Journal of Shock and Hemodynamics

Volume 1 | Issue 1 Article 4

2022

Hemodynamics of Prolonged Percutaneous Mechanical Circulatory Support – When Vasodilatation Sets

Holger Thiele
Heart Center Leipzig, University of Leipzig, Holger.Thiele@medizin.uni-leipzig.de

Symposium Presentation *Journal of Shock and Hemodynamics*

September 16, 2022 https://doi.org/10.57905/josh/e2022114

2022 Symposium Presentation

Hemodynamics of Prolonged Percutaneous Mechanical Circulatory Support – When Vasodilatation Sets

Holger Thiele, MD
Heart Center Leipzig, University of Leipzig, Germany

Email: Holger.Thiele@medizin.uni-leipzig.de

Received August 31, 2022
Published September 16, 2022

Abstract

Hemodynamics play an important role in cardiogenic shock assessment for prognosis estimation and for phenotyping cardiogenic shock. This is best done by pulmonary artery catheters. In general, at the beginning of cardiogenic shock, patients have vasoconstriction, which over time may lead to vasodilation. This is often triggered by percutaneous mechanical circulatory support. This review will elucidate the hemodynamics and the factors that possibly lead to vasodilation in patients with mechanical circulatory support.

Keywords: percutaneous mechanical circulatory support, cardiogenic shock, vasodilation

Background

At present, there is a dearth of published literature concerning the hemodynamics of prolonged percutaneous mechanical circulatory support (pMCS). However, the pathophysiology of vasodilation in patients with acute myocardial infarctions has been described in particular after longer persistence of cardiogenic shock.[1,2] A similar pathophysiology is seen in patients with congestive heart failure-related cardiogenic shock. Initially, reduced cardiac output and stroke volume are noted, and the body is trying to counteract the reduced blood pressure by vasoconstriction, at least in the beginning. Over time, inflammation increases and leads to a vasodilation state because of a pro-inflammatory response which might even be triggered by pMCS.

Unfortunately, we do not know when vasodilation truly occurs. We hypothesize it occurs earlier in patients that have undergone resuscitation, received MCS, or have active bleeding. We also know that patients with cardiogenic shock can develop concomitant septic shock and vice versa. Thus, a combination of cardiogenic and septic shock can affect treatment and outcomes.

Current Knowledge

Multiple options are currently available for MCS. Each option differs regarding flow, pump speed, cannulation, placement options, and ability to unload the right or left ventricle.[3] The Society for Cardiovascular Angiography and Interventions (SCAI) recently updated and published its definition of cardiogenic shock.[4] The SCAI SHOCK II definition advises the team to use MCS to reverse severe cardiogenic shock when a patient reaches stages D or E. The goal of MCS at those stages is to stabilize the patients and revert them to the A or B classification.

In addition, data from trials and registries have identified the cardiac power index as one of the strongest hemodynamic parameters to predict outcomes.[5] It is calculated by multiplying the cardiac index by the mean blood pressure and a factor (i.e., 0.0022). Some randomized trials have shown that MCS can improve the cardiac index and, thereby, the cardiac power index.[6] However, in an individual patient data meta-analysis, this could not be shown for MCS versus control.[6]

Importantly, reperfusion can stop ischemia. In general, it is thought that MCS along with inotropes and vasopressors

can increase inflammation, which leads to vasodilation.[1,2] The timing and variance of inflammation may differ based on the type of device used. The answer remains unknown, and comparative data are needed.

In the intensive care unit, patients on MCS develop inflammation and vasodilation over time. This common observation is supported by a recent review article from Krychtiuk and colleagues.[7] Once a patient enters the severe cardiogenic shock stages of D and E, often the systemic vascular resistance goes down. At the same time, the inflammatory activation increases, and systemic inflammatory response syndrome is observed. Of note, this summary of cardiogenic shock progression remains a theory and needs more data over time.

Jentzer and colleagues studied concomitant sepsis in patients with cardiogenic shock.[8] Since 2000, the incidence of concomitant cardiogenic shock and sepsis has increased. The incidence is higher in patients with non-ST-elevation myocardial infarction (NSTEMI) than in those with STEMI. Without question, sepsis leads to vasodilation; thus, this parameter must be considered when treating cardiogenic shock.

The search for fast, objective, biomarker-based scores for cardiogenic shock prognosis continues. The CLIP-Score was recently developed from trial data and combines measures of renal function, tissue hypoxemia, inflammation, and heart failure (cystatin C, lactate, interleukin-6, and N-terminal [NT]-pro hormone Brain natriuretic peptide, respectively).[9] This underlines the importance of inflammation in the cardiogenic shock progress, as shown in this objective prognosis score.

Among treatments with extracorporeal membrane oxygenation (ECMO), the combination with Impella devices (ECMELLA) for venting recently emerged. In theory, the concomitant use results in better pressure-volume curves and improved outcomes compared to ECMO alone, as shown in propensity-matched studies.[10] However, patients with ECMELLA had more complications, including severe and moderate bleeding and hemolysis, access-site-related ischemia, and abdominal compartment syndrome.[10]

In terms of medical therapy, vasodilation is generally treated with vasopressors in the ICU. Norepinephrine is likely the strongest and best vasopressor we currently have; thus, it is recommended for use in patients with Impella or ECMO support who remain in hypotensive cardiogenic shock.[1]

Currently, there is a lack of randomized evidence in cardiogenic shock. Only a few adequately powered trials have provided relevant data. There is strong evidence for early revascularization for an acute myocardial infarction, as it can reduce mortality.[3] Multivessel coronary artery disease is present in roughly 80% of the patients with cardiogenic shock, and the CULPRIT-SHOCK trial determined that culprit lesion-only PCI is better than immediate-multivessel PCI.[11] Multiple trials have confirmed norepinephrine is better than any other vasopressor.[3] However, more research is needed; thus, multiple large-scale randomized trials are underway.[3]

Disclosures

Dr. Thiele has received research funding for his institution from the German Research Foundation, German Heart Research Foundation, German Cardiac Society, European Union, Else-Kroner-Fresenius Foundation, Schwiete Foundation, and Boston Scientific.

References

[1] van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: A scientific statement from the American Heart Association. *Circulation*. 2017;136(16):e232-e268. doi: 10.1161/CIR.0000000000000525.

[2] Thiele H, Allam B, Chatellier G, Schuler G, Lafont A. Shock in acute myocardial infarction: the Cape Horn for trials? *Eur Heart J*. 2010;31(15):1828-35. doi: 10.1093/eurheartj/ehq220.

[3] Thiele H, Ohman EM, de Waha-Thiele S, Zeymer U, Desch S. Management of cardiogenic shock complicating myocardial infarction: an update 2019. *Eur Heart J*. 2019;40(32):2671-2683. doi: 10.1093/eurheartj/ehz363.

[4] Naidu SS, Baran DA, Jentzer JC, et al. SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies. *J Am Coll Cardiol*. 2022;79(9):933-946. doi: 10.1016/j.jacc.2022.01.018.

[5] Fincke R, Hochman JS, Lowe AM, et al. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. *J Am Coll Cardiol*. 2004;44(2):340-8. doi: 10.1016/j.jacc.2004.03.060.

[6] Thiele H, Jobs A, Ouweneel DM, et al. Percutaneous short-term active mechanical support devices in cardiogenic shock: a systematic review and collaborative meta-analysis of randomized trials. *Eur Heart J*. 2017 De;38(47):3523-3531. doi: 10.1093/eurheartj/ehx363.

[7] Krychtiuk KA, Vrints C, Wojta J, Huber K, Speidl WS. Basic mechanisms in cardiogenic shock: part 1-definition and pathophysiology. *Eur Heart J Acute Cardiovasc Care*. 2022;11(4):356-365. doi: 10.1093/ehjacc/zuac021.

[8] Jentzer JC, Bhat AG, Patlolla SH, et al. Concomitant Sepsis Diagnoses in Acute Myocardial Infarction-Cardiogenic Shock: 15-Year National Temporal Trends, Management, and Outcomes. *Crit Care Explor*. 2022;4(2):e0637. doi: 10.1097/CCE.0000000000000637.

[9] Ceglarek U, Schellong P, Rosolowski M, et al. The novel cystatin C, lactate, interleukin-6, and N-terminal pro-B-type natriuretic peptide (CLIP)-based mortality risk score in cardiogenic shock after acute myocardial infarction. *Eur Heart J*. 2021;42(24):2344-2352. doi: 10.1093/eurheartj/ehab110.

[10] Schrage B, Becher PM, Bernhardt A, et al. Left Ventricular Unloading Is Associated With Lower Mortality in Patients With Cardiogenic Shock Treated With Venoarterial Extracorporeal Membrane Oxygenation: Results From an International, Multicenter Cohort Study. *Circulation*. 2020;142(22):2095-2106. doi: 10.1161/CIRCULATIONAHA.120.048792.

[11] Thiele H, Akin I, Sandri M, et al. PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock. *N Engl J Med*. 2017;377(25):2419-2432. doi: 10.1056/NEJMoa1710261.