Extracorporeal Membrane Oxygenation (ECMO) or Extracorporeal Life Support (ECLS)

Michael A*
Seattle Children's Research Institute, Seattle, Washington, USA

Extracorporeal Membrane Oxygenation

Extracorporeal membrane oxygenation (ECMO), otherwise called extracorporeal life support (ECLS), is an extracorporeal strategy of giving delayed cardiovascular and respiratory help to people whose heart and lungs can't give a sufficient measure of gas exchange or perfusion to support life. The innovation for ECMO is generally gotten from cardiopulmonary bypass, which furnishes more limited term support with captured native circulation. The gadget utilized is a membrane oxygenator, called as artificial lung.

ECMO works by briefly drawing blood from the body to permit counterfeit oxygenation of the red blood cells and expulsion of carbon dioxide. It is utilized either post-cardiopulmonary detour or in late-stage treatment of an individual with significant heart/lung failure, in spite of the fact that it is currently considering use to be a treatment for heart failure in specific places, permitting treatment of the hidden reason for capture while flow and oxygenation are supported. ECMO is used to help patients with the intense viral pneumonia related with COVID-19 in situations where artificial ventilation alone is not sufficient to sustain blood oxygenation levels.

Extracorporeal membrane oxygenation (ECMO) offers a route to recovery after myocardial stunning and damage sustained during cardiovascular surgery in infants and children. Approximately 2% to 5% of children undergoing surgery for congenital heart defects with cardiopulmonary bypass require subsequent support by ECMO. Additional indications for cardiac support by ECMO include ventricular arrest and myocarditis. The majority of these patients undergoing ECMO for cardiac indications are infants and young children older than neonates and the Extracorporeal Life Support Organization (ELSO) database shows that these numbers are rising. Veno-arterial ECMO for cardiac support unloads the ventricles, supports the systemic blood pressure, and maintains blood oxygenation. Adversely, ventricular volume unloading by the ECMO circuit promotes an inflammatory response, which disrupts hormonal homeostasis and induces metabolic stress. Despite these stresses, the immature heart maintains metabolic flexibility and protein synthesis under ECMO conditions. However, weaning from ECMO reintroduces cardiac work requirements. Though many patients tolerate initial weaning, mortality in the subsequent 30 days remains high in part due to inability to adequately sustain cardiac output. The myocardial energy requirements and metabolic shifts associated with weaning are unknown but may play an important role in determining cardiac functional recovery. Furthermore, appropriate nutritional support may be critical in maintaining cardiac function after weaning particularly in the immature heart, which must also sustain normal growth. Carbohydrates and fatty acids represent the primary energy substrates used by the heart. However, to a lesser extent amino acids, which can be shuttled into protein synthesis, are also used as sources for ATP production in the citric acid cycle. In fact, amino acids contribute carbons to intermediates both through oxidative pathways and anaplerosis. Thus, amino acids provide integration between oxidative metabolism and protein synthesis. The branched chain amino acid, leucine, in particular regulates protein turnover in the heart, and also behaves as a ketogenic species broken down into acetylcoenzyme A and acetacetyl-CoA. They tested the hypothesis that ventricular reloading after ECMO increases energy requirements and modulates both substrate entry into CAC and myocardial protein synthesis. Elucidation of these modifications could identify potential therapeutic targets for sustaining adequate cardiac function after weaning.

In the study entitled Myocardial reloading after extracorporeal membrane oxygenation alters substrate metabolism while promoting protein synthesis conducted by Masaki Kajimoto, they used an established immature piglet model, which emulates ECMO and weaning in infants and children supported for cardiac indications. They also used 13-Carbon (13C)-labeled substrates as well as nuclear magnetic resonance (NMR) and gas chromatography/mass spectroscopy (GC-MS) methods to track metabolism and protein synthesis. Coronary venous flow and myocardial oxygen consumption (MV02) were measured via a shunt created between the coronary sinus and the superior vena cava. A miniaturized extracorporeal circuit consisting of a roller peristaltic pump console (Sarn8000 Terumo) and a hollow fiber membrane oxygenator (CX-RX05RW, Terumo) was utilised. The circuit was primed with about 80 mL containing dextran 40 in 0.9% sodium chloride, 5% dextrose and 2000 units of heparin. The ascending aorta and right atrium were cannulated to create a veno-arterial ECMO circuit. Management during ECMO kept the pump flow rates of 80 to 100 mL/kg per minute. They maintained a pH of 7.35 to 7.45, an arterial Pco2 of 35 to 45 mm Hg, an arterial PO2 of >120 mm Hg, and a rectal temperature of 36 to 37.5°C. ECMO duration time was 8 hours. Perfusion flow of ECMO was decreased gradually for 30 minutes and then ECMO was weaned. After 1.5 hours of ECMO wean, all parameters were measured.

ECMO unloads the heart, providing a bridge to recovery in children after myocardial stunning. ECMO also induces stress which can adversely affect the ability to reload or wean the heart from the circuit. Metabolic impairments induced by altered loading and/or stress conditions may impact weaning. However, cardiac substrate and amino acid requirements upon weaning are unknown. They assessed the hypothesis that ventricular reloading with ECMO modulates both substrate entry into the citric acid cycle (CAC) and myocardial protein synthesis.

*Address for Correspondence: Michael A, Seattle Children’s Research Institute, Seattle, Washington, USA

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