# LV Unloading using an Impella CP Reduces Wall Stress and Improves Coronary Flow and Perfusion in Infarcted Myocardium

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### Background

LV unloading may improve coronary perfusion by increasing cardiac output and reducing LV wall stress. Whether sustained LV unloading improves myocardial perfusion in a post-MI setting remains uncertain.

## Hypothesis

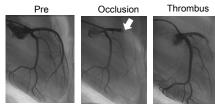
Unloading a post-MI LV with an Impella CP increases coronary flow and myocardial perfusion by reducing LV wall stress and increasing cardiac output.

# Methods

To mimic patients who need LV support, large anterior transmural MI was induced by occluding the proximal LAD for 90 minutes in **Yorkshire pigs (n=5, 40-50 Kg)** followed by a thrombus injection through the balloon lumen to induce total occlusion of the LAD. Two-weeks after the MI, animals underwent LV unloading with an Impella CP for 120 minutes. Epicardial coronary flow was assessed by coronary flow wire before, 5 minutes and 120 minutes after LV unloading. Myocardial perfusion was assessed using fluorescent microspheres before and 120 minutes after LV unloading.

### Model

Proximal left anterior descending artery was percutaneously occluded for 90 min followed by thrombus injection through the balloon lumen.



### Protocol



CF: Coronary flow measurement using Flowire (Volcano)

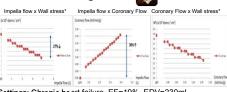
MP: Myocardial perfusion assessed by fluorescent microspheres

Echo: Echocardiographic volume assessment

# Results

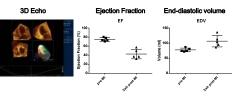
Coronary angiograms revealed TIMI 2 flow in 4 animals, and TIMI 3 flow in one animal. LV unloading with maximal pump support (P8) for two hours resulted in an increase in total cardiac output (3.08 to 3.93 l/min, P=0.07). Impella support significantly reduced end-diastolic volume (109±17 to 85±12 mL, P=0.02) and end-diastolic pressure (29.3±5.6 to 19.2±6.9 mmHg) resulting in a significant decrease in LV end-diastolic wall stress (infarct: 34.4±5.5 to 20.2±5.2 kdvnes/cm2. P=0.03; remote: 32.4±8.6 to 19.3±6.0 kdynes/cm2, P=0.03). Coronary flow increased acutely (LAD: 6.7±1.8 to 10.2±1.5 cm/s, P=0.03, LCx: 8.4±2.7 to 10.4±3.6 cm/s, P=0.31) and remained elevated at 120 minutes (LAD: 9.8±1.3 cm/s, P=0.058, LCx: 12.4±4.2 cm/s, P=0.058). Compared to baseline, myocardial perfusion as measured by fluorescent microspheres within the infarct zone was significantly increased (87±67%, P=0.02), while perfusion of the remote nonischemic myocardium was similar compared to the baseline (-2±15%, P=0.89), likely due to autoregulation in the non-injured myocardium.

#### Simulation using Harvi



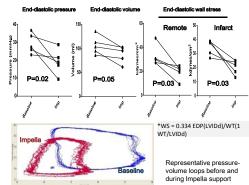
Settings: Chronic heart failure, EF=19%, EDV=230ml, EDP=30mmHg, CO=4l/min, LM stenosis=0%, LV mass=205g

#### Impaired cardiac function 2 weeks after MI induction



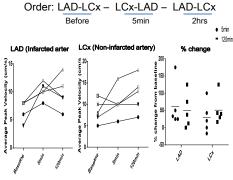
#### LV unloading using Impella CP reduces LV wall stress

Two hours after Impella support (P8), both enddiastolic pressure (EDP) and end-diastolic volume (EDV) decreased in all pigs, resulting in significant reduction of end-diastolic wall stress.

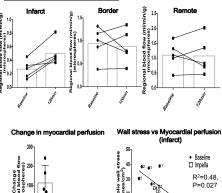


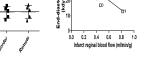
#### Increased coronary flow assessed by flowire

Coronary flow was measured using flowire before Impella, 5 min, & 2 hrs after Impella initiation.



#### Increased myocardial perfusion in the infarct & its relation to enddiastolic wall stress





### Summary

- Impella support increased coronary flow in both infarcted and non-infarcted artery.
- Myocardial perfusion was improved in only the infarcted area.
- There was a linear relationship between myocardial perfusion in the infarct and the end-diastolic wall stress.

# Conclusion

Sustained LV unloading using an Impella CP increases coronary flow and perfusion of the infarcted myocardium for at least 2 hours.