MECHANICAL FUNCTION IN THE INFARCTED HEART SUPPORTED BY A REGENERATIVE ASSIST DEVICE: A COMPUTATIONAL STUDY

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Introduction

Adverse ventricular remodelling following acute myocardial infarction (MI) may induce ventricular dilation, fibrosis and loss of global contractile function, potentially resulting in heart failure. Cardiac patches, composed of living cardiac (https://projectbrave.eu/) may be able to restore cardiac function and reduce adverse ventricular remodelling post-MI. Computational modelling can aid in device design without the complex environment and intra-patient variability that make experimental techniques difficult and time-consuming. In this study, we employ computational modeling to assess the role of patch fiber orientation on functional improvement in cardiac pump function and local tissue mechanics.

Methods

We extended the finite element model of cardiac mechanics in [1] to model a cardiac patch, implanted over a chronically remodeled infarct area. The infarct had a circular shape, lacked active contraction and had a 10-fold increase in passive stiffness. A cardiac patch was modeled as a rectangular strip of material measuring 6 by 4 cm with a thickness of 2 mm. We assumed material properties of healthy myocardium within the cardiac patch. Fibers were aligned along the long side of the patch. In simulations P0, P45 and P90, the long side was oriented at an angle of 0, 45 and 90 degrees with respect to the LV circumferential direction. Cardiac function was assessed through global hemodynamics and local myofiber mechanics, computed in 3 points throughout the geometry. Local mechanics in the cardiac patch were averaged for a grid of 25 centrally positioned sites.

Results

The infarct, 15% in size, reduced stroke work by 30% compared to the healthy heart. Cardiac patch simulations showed minor increases in stroke volume and systolic pressure. Of the total loss in stroke work, 5.2%, 7.5% and 1.6% was recovered in simulations P0, P45 and P90 respectively. In the myocardium, stress-strain loops at locations UM, CI and LL (fig 1a.) were similar in the healthy case (REF); work density, represented by the surface area enclosed, was similar as well. In chronic MI (CMI), loops in healthy tissue at locations UM and LL skewed leftward, indicating reduced work density, and disappeared in CI. Loops in UM and LL recovered best in patch simulations P45. Stress-strain loops within the patch (P) generally showed reduced strain excursion, low fiber stress, and low work density. Furthermore, the stress-strain loop in simulation P90 was shifted to the left.

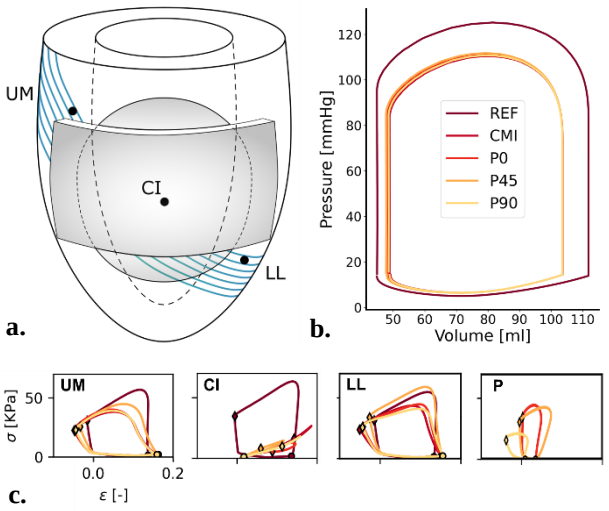

*Figure **1a**: LV geometry including cardiac patch, upper-medial (UM), central-infarct (CI) and lower-lateral (LL) sites for analysis of local fiber mechanics. (**b**) Pressure-volume loops for healthy (REF), infarcted (CMI) and supported heart (P0-90). (**c**) Cauchy stress in fiber direction vs. fiber strain over a cardiac cycle in the healthy, infarcted and supported ventricle.*

Discussion

The amount of pump function lost due to MI exceeded the loss in healthy tissue by about two-fold. This disproportional loss was attributed to unfavourable mechanical interactions between infarcted and healthy tissue, adjacent to the infarct area, resulting in a reduced ability to develop stress. The low patch-induced improvement of cardiac function is due both to the low patch volume (4ml) and to the limited strain excursion, resulting from tethering to the underlying stiff infarct. In varying the patch orientation, simulation P45 showed the largest amount of functional recovery. Work density in the patch was higher, but also work density in the native cardiac tissue improved the most. Because force generated here was best transmitted through the patch.

References

1. Janssens et al., 2023 [Manuscript submitted for publication]

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