

# HEMODYNAMIC PERFORMANCE OF VENO-PULMONARY ECMO IN A MOCK CIRCULATORY LOOP.

Prashant Chand (1), Avishka Wickramarachchi (1) Andrew Stephens (1), Hakeem Yusuff (2,3), Vasileios Zochios (4,5), Chris Joyce (6), Kiran Shekar (7), Shaun Gregory (1,8)

1. Cardio-Respiratory Engineering and Technology Laboratory, Department of Mechanical and Aerospace Engineering, Monash University, Australia; 2. Department of Respiratory Sciences, University of Leicester, UK; 3. NIHR Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, UK; 4. Department of Cardiovascular Sciences, University of Leicester, Leicester, UK; 5. University hospitals of Leicester NHS Trust, Glenfield Hospital ECMO Unit, Leicester, UK; 6. Princess Alexandra Hospital, Australia; 7. Prince Charles Hospital, Australia; 8. Centre for Biomedical Technologies and School of Mechanical, Medical, and Process Engineering, Queensland University of Technology, Australia

## Introduction

Veno-Pulmonary extracorporeal membrane oxygenation (VP ECMO) is a treatment strategy that provides right ventricular (RV) and respiratory support to patients. VP ECMO provides pulmonary circulation support by directing flow into the pulmonary artery (PA) with negligible recirculation [1]. The hemodynamic impact of direct non-pulsatile flow in the pulmonary vasculature of an acute respiratory distress syndrome patient with RV failure is not well represented in the current literature. The aim of this in-vitro study is to explore the hemodynamic effects of different flow rates in a mock circulatory loop (MCL). This MCL simulates patients with varying levels of abnormal RV function at different pulmonary hypertension (PH) states.

## Methods

A VP ECMO circuit was connected to a MCL via the right atrial (RA) and pulmonary artery (PA) chambers using a 23 Fr drainage and 17 Fr pulmonary arterial cannula, respectively. The MCL was used to simulate patients with various levels of RV function (normal to severe dysfunction) and pulmonary hypertension by adjusting the pulmonary vascular resistance (PVR) from 100-600 dynes/sec/cm<sup>-5</sup>. The hemodynamic response in each of these clinical scenarios at different levels of ECMO flow rates was recorded (1-5 L/min).

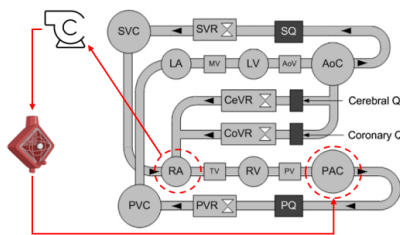


Figure 1: VP ECMO and the MCL experimental setup.

## Results

VP ECMO was able to successfully perfuse the pulmonary circulation and maintain adequate cardiac output. Mean pulmonary artery pressures (mPAP) were observed to increase - with a steeper gradient in patients with higher PVR. At flow rates of 3-4 L/min, mPAP values increased beyond 35 mmHg in the simulated patient with mild RV injury and high PVR (> 300).

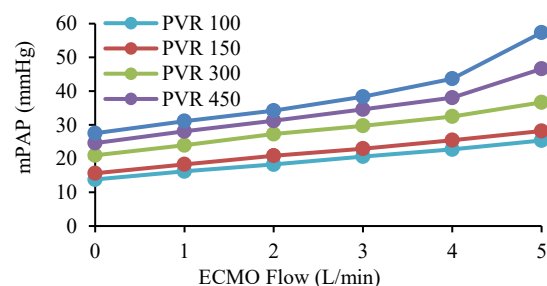


Figure 2: Graph of mean pulmonary artery pressures against ECMO flow rate for a patient with abnormal RV function and various degree of pulmonary hypertension.

Left atrial pressures also increased with increasing ECMO flowrates. However, the RA pressures decreased. Additionally, the pulmonary arterial pulsatility index (PAPi) decreased with increasing ECMO flow rates. There was complete loss of pulsatility at lower flow rates for patients with high PVR, suggesting a correlation with PH. The prolonged loss of PAPi in certain groups of patients has been shown to have a negative impact on ECMO weaning outcomes [2].

## Discussion

Although VP ECMO potentially offers significant cardiac and respiratory support to patients without recirculation challenges, careful consideration is required when selecting patients for this treatment modality. At high ECMO flows, mPAP values increase significantly, and this could potentially result in irreversible damage to the pulmonary vasculature, causing pulmonary hemorrhage. Unlike veno-venous ECMO, VP support cannot be run at very high flows (> 4 L/min), and this may affect its ability to maintain blood gas exchange. Therefore, this study shows that PVR as well as RV function, in addition to respiratory support, need to be considered when selecting VP flow rates to prevent damage to the pulmonary vascular bed and ensure successful liberation from ECMO.

## References

1. Zochios. V et al, Asaio, 69:511-518, 2023.
2. Martin-Suarez. S et al, J Clinical Medicine, 11:4353, 2022.