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Coronary Flow and Unloading in Acute Myocardial Infarction
Shock
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Abstract

In patients with cardiogenic shock that undergo successful coronary intervention, there are still factors complicating myocardial recovery. There is room for improvement in coronary flow using mechanical circulatory devices, specifically by left ventricular unloading. This idea was further explored in a research study using pigs. Results showed that subjects with acute myocardial infarction who have reduced cardiac contractility and/or high diastolic pressure would benefit from support strategies targeting left ventricular unloading.

Keywords: left ventricular unloading, myocardial infarction, coronary flow, Impella

Background

Previous studies showed that multivessel percutaneous coronary intervention (PCI) might not be an ideal treatment for acute myocardial infarction (AMI). For patients with ST-segment Elevation Myocardial Infarction (STEMI) and multivessel disease, non-culprit PCI with complete revascularization was superior to culprit lesion-only PCI. In contrast, for patients with AMI and cardiogenic shock, culprit lesion PCI was a better treatment option than multivessel PCI. Therefore, alternative strategies are needed to improve the outcomes of patients with AMI and cardiogenic shock.

In patients with cardiogenic shock, multiple factors impair coronary flow even after a successful PCI: 1) decreased cardiac output reduces coronary perfusion pressure, 2) increased diastolic pressure causes vasculature compression, and 3) lung congestion leads to hypoxia. All contribute to myocardial ischemia and can progressively worsen cardiogenic shock, creating a positive feedback loop. To break this loop, there is room for improvement in mechanical circulatory device use.

Left Ventricular Unloading with Impella in Pigs

An experimental pig study investigated the effects of left ventricular (LV) support on coronary flow (unpublished data). In one case, the pig underwent 90 minutes of the left anterior descending artery (LAD) balloon occlusion followed by reperfusion for 2 hours. A coronary flow/pressure wire was then placed into the LAD, together with LV pressure monitoring using a pressure catheter. There was a significant decrease in LV diastolic pressure with Impella (Abiomed) support. Meanwhile, it increased coronary pressure and led to an increase in driving pressure, which can be calculated by subtracting LV pressure from coronary pressure. Coronary flow was also increased by Impella support. The results from this case suggested that mechanical LV support increases coronary pressure and improves flow.

These results are unlikely to be limited to the Impella since all support devices can improve systemic flow by replacing cardiac output. However, one device that complicates this assumption is the intra-aortic balloon pump (IABP), which is known to increase coronary diastolic...
pressure and flow but does not improve clinical outcomes.\textsuperscript{3,4} The reason for the discrepancy might be that newer devices may provide more adequate support for LV diastolic pressure than the IABP.

Following an AMI, LV diastolic pressure increases because of insufficient pumping action, compressing the ischemic wall that prevents coronary flow during diastole. By unloading the LV via mechanical support, the goal is to improve flow by reducing the compression on the wall. To demonstrate this concept, porcine models underwent the previously described procedure and were monitored for pressure and volume during LV unloading.\textsuperscript{5} Impella support reduced end-diastolic volume and pressure, resulting in a significant reduction in wall stress during diastole. In this study, the flow was measured with a microsphere technique instead of a coronary wire; millions of spheres were mixed with blood and injected into the left atrium. These spheres are large enough to get trapped in the capillaries of the myocardium, allowing quantitation of the tissue flow by counting the spheres in retrieved tissues.\textsuperscript{5}

LV unloading resulted in an increase in infarct tissue perfusion, but not in the border or remote areas. This is likely due to the autoregulation that takes place in normal tissue to maintain a normal range of flow. This mechanism is disrupted in infarct tissues, allowing the flow to become reliant on external factors. Based on the infarct perfusion results and diastolic wall stress, the relationship indicated a reliance on this LV wall stress.

The study was expanded to identify factors that could predict infarct flow improvement with LV unloading.\textsuperscript{6} Univariate analysis in data from 15 pigs identified cardiac output, mean pulmonary arterial wedge pressure, mean left arterial pressure, minimum LV pressure, end-diastolic LV pressure, end-diastolic pressure-volume relationship (EDPVR), and maximum ventricular contractility (dP/dt) to be the significant factors predicting flow improvement. Multivariate analysis showed that reduced dP/dt maximum and higher EDPVR were associated with improved infarct flow.\textsuperscript{6} These results suggest that patients with reduced cardiac contractility, high diastolic pressure, or AMI shock benefit the most from mechanical LV unloading strategies.

Another study showed improved outcomes in patients treated with a combination of extracorporeal membrane oxygenation (ECMO) and Impella compared to those individuals treated with ECMO alone.\textsuperscript{7} This data further supports the importance of LV unloading as a key factor in recovery from cardiogenic shock.

\textbf{Conclusion}

In conclusion, LV unloading in AMI shock improves myocardial perfusion by increasing coronary arterial pressure and decreasing LV diastolic wall stress. However, there is currently not enough data to prove the clinical benefit, so further research should focus on flow during support in AMI shock.

\textbf{References}


