificantly different between days 0 and 1, but it increased significantly at day 3 and day 7 (Table 2).

Table 1 Comparison of demographic and clinical characteristics between the studied groups

		CMV group (n = 29)	HFOV group (n = 29)	P
Sex	Male	15 (51.7%)	16 (55.2%)	0.792
	Female	14 (48.3%)	13 (44.8%)	
Gestational age (weeks)		38.2 ± 0.7	38.5 ± 0.6	0.091
Weight (kg)		3.3 ± 0.5	3.5 ± 0.3	0.154
APGAR	1 min	5 ± 1	4 ± 1	0.004
	5 min	8 ± 1	7 ± 0.4	< 0.001
New Ballard score (weeks)		37.2 ± 0.8	36.9 ± 0.9	0.208
Down score		7.4 ± 0.4	7.3 ± 0.2	0.117
Inotropes		13 (44.8%)	21 (72.4%)	0.033

CMV conventional mechanical ventilator, HFOV high-frequency oscillatory ventilator

Table 2 Vital signs, and ABG of the studied groups

		CMV group (n = 29)	HFOV group (n = 29)	P
Heart rate (beat/min)	0	144.7 ± 10.47	147.3 ± 7.35	0.275
	1	139.4 ± 9.6	140.24 ± 8.3	0.231
	3	126.3 ± 7.2 ^{ab}	128.2 ± 8.1 ^{ab}	0.258
	7	113.2 ± 6.7 ^{ab}	115.5 ± 5.2 ^{ab}	0.281
Systolic blood pressure (mmHg)	0	66.2 ± 4.3	60.9 ± 5.2	< 0.001
	1	66.8 ± 5.1	61.1 ± 3.9	< 0.001
	3	70.9 ± 4.9 ^{ab}	64.6 ± 4.8 ^{ab}	0.002
	7	78.6 ± 4.5 ^{ab}	74.1 ± 3.2 ^{ab}	0.02
Diastolic blood pressure (mmHg)	0	37.7 ± 5.6	32.1 ± 4.3	< 0.001
	1	39.1 ± 7.7	32.2 ± 4.9	< 0.001
	3	43.1 ± 3.7 ^{ab}	36.3 ± 5.0 ^{ab}	< 0.001
	7	48.1 ± 3.9 ^{ab}	43.4 ± 4.1 ^{ab}	0.040
PH	0	7.28 ± 0.03	7.27 ± 0.04	0.895
	1	7.31 ± 0.05	7.30 ± 0.03	0.894
	3	7.36 ± 0.05 ^{ab}	7.34 ± 0.03 ^{ab}	0.122
	7	7.40 ± 0.05 ^{ab}	7.39 ± 0.03 ^{ab}	0.122
PCO ₂ (mmHg)	0	43.07 ± 9.10	54.3 ± 6.1	< 0.001
	1	38.07 ± 11.29	54.07 ± 4.06	< 0.001
	3	38.21 ± 4.76 ^a	41.69 ± 4.93 ^{ab}	0.008
	7	39.83 ± 3.45 ^a	38.52 ± 4.34 ^{ab}	0.209
HCO ₃ (mmol/L)	0	14.60 ± 1.54	14.72 ± 3.28	0.209
	1	15.54 ± 2.21	14.62 ± 3.35	0.218
	3	19.33 ± 2.41 ^{ab}	18.61 ± 1.95 ^{ab}	0.051
	7	20.10 ± 1.91 ^{ab}	19.97 ± 1.42 ^{ab}	0.144

HFOV high-frequency oscillatory ventilator, PH potential of hydrogen, PCO₂ partial pressure of carbon dioxide, HCO₃ bicarbonate, CO cardiac output

^a Significant difference with the corresponding value at day 0

^b Significant difference with the corresponding value at day 1, conventional mechanical ventilator

Cardiac output (CO), ICON, and SV were significantly lower in the HFOV group compared to the CMV group at all time points (days 0, 1, 3, and 7). SVR, TFC, and SPAP did not show significant differences between the HFOV and CMV groups at any of the time points (days 0, 1, 3, and 7). In both groups, CO (Fig. 1), ICON (Fig. 2), TFC, and SV (Fig. 3) remained unchanged at days 0 and 1, and then increased significantly by days 3 and 7. In contrast, SPAP remained unchanged at days 0 and 1, and then decreased significantly by days 3 and 7. However, SVR showed no significant changes at any time point of follow-up in both groups (Fig. 4, Table 3).

Discussion

To the best of our knowledge, this is the first study that evaluates the hemodynamic effect of HFOV versus CMV in neonates with PPHN. We found that both ventilatory modes significantly improved PPHN and improved cardiac output, but the improvement was less significant in the HFOV group.

In the current study, HFOV group patients had significantly lower Apgar scores at 1 and 5 min than CMV group patients. This is in agreement with other studies that reported that the Apgar score at 5 min was significantly less in severe cases with PPHN and non-survivors [12, 13]. This is explained by the fact that only severe cases failed to respond to CMV and had to be shifted to HFOV.

In our study, the need for inotropic support was significantly higher in the HFOV group compared to the CMV group. We used dopamine and milrinone. Milrinone is a phosphodiesterase 3 inhibitor with lusitropic, inotropic, and vasodilator effects used in cases of severe PPHN with ventricular dysfunction, resulting in an improvement of cardiac function and CO [14]. While dopamine is a common vasopressor used in PPHN due to its vasoconstrictor effect that increases SVR and blood pressure [4]. Hemodynamics of the included neonates can be affected by the intake of these drugs; however, HFOV exhibited less improvement in hemodynamics and CO in spite of

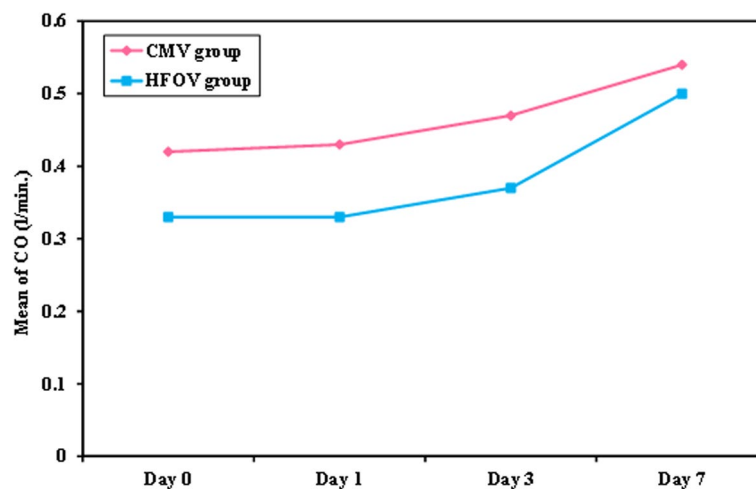


Fig. 1 Changes of cardiac output throughout the study in the HFOV and CMV groups

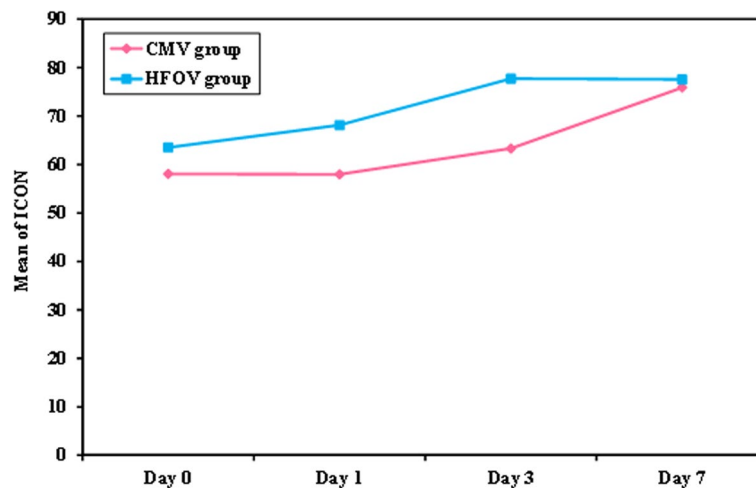


Fig. 2 Changes of ICON throughout the study in the HFOV and CMV groups

more significant use of these inotropes, reflecting more compromised hemodynamics.

In our present study, HR significantly decreased in both groups at day 3 and day 7 after ventilation and treatment, with no significant difference on comparing the two studied groups at days 0, 1, 3, and 7 of ventilation.

Aggarwal and Natarajan [15] compared ventricular function and hemodynamics of ventilated neonates with PPHN with normal controls and they found that HR was significantly higher in the PPHN group compared to the control group. In addition, a study by McNamara et al. [16] found that milrinone improved oxygenation with subsequent improvement in HR. However, it was unclear whether the HR improvement was directly due to milrinone's effect on the pulmonary vascular bed or due to

improvements in myocardial performance, which may explain the similar HR improvements observed in our groups after the first day of treatment.

Regarding BP in our study, SBP and DBP were significantly lower in the HFOV group in comparison to the CMV group at day 0, 1, 3, and 7 after ventilation. Moreover, SBP and DBP significantly increased at day 3 and day 7 after ventilation in both groups.

The improvement of SBP and DBP can be explained by reducing pulmonary afterload, improving RV compliance, and increasing myocardial contractility, hence increasing cardiac output and blood pressure stability [16].

Tachycardia and decreased blood pressure presented in our neonates can be explained by the findings of Qasim

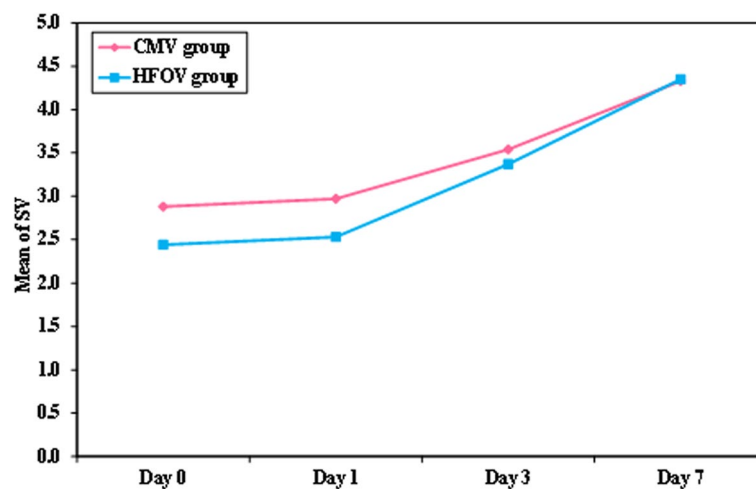


Fig. 3 Changes of SV throughout the study in the HFOV and CMV groups

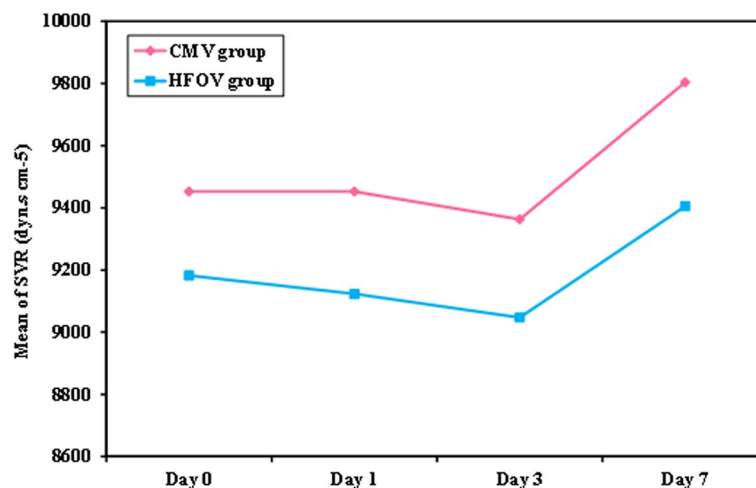


Fig. 4 Changes of SVR throughout the study in the HFOV and CMV groups

et al. [17] who found that patients with PPHN typically presented with symptoms of shock, called “obstructive shock” with tachycardia, prolonged capillary refill time (> 3 s), oliguria, systemic hypotension, and lactic acidosis.

In the current study, blood gases significantly improved at day 3 and day 7 after ventilation and treatment in both groups. This comes in agreement with other studies [18, 19]. This improvement was accompanied by a significant decrease in SPAP and improved oxygenation in both groups by sildenafil treatment and MV support.

We found that cardiac output, ICON, and SV improved significantly in both groups, but less improvement was observed in the HFOV group, which necessitates keeping an eye on the hemodynamics in neonates with PPHN who were shifted to HFOV.

The pressure loaded RV in PPHN may lead to paradoxical septal bowing and deviation, which limits LV filling and alters its shape to be banana-like, hence negatively impacting its contractility and cardiac output [20]. Thereby, decreasing RV pressure and overload by drug and ventilation improves LV stroke volume and output. Moreover, the observed lower CO/SV in HFOV could reflect higher intrathoracic pressures impairing venous return in these neonates [21].

As regards TFC and SVR, they were comparable between the two studied groups at day 0, 1, 3, and 7. TFC is an indicator of total fluid volume and represents an indirect measure of lung congestion and/or hypervolemia [22, 23]. Hence, we did not expect any change of TFC in neonates with PPHN.

Table 3 Cardiometry findings and pulmonary pressure of the studied groups

Parameter	Day	CMV group (n = 29)	HFOV group (n = 29)	P
CO (l/min.)	0	0.42 ± 0.07	0.33 ± 0.04	< 0.001
	1	0.43 ± 0.07	0.33 ± 0.04	< 0.001
	3	0.47 ± 0.07 ^{ab}	0.37 ± 0.04 ^{ab}	< 0.001
	7	0.54 ± 0.04 ^{ab}	0.50 ± 0.05 ^{ab}	< 0.001
ICON	0	63.52 ± 6.94	58.07 ± 7.34	0.032
	1	68.14 ± 7.18	57.97 ± 9.42	0.040
	3	77.7 ± 2.2 ^{ab}	65.3 ± 6.2 ^{ab}	0.010
	7	77.6 ± 2.2 ^{ab}	75.90 ± 3 ^{ab}	0.020
SVR (dyn.s cm-5)	0	9452.7 ± 9452.7	9182.5 ± 656.8	0.063
	1	9452.6 ± 387.6	9123.5 ± 800.1	0.053
	3	9363.3 ± 584.7	9047.4 ± 657.1	0.058
	7	9804.1 ± 690.5	9405.5 ± 859.7	0.057
TFC	0	23.76 ± 1.53	23.76 ± 1.53	1.000
	1	23.79 ± 1.52	23.86 ± 1.51	0.863
	3	26.17 ± 0.71 ^{ab}	26.17 ± 0.71 ^{ab}	1.000
	7	29.93 ± 1.71 ^{ab}	29.93 ± 1.71 ^{ab}	1.000
SV (ml)	0	2.88 ± 0.52	2.44 ± 0.52	0.002
	1	2.97 ± 0.57	2.53 ± 0.42	0.001
	3	3.54 ± 0.51 ^{ab}	2.94 ± 0.48 ^{ab}	0.001
	7	4.33 ± 0.28 ^{ab}	3.81 ± 0.22 ^{ab}	0.02
SPAP (mmHg)	0	58.4 ± 8.1	65.1 ± 9.5	0.841
	1	55.9 ± 7.5	60.1 ± 8.4	0.154
	3	44.3 ± 6.7 ^{ab}	49.8 ± 6.3 ^{ab}	0.342
	7	21.4 ± 3.5 ^{ab}	23.6 ± 4 ^{ab}	0.627

HFOV high-frequency oscillatory ventilator, CMV conventional mechanical ventilator, CO cardiac output, ICON index of contractility, SVR systemic vascular resistance, TFC thoracic fluid content, SV stroke volume, SPAP systolic pulmonary artery pressure

^a Significant difference with the corresponding value at day 0

^b Significant difference with the corresponding value at day 1

Few neonatal studies are available showing that hemodynamic monitoring leads to improved outcomes. A recent study reported that EC is not recommended as an accurate determinant of CO or SV [24]. However, they stated that EC may be useful for monitoring longitudinal changes from baseline of hemodynamic parameters; otherwise echocardiography is a better tool for CO and hemodynamic measurements.

Based on our results, hemodynamic measurements using EC are recommended for all PPHN patients, as they provide valuable insights into cardiac hemodynamics follow up especially in neonates on HFOV. HFOV's hemodynamic impact is acceptable as it improves oxygenation despite lower CO. Future studies with larger sample size and longer follow-up are required to validate our results.

Limitations of the study

First, the relatively small sample size may explain the non-significant SVR/TFC differences; however, sample size estimation ensures the adequate power of the study. Second, the short duration of follow up. Third, clinical outcomes of the included neonates, e.g., duration of MV, mortality, and duration of hospitalization were not studied. Fourth, HFOV group comprised neonates who failed CMV (sicker neonates) that may confound comparisons.

Conclusions

Both HFOV and CMV improve hemodynamics in full-term neonates with PPHN. However, hemodynamic improvement was more obvious in the CMV group than in the HFOV group. Hence, HFOV should be used in PPHN only in severe cases.

Abbreviations

ANOVA	Analysis of variance
CMV	Conventional mechanical ventilation
CO	Cardiac output
DBP	Diastolic blood pressure
EC	Electrical cardiometry
FIO ₂	Fraction of inspired oxygen
HFOV	High-frequency oscillatory ventilator
HR	Heart rate
ICON	Index of contractility
MAP	Mean airway pressure
MAP	Mean arterial pressure
NICU	Neonatal intensive care unit
PAC	Pulmonary artery catheter
PEEP	Positive end-expiratory pressure
PPHN	Persistent pulmonary hypertension of neonates
PVR	Pulmonary vascular resistance
RV	Right ventricle
SBP	Systolic blood pressure
SD	Standard deviation
SVV	Stroke volume variation
SVR	Systemic vascular resistance
TFC	Thoracic fluid content

Authors' contributions

HK and DE are responsible for Conceptualization and methodology. DE, HK, and NA: Methodology, writing—reviewing and editing of the manuscript; AE, MA, DE, and HK: data curation, and methodology. The manuscript was reviewed and agreed by all authors.

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Data availability

All data of this manuscript is available from the corresponding author on request.

Declarations

Ethics approval and consent to participate

The study obtained ethical approval from our Faculty of Medicine's local Ethical Committee with ethical approval code (34297/11/20). The patients were enrolled following the principles of the Declaration of Helsinki. Participants and their parents provided a written consent form.

Competing interests

The authors declare no competing interests.

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