Noninvasive Cardiac Output Monitoring in Newborn with Hypoplastic Left Heart Syndrome

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Abstract

Objective This study aimed to describe the first two cases of electrical cardiometry applied to newborn with hypoplastic left heart syndrome for hemodynamical assessment in the first days of life before surgical correction and see if this can help decision making process in these patients.

Study Design We describe two case series of two full-term newborn with hypoplastic left heart syndrome in the Neonatal Intensive Care Unit, ASST Grande Ospedale Metropolitano Niguarda, between December 2019 and January 2020.

Results Case 1 was persistently hemodynamically stable with prostaglandin E1 infusion at 0.01 mcg/kg/min, showing good capillary refill time, good diuresis, no difference between pre- and postductal values of oxygen saturation or blood pressure. Electrical cardiometry monitoring constantly showed cardiac output values higher than 300 mL/kg/min. Case 2 showed poor clinical condition needing prostaglandin E1 infusion up to 0.05 mcg/kg/min, intubation and septostomy associated with low cardiac output around 190 mL/kg/min. Once cardiac output has begun to rise and reached values constantly over 300 mL/kg/min, clinical condition improved with amelioration in oxygen saturation, diuresis, blood pressure, and blood gas analysis values. She was then extubated and finally clinically stable until surgery with minimal infusion of prostaglandin E1 at 0.01 mcg/kg/min.

Conclusion This case highlights how hemodynamic information provided by electrical cardiometry can be used to supplement the combined data from all monitors and the clinical situation to guide therapy in these newborns waiting surgery.

Keywords
- electrical cardiometry
- hypoplastic left heart syndrome
- newborn
- hemodynamic
- thoracic electrical bioimpedance

Key Points

- This is the first report of electrical cardiometry (EC) use in newborn with hypoplastic left heart syndrome (HLHS).
- In HLHS patients, it is impossible to measure cardiac output without being invasive.
- EC helps in guiding therapy in HLHS patients in a noninvasive way.

Electrical cardiometry (EC) is a noninvasive method that measures thoracic electrical bioimpedance (TEB) and derives stroke volume and cardiac output (CO). Although infants and children had been studied with impedance cardiography in the past, its use in patients with congenital heart disease has rarely been reported. Only recently, it has been demonstrated a good correlation in measuring CO between EC and invasive techniques in a pediatric population with structural heart disease.[1]

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These are, to our knowledge, the first two cases that described EC applied to newborn with congenital heart disease for hemodynamical assessment in the first day of life before surgical correction.

CO (mL/kg/min) was measured by TEB-EC (ICON Osypka Medical GmbH, Berlin, Germany). The device was connected and patient demographic and anthropometric data (age, weight, and height) were entered. Four skin electrodes (iSense Electrical Cardiometry Skin Sensors; Osypka Medical) were applied per manufacturer recommendations. EC assumes that erythrocytes are in random orientation before the aortic valve opening and then align in a parallel fashion with pulsatile blood flow.[2] This parallel state results in increased conductance and conversely decreased impedance. The difference in conductance, between these two states, provides the baseline data that allow the calculation of CO using mathematical algorithms.

Case Report 1

A male neonate was born full term with a prenatal diagnosis of hypoplastic left heart syndrome (HLHS) with Apgar scores of 8/8 and birth weight of 3,070 g. Postnatal echocardiogram confirmed the diagnosis, showing mitral atresia and severe hypoplastic aorta in its valvular and ascending parts. Atrial level communication was thought to be adequate and as such no intervention was needed at that time. To maintain the ductus arteriosus patency, an infusion of prostaglandin E1 (PGE1) was started at 0.03 µg/kg/min which was later tapered down to 0.01 µg/kg/min for apneas appearance. The EC monitoring constantly showed CO values higher than 300 mL/kg/min (►Fig. 1A) and the patient was persistently hemodynamically stable showing good capillary refill time, good diuresis, no difference between pre- and postductal values of oxygen saturation or blood pressure. Hemogas analysis showed only a maximum value of lactate of 2.7 mmol/L in the first hour of life. He did not need oxygen supplementation despite a minimal ventilatory support with high-flow nasal cannula (5 L/min). Norwood operation was performed after 9 days.

Case Report 2

A full-term female, birth weight of 3,720 g, with antenatal diagnosis of HLHS was hypoxic at birth (oxygen saturations 55–60%) with no improvement on supplemental oxygen. Postnatal echocardiogram confirmed the diagnosis with atrial level communication of 4 mm. Initial clinical stabilization was obtained starting nasal continuous positive airway pressure for respiratory distress and PGE1 infusion at 0.03 µg/kg/min with pre- and postductal values of oxygen saturation around 80%. Hemogas analysis showed pH 7.27, pCO_2 54 mm Hg, PaO_2 36 mm Hg, HCO_3^- 24.8 mmol/L, BE −3.1, and lactate 1.9 mmol/L. After few hours, PGE1 infusion was increased to 0.05 µg/kg/min due to clinical deterioration: polypnea, diminished spontaneous activity, oxygen saturation up to 65%, and mixed acidosis (pH 7.21, pCO_2 54 mm Hg, PaO_2 29 mm Hg, HCO_3^- 21.3 mmol/L, BE −7.4, and lactate 4.2 mmol/L). The patient was therefore intubated and balloon atrial septostomy was performed, but it was technically influenced by laxity of interatrial septum so that there was not significant improvement in the dimension of the foramen ovale (4 mm). Back from the hemodynamic theater, EC was available and it was applied to the patient showing low CO around 190 mL/kg/min (►Fig. 1B). Oxygen saturation was around 70% with mechanical ventilation settings: SIPPV + Vg (26 mL), RR 45/min, TI 0.4 seconds, FiO_2 21%.

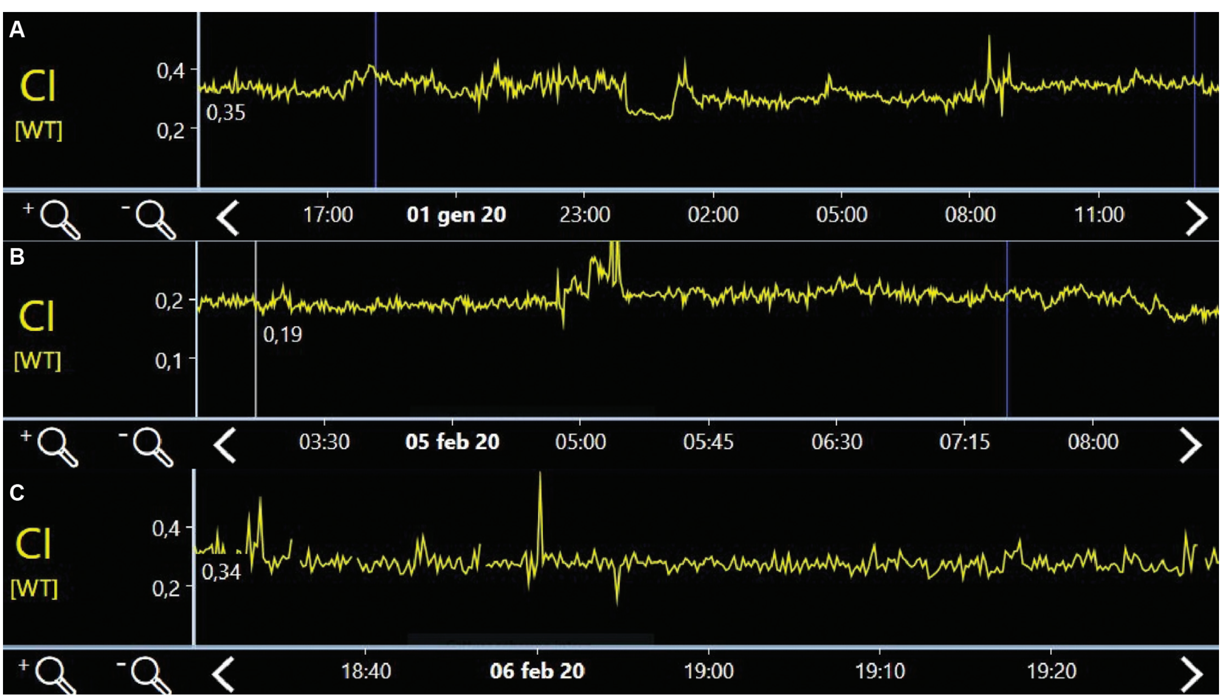

Fig. 1 Cardiac output monitoring expressed as cardiac index (CI, indexed for weight) of patient 1 (A) and patient 2 immediately after balloon atrial septostomy (B) and after stabilization (C).

Blood gas analysis showed pH 7.44, pCO_2 25 mm Hg, PaO_2 88 mm Hg, HCO_3^- 17.2 mmol/L, BE −4.5, and lactate 4.1 mmol/L. She was anuric with blood pressure 43/20 (32) mm Hg. A 10 mL/kg bolus of saline solution was administered followed by adequate fluid maintenance, PGE1 were continued at 0.02 µg/kg/min, and mechanical ventilation parameters were modified.

After few hours, EC showed gradual increase in CO (around 340 mL/kg/min) with amelioration in oxygen saturation (90–92%), diuresis (4,7 mL/kg/min), blood pressure, and blood gas analysis values (pH 7.4, pCO_2 30 mm Hg, PaO_2 76 mm Hg, HCO_3^- 18 mmol/L, BE −4.7, and lactate 2.4 mmol/L). After 24 hours, she was then extubated, maintaining CO parameters constantly more than 300 mL/kg/min (►Fig. 1C), good diuresis, and oxygen saturation persistently more than 90%. Norwood operation was performed after 7 days of life.

CO is a fundamental physiological measure used for diagnosis and guiding therapy in many clinical conditions especially in newborn.[3] Transthoracic echocardiogram (TTE) with transaortic Doppler is traditionally used in daily clinical practice, but it needs aortic blood flow to measure velocity and diameters to provide an estimation of CO.[4] In some structural heart diseases such as HLHS in which there is no outflow from the left ventricle, measuring CO with TTE is not possible. With the exception for invasive approach,[5] there is no single technique available for measuring CO continuously and accurately in these patients. Even if research is limited in newborn with structural congenital heart disease, it has been recently demonstrated a good correlation in measuring CO between EC and invasive techniques in children with structural heart disease undergoing cardiac catheterization.[1]

We applied EC to newborn with HLHS to monitor CO and correlate it with clinical conditions. No reference values are available in this kind of population since EC has been evaluated previously in hemodynamically stable term and preterm infants (including low birth weight and very low birth weight).[6–9] Comparing our two cases, we can speculate that CO around 300 mL/kg/min can be suitable to have hemodynamic balance in patients with HLHS. In case 1, in fact, CO was persistently higher than 300 mL/kg/min, while case 2 showed a low CO (around 190 mL/kg/min) even postballoon atrial septostomy which was not resolved. After a few hours, probably with the decrease in pulmonary vascular resistance and adequate fluid replacement, there was an increase in right ventricular output with increased pulmonary blood flow and consequent improvement in oxygen saturation, amelioration of blood flow through the foramen ovale and through the ductus arteriosus. Postductal measurement of CO provided by EC gradually improved probably reflecting these physiological mechanisms, arranging around 300 mL/kg/min which, we suppose, can be considered appropriate for these kinds of patients as demonstrated by the improvement of her clinical condition.

In conclusion, TEB-EC can play an important role to give continuously hemodynamic function assessment when CO cannot be evaluated with TTE because of cardiac structural abnormalities. Hemodynamic information provided by EC can be used to supplement the combined data from all monitors and the clinical situation to guide therapy in these newborns waiting Norwood surgery.

However, more studies are needed to define normal value of CO in this population to better evaluate hemodynamic status during the first day of life.

Authors' Contributions

All the authors had a determining role in the clinical management of the patients. I.F.G. and O.V. made substantial contributions to the conception, the design of the manuscript, and drafted the manuscript. S.M. advised on the case management, made substantial contributions to the design of the work, and substantively revised the manuscript. All authors read and approved the final manuscript.

Conflict of Interest

None declared.

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