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## HEMODYNAMIC ADAPTATION MECHANISMS OF HEART FAILURE TO PERCUTANEOUS VENOARTERIAL EXTRACORPOREAL CIRCULATORY SUPPORT

PAVEL HÁLA



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**Hemodynamic Adaptation Mechanisms of Heart Failure  
to Percutaneous Venoarterial Extracorporeal  
Circulatory Support**

**Pavel Hála**

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Reviewers:

doc. MUDr. Štěpán Havránek, Ph.D.

doc. MUDr. Ondřej Szárszoi, Ph.D.

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# Abbreviations

**ANP, BNP** – atrial and brain natriuretic peptides  
**CO** – cardiac output  
**dP/dt<sub>max</sub>** – maximal positive pressure change  
**dP/dV** – diastolic stiffness  
**E<sub>a</sub>** – effective arterial elastance  
**EBF** – extracorporeal blood flow  
**ECLS** – extracorporeal life support  
**ECMO** – extracorporeal membrane oxygenation  
**EDA, ESA** – end-diastolic and end-systolic area  
**EDD** – end-diastolic diameter  
**EDP, ESP** – end-diastolic and end-systolic pressure  
**EDV, ESV** – end-diastolic and end-systolic volume  
**E<sub>es</sub>** – slope of ESPVR  
**EF** – ejection fraction  
**ELSO** – Extracorporeal Life Support Organization  
**FAC** – fractional area change  
**HF** – heart failure  
**HR** – heart rate  
**LV** – left ventricle  
**LVAD** – LV assist device  
**MVO<sub>2</sub>** – myocardial oxygen consumption  
**PE** – myocardial potential energy  
**PI** – pulsatility index  
**PV (loop)** – pressure-volume (loop)  
**PVR** – pressure-volume relationship  
**rSO<sub>2</sub>** – regional tissue oxygenation  
**RV** – right ventricle  
**SV** – stroke volume  
**SvO<sub>2</sub>** – mixed venous blood saturation  
**SW** – stroke work  
**TAPSE** – tricuspid annular plane systolic excursion  
**VPO** – ventricular power output





# Abstract

**Introduction:**

Venoarterial extracorporeal membrane oxygenation (VA ECMO) is widely used in the treatment of circulatory failure, but repeatedly, its negative effects on the left ventricle (LV) have been observed. The purpose of this study is to assess the influence of extracorporeal blood flow (EBF) on systemic hemodynamic changes and LV performance parameters during VA ECMO therapy of decompensated heart failure.

**Methods:**

Porcine models of low-output chronic and acute heart failure were developed by long-term fast cardiac pacing and coronary hypoxemia, respectively. Profound signs of circulatory decompensation were defined by reduced cardiac output and tissue hypoperfusion. Subsequently, under total anesthesia and artificial ventilation, VA ECMO was introduced. LV performance and organ specific parameters were recorded at different levels of EBF using an LV pressure-volume loop analysis, arterial flow probes on carotid and subclavian arteries, and transcutaneous probes positioned to measure cerebral and forelimb regional tissue oxygen saturations.

**Results:**

Conditions of severely decompensated heart failure led to systemic hypotension, low tissue and mixed venous oxygen saturations, and increase in LV end-diastolic pressure. By increasing the EBF from minimal flow to 5 L/min, we observed a gradual increase of LV peak pressure, reduced arterial flow pulsatility, and an improvement in organ perfusion. On the other hand, cardiac performance parameters revealed higher demands put on LV function: LV end-systolic volume and end-diastolic pressure and volume all significantly increased (all  $P < 0.001$ ). Consequently, the LV stroke work increased ( $P < 0.05$ ) but LV ejection fraction did not. Also, the isovolumetric contractility index did not change significantly.

**Conclusions:**

In decompensated chronic and acute heart failure, excessive VA ECMO flow increases demands on left ventricular workload and can be potentially harmful. To protect the myocardium, VA ECMO flow should be adjusted with respect to not only systemic perfusion, but also to LV parameters.

**Key words:**

Extracorporeal membrane oxygenation; Heart failure; Hemodynamics; Heart ventricles; Artificial cardiac pacing