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# **Electrical Cardiometry Changes in Neonates with Patent Ductus Arteriosus Before and After Ventilation**

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#### Abstract

**Background:** Mechanical ventilation is a life-support procedure that is invasive and has a significant impact on the cardiopulmonary system. Electrical cardiometry (EC) is a method that can be used to assess hemodynamic parameters. This work aimed to evaluate the EC changes in neonates with PDA before and after ventilation.

*Methods:* This prospective cohort research included 25 premature infants whose gestational age was less than 37 weeks with PDA on conventional mechanical ventilation.

**Results:** Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), cardiac output (CO), stroke volume (SV), and systemic vascular resistance (SVV) were insignificantly different on day 1 compared to day 0. HR, CO, SV, and SVV were significantly lower on day 3 and 7 in contrast to day 0 (P<0.05). SBP, DBP, and SVR were significantly higher on day 3 and 7 when contrasted with day 0 (P<0.05). FTC was substantially lower on day 1, day 3, and 7 in comparison to day 0.

**Conclusions:** EC is a viable approach of determining the hemodynamic effects of negative pressure ventilation. Ventilation decreases HR, PaCO2, CO, SV, and SVV and increases SBP, DBP, pH, HCO3, and SVR.

Key words: Electrical Cardiometry, Neonates, Patent Ductus Arteriosus, Ventilation.

#### Introduction

Patent ductus arteriosus (PDA) is a congenital heart condition where the ductus arteriosus, a blood vessel that is supposed to close after birth, remains open (patent). After birth, when the lungs take over oxygenation, the ductus arteriosus normally closes within the first few hours or days of life. In PDA, the vessel fails to close, resulting in an abnormal passage of blood between the pulmonary artery and aorta [1].

Mechanical ventilation is a life-support procedure that is invasive and has a significant impact on the cardiopulmonary system. It is widely recognized that the impact of positive pressure ventilation on hemodynamic parameters is intricate [2].

Electrical cardiometry (EC) is a method that can be used to assess hemodynamic parameters by detecting changes in the electrical conductivity of the thoracic cavity as blood is pumped through the heart. It is particularly useful in neonates due to its non-invasive nature and ability to provide continuous monitoring [3]. The following are the most frequently evaluated EC parameters: cardiac output (CO), stroke volume (SV), heart rate (HR), and systemic vascular resistance (SVR) [4].

The EC is a device that provides a real-time cardiovascular assessment in an absolute number and is based on impedance. During the cardiac cycle, the degree of red blood cell (RBC) alignment in the aorta is the primary determinant of fluctuations in thoracic electrical impedance. This principle is associated with these fluctuations. When aortic flow ceases during diastole, red blood cells (RBCs) are erratically oriented and inhibit electrical conduction. In contrast, the red blood cells (RBCs) align in parallel with the aortic flow as the left ventricle (LV) contracts during systole. As a result, the impedance of the electrical current in the aorta is diminished. This results in a decrease in impedance and an increase in conductivity. The pulsatile change of impedance in relation to the cardiac cycle is the basis for the calculation of hemodynamic measures [5, 6].

Nevertheless, the EC measurement of CO is contingent upon the aortic flow in order to calculate CO. Consequently, any circulatory channel between thoracic vessels that affects the aortic flow may subsequently impact the EC measurement [7].

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This work aimed to evaluate EC changes in neonates with PDA before and after ventilation..

# Patient and method:

This prospective cohort study was conducted on 25 preterm neonates who were born 37 weeks before gestation diagnosed with PDA on conventional mechanical ventilation. The study was conducted at the Neonatal Intensive Care Unit (NICU), Tanta University Hospitals from December 2020 to December 2022 after approval from the Ethical Committee of Tanta University Hospitals, Egypt. Informed written consent was obtained from the patient or relatives of the patient.

The criteria for exclusion were major congenital anomalies, dysrhythmias, chest deformities, maternal diabetes, congenital heart disease other than a patent foreman ovale or a small atrial septal defect, hydrops fetalis, and severe intraventricular hemorrhage.

PDA was classified as hemodynamically significant (hs-PDA) if the LA/Ao ratio was greater than 1.6, the pulmonary artery exhibited diastolic turbulence (backflow) on Doppler, the internal diameter of the duct was greater than 1.5 mm, and/or the descending aorta/mesenteric artery exhibited reverse end diastolic flow [8]. Full history taking, clinical examination [score system: Apgar score at 1 and 5 minutes [9], modified new Ballard score [10] and down score [11]], general examination [anthropometric measurement: (weight, length and head circumference)], vital signs [-heart rate, respiratory rate, blood pressure (using non-invasive technique with appropriate neonatal cuff) and temperature] and laboratory methods [complete blood count, c-reactive protein (CRP) quantitative assay, renal function tests, liver function, arterial blood gases: by gas state analyzer] were recorded.

# **Electrical Cardiometry [12]:**

The ICON ® hemodynamic monitor (ICON Cardiotronics, Inc., La Jolla, CA 92307; Osyka Medical GmbH, Berlin, and Germany, model C3, Serial no: 1817406) was used to take measurements. At the level of the xiphoid process, the neonate's forehead, left lower neck, left mid-axillary line, and lateral aspect of the left quadriceps were all covered with four standard surface electrocardiogram electrodes. Hemodynamic status was assessed using electrical cardiometry before ventilation with the diagnosis of hs-PDA and at 1st, 3rd, and 7th days of starting ventilation. The EC calculation was based on the weight at the moment of the experiment. The sensor cable was connected to the EC, and the case data (blood pressure - HR – SpO2 – Hb) were fed. Continuously, the EC displayed SVR, ICON, CO, and SV.

Hemodynamic parameters: [Blood flow: SV/SI: Stoke volume/Stroke index, HR, and CO/(cardiac index)/CI]. Vascular System: [SVR/SVRI: calculated from the input of central venous pressure (CVP) and mean arterial pressure (MAP)]. Contractility: ICON: Index of contractility, VIC: Variation of index of contractility, and CPI: Cardiac performance index. Fluid Status: [TFC: Thoracic fluid content, SVV: stroke volume variation, and FTC: Corrected flow time].

Each neonate under investigation underwent EC measurements. To prevent agitation, the measurements were conducted in the supine position during sleep, whenever possible. During the measurement, phototherapy was temporarily suspended. Blood pressure was measured non-invasively with a neonatal cuff immediately prior to EC measurement.

#### Statistical analysis

Statistical analysis was performed using SPSS v26 (IBM Inc., Chicago, IL, USA). The Shapiro-Wilks test and histograms were employed to evaluate the normality of the data distribution. A paired T-test was employed to present and compare the mean and standard deviation (SD) of quantitative parametric data. The qualitative variables, which were presented as frequency and percentage (%), were compared using the Chi-square test. A two-tail P value that was less than or equal to 0.05 was considered statistically significant.

#### Results:

The mean  $\pm$  SD of gestational age was  $31.9 \pm 1.24$  weeks. There were 12 (48%) males and 13 (52%) females. The mean  $\pm$  SD of weight was  $1.7 \pm 0.4$  kg. The mean  $\pm$  SD of length was  $45.3 \pm 0.57$  cm. The mean  $\pm$  SD of HC was  $32.8 \pm 0.56$  cm. The median (IQR) of APGAR score 1 min was 4 (4 - 5), and 5 min was 7 (7 - 8). The median (IQR)

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of the new Ballard score was 32 (31 - 33). The median (IQR) of the Down score was 8 (7 - 8). The mode of delivery was NVD in 6 (24%) patients and CS in 19 (76%) patients. Cardiomegaly was present in 6 (24%) patients. Lung congestion was present in 4 (16%) patients. RDs grade1 was present in 2 (8%) patients. RD grade 2 was present in 4 (16%) patients. RDs grade3 was present in 8 (32%) patients. RDs grade4 was present in 1 (4%) patient. Table 1

**Table 1: Patient characteristics of the studied patients** 

Table 1. I attent characteristics of the studied patients						
		Patients $(n = 25)$				
Gestational ag	ge (weeks)	$31.9 \pm 1.24$				
Sex	Male	12 (48%)				
	Female	13 (52%)				
	Weight (kg)	$1.7 \pm 0.4$				
	Length (cm)	$45.3 \pm 0.57$				
	HC (cm)	$32.8 \pm 0.56$				
	APGAR score 1 min	4 (4 - 5)				
	APGAR score 5 min	7 (7 - 8)				
Ne	w Ballard score (weeks)	32 (31 - 33)				
	Down score	8 (7 - 8)				
Mode of	NVD	6 (24%)				
delivery	CS	19 (76%)				
X ray	Cardiomegaly	6 (24%)				
finding	Lung congestion	4 (16%)				
	RDs grade1	2 (8%)				
	RDs grade2	4 (16%)				
	RDs grade3	8 (32%)				
	RDs grade4	1 (4%)				

Data are presented as mean  $\pm$  SD or frequency (%). BMI: Body mass index, HC: Head circumference, APGAR: Appearance, Pulse, Grimace, Activity, and Respiration, NVD: Normal vaginal delivery, CS: Cesarean section. The mean  $\pm$  SD of hemoglobin was  $15.7 \pm 1.07$  g/dl. The mean  $\pm$  SD of platelets was  $233.2 \pm 59.31 \times 109$ /L. The mean  $\pm$  SD of TLC was  $10607.8 \pm 788.34$  cells per microliter of blood. The mean  $\pm$  SD of serum creatinine was  $0.6 \pm 0.08$  mg/dl. The mean  $\pm$  SD of Serum urea was  $30 \pm 7.22$  mg/dl. The mean  $\pm$  SD of ALT was  $27.2 \pm 3.56$  IU. The mean  $\pm$  SD of AST was  $27.6 \pm 2.55$  IU.

Table 2: Laboratory investigations of the studied patients

	Patients $(n = 25)$
Hemoglobin (g/dl)	$15.7 \pm 1.07$
Platelets (× 10 <sup>9</sup> /L)	$233.2 \pm 59.31$
TLC (cells per microliter of blood)	$10607.8 \pm 788.34$
CRP (Negative)	25 (100%)
Serum creatinine (mg/dl)	$0.6 \pm 0.08$
Serum urea (mg/dl)	$30 \pm 7.22$
ALT (IU)	$27.2 \pm 3.56$
AST (IU)	$27.6 \pm 2.55$

Data are presented as mean  $\pm$  SD or frequency (%). TLC: Total Leukocyte count, CRP: C-reactive protein, ALT: Alanine transaminase, AST: Aspartate aminotransferase.

HR, SBP, and DBP were insignificantly different on day 1 in comparison to day 0. HR was notably decrease on day 3 and 7 in comparison to day 0. SBP and DBP were significantly higher on day 3 and 7 in contrast to day 0 (P<0.05).

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Table 3: Vital signs of the studied patients

	Day 0	Day 1	Day 3	Day 7
Heart rate (beats/min)	$142 \pm 8.05$	$141.8 \pm 8.17$	$135.8 \pm 9.97$	$124.3 \pm 7.15$
P value		0.327	<0.001*	<0.001*
Systolic blood pressure (mmHg)	$57.3 \pm 6.7$	$60.4 \pm 17.68$	$60.6 \pm 4.88$	$68.4 \pm 2.77$
P value		0.430	0.016*	<0.001*
Diastolic blood pressure (mmHg)	$23.8 \pm 3.72$	$29.8 \pm 23.15$	$31.9 \pm 2.71$	$42.4 \pm 2.55$
P value		0.217	<0.001*	<0.001*

PaCO2 was insignificantly different on day 3 and 7 in contrast to day 0 and was a substantial decrease on day 1 contrasted with day 0 (P<0.001). pH and HCO3 were significantly higher on day 1, day 3, and 7 in contrast to day 0 (P<0.001).

Table 4: ABG of the studied patients

	Day 0	Day 1	Day 3	Day 7
рН	$7.27 \pm 0.03$	$7.29 \pm 0.02$	$7.4 \pm 0.03$	$7.4 \pm 0.02$
P value		<0.001*	<0.001*	<0.001*
PaCO2 (mmHg)	$34 \pm 4.12$	$32.5 \pm 4.43$	$34.5 \pm 3.22$	$35.5 \pm 1$
P value		<0.001*	0.612	0.104
HCO3 (mEq/L)	$14.2 \pm 1.56$	$15.6 \pm 1.61$	$18.4 \pm 0.96$	$21.3 \pm 1.17$
P value		<0.001*	<0.001*	<0.001*

PaCO2: Partial pressure of carbon dioxide, HCO3: Bicarbonate.

CO, ICON, SV, and SVV were insignificantly different on day 1 in contrast to day 0 and were a substantial decrease on day 3 and 7 compares to day 0 (P<0.001). SVR was insignificantly different on day 1 compared to day 0 and was notably higher on day 3 and 7 compared to day 0 (P<0.001). FTC was significantly lower on day 1, 3, and 7 in contrast to day 0 (P<0.001). CPI, CI, and SI were insignificantly different on day 1, 3, and 7 in contrast to day 0.

Table 5: EC parameters of the studied patients

	Day 0	Day 1	Day 3	Day 7
CO (L/min)	$0.6 \pm 0.1$	$0.6 \pm 0.1$	$0.5 \pm 0.04$	$0.4 \pm 0.03$
P value		0.161	<0.001*	<0.001*
ICON	$132.8 \pm 26.44$	$132.7 \pm 26.46$	$113\pm10.16$	$85.4 \pm 2.47$
P value		0.327	<0.001*	<0.001*
TFC (kOhm-1)	$29.8 \pm 0.74$	$29.8 \pm 0.73$	$29.8 \pm 0.72$	$28.4 \pm 1.25$
P value		0.327	0.164	<0.001*
SV (ml)	$5.6 \pm 1.68$	$5.5 \pm 1.9$	$4.3 \pm 0.53$	$2.6 \pm 0.27$
P value		0.312	<0.001*	<0.001*
SVR (dyn.s cm-5)	$5415.1 \pm 763.11$	$5448 \pm 779.7$	$6254.2 \pm 491.85$	$8374.9 \pm 3352.24$
P value		0.116	<0.001*	<0.001*
SVV (%)	$14.7 \pm 4.6$	$14.6 \pm 4.38$	$11.5 \pm 4.42$	$9.1 \pm 2.1$
P value		0.967	0.019*	<0.001*

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FTc (milliseconds)	$249.4 \pm 18.61$	$229.5 \pm 22.28$	$222.6 \pm 21.58$	$216.6 \pm 23.8$
P value		<0.001*	<0.001*	<0.001*
CPI	$0.39 \pm 0.03$	$0.38 \pm 0.03$	$0.38 \pm 0.03$	$0.38 \pm 0.03$
P value		0.327	0.327	0.185
CI	$3.68 \pm 0.17$	$3.73 \pm 0.18$	$3.73 \pm 0.17$	$3.74 \pm 0.17$
P value		0.319	0.299	0.286
SI	$17.3 \pm 6.46$	$20.3 \pm 6.57$	$18.8 \pm 4.45$	$17.5 \pm 3.35$
P value		0.145	0.384	0.927

CO: Cardiac output, TFC: Triangular fibrocartilage disc, SV: Stroke volume, SVR: Peripheral vascular resistance, SVV: Stroke volume variation, FTC: Corrected flow time, CPI: Cardiac power index, CI: Cardiac index..

## **Discussion**

In our study, HR was notably decreased on day 3 and 7 in contrast to day 0. SBP and DBP were significantly higher on day 3 and 7 in comparison to day 0.

In neonates with PDA, the HR typically increases before ventilation due to the strain caused by increased pulmonary blood flow and oxygen demand. After the initiation of ventilation, improvements in oxygenation and reduced respiratory effort can help stabilize or lower HR, which can result in more stable blood pressure (BP) and reduced hemodynamic instability [13].

In agreement with our findings, Ayoub et al. [14] showed that in preterm neonates with respiratory distress syndrome, blood pressure increased following ventilation, but there was no adverse impact on cardiac or systemic hemodynamics. Nevertheless, they discovered that there was no difference that is statistically significant in heart rate during the follow-up measurements following ventilation.

In contrast, Rachel et al. [15] found that HR was significantly increased after extubation compared to before extubation. Moreover, Simma et al. [16] found that arterial BP remained unchanged after the use of HFOV.

In our findings, PaCO2 was substantially lower on day 1 in contrast to day 0. pH and HCO3 were a substantial elevate on day 1, 3, and 7 in comparison to day 0.

Consistent with our findings, Slee-Wijffels et al. [17] found that the PaCO2 rapidly decreased after the transition to ventilation, that's evidenced by its use as rescue therapy in patients with refractory hypercapnia. Similarly, El-Atawi et al. [18], Erdeve et al. [19], and Ayoub et al. [14] stated that ABG improved significantly after the shift to a mechanical ventilator.

Our results revealed that CO, ICON, SV, and SVV were substantially lower on day 3 and 7 in contrast to day 0. SVR was notably higher on day 3 and 7 in comparison to day 0. FTC was significantly lower on day 1, 3, and 7 in contrast to day 0.

Significant ductal shunting and PDA severity have been positively correlated with increased CO. The fundamental reason is that a PDA with a substantial left-to-right flow may induce a compensatory increase in CO to preserve systemic blood flow. In fact, CO will return to its normal state after the ductus is closed [20].

In neonates with PDA, SV is often reduced before ventilation due to increased preload and volume overload on the heart. Ventilation, especially with positive pressure, typically improves SV by increasing venous return and oxygenation while reducing the work of breathing. SVV is usually high before ventilation due to respiratory fluctuations and hemodynamic instability, but after ventilation, SVV generally decreases as oxygenation improves and the neonate's cardiovascular System stabilizes [21].

These results were supported by Rachel et al. [15] who found that 70 % of neonates had a statistically substantial decline in CO after extubation. Also, they found that SV was significantly decreased after extubation compared to before extubation. Matched to our results, Rodríguez Sánchez de la Blanca et al. [22] reported a statistically a considerable decline in SV 72h after the initiation of treatment of hs-PDA. Moreover, Lien et al. [23] reported that The SVR was substantially enhanced following the ligation and remained elevated following the surgery. Infants with enlarged PDAs exhibited a greater degree of SVR elevation. Due to ductal shunting, the vascular resistance may have been initially lower, and its subsequent increase may suggest a return to a normal level.

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Limitations of the study included that the sample size was relatively small. Different types of mechanical ventilation apparatus and exclusion of preterm neonates who failed to close PDA by the 7th day.

## Conclusion

EC is an effective approach to assessing the hemodynamic consequences of negative pressure ventilation. Ventilation decreases HR, PaCO2, CO, ICON, SV, and SVV and increases SBP, DBP, pH, HCO3, and SVR.

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