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

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Acknowledgments

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While learning about the principles of circulatory supports, facilities of the Experimental laboratory at Albertov became an amazing place where our initial ideas were transformed into real practice. In team cooperation with fellow laboratorians, scientists, and doctors, we were able to design and set novel experiments to help me understand and explain pathophysiological mechanisms of circulatory supports. It was a school of great value and pleasant nature at the same time.

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I endlessly value the facts that on this long and winding road many of my close friends helped and many of my colleagues became my friends. Without their little help, the presented work would never get by. A shared passion became our motivation for every day in the life of physiology, medicine, and research.

Abbreviations

ANP, BNP – atrial and brain natriuretic peptides
CO – cardiac output
dP/dt_{max} – maximal positive pressure change
dP/dV – diastolic stiffness
Ea – 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
Ees – 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_2 – myocardial oxygen consumption
PE – myocardial potential energy
PI – pulsatility index
PV (loop) – pressure-volume (loop)
PVR – pressure-volume relationship
rSO_2 – regional tissue oxygenation
RV – right ventricle
SV – stroke volume
SvO_2 – 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