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Understanding the effect of moderately increased heart rate on heart failure patients with preserved ejection fraction

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Heart failure (HF) occurs when the heart cannot, for a variety of reasons, pump out and/or fill up with enough blood. This condition can be quantified by ejection fraction, defined as the percentage of blood in the left ventricle that is pumped out of the heart with every heartbeat. Approximately half of the patients suffering from HF have heart failure with preserved ejection fraction (HFpEF). A preserved ejection fraction is associated with concentric cardiac remodeling of the left ventricle. This remodeling could be due to not only a change in the mechanics of the heart such as strain, but also due to hormones such as the angiotensin system which catalyzes cardiac growth.

Patients with HFpEF have not reacted positively to the current treatments for HF nor have many interventions improved quality of life. A new alternative treatment, consisting of moderately increasing heart rate, has shown promising results in restoring normal cardiac function in pigs with HFpEF. Over a period of three weeks, the treatment restored normal left ventricle cavity volume and wall thickness. The therapy consists of implanting a pacemaker and increasing the subjects' heartrates from 93 to 125 beats per minute. While the study ascertained the desired results, it is still unknown why exactly increased heartrate has this effect on patients with HFpEF.

The goal of my research is to understand why increased heart rate reverses the remodeling of HFpEF and restores normal cardiac function. I hypothesized that heart rate induced reverse growth (reduction of left ventricle size) is caused by a combination of mechanical factors (e.g., strain), and hormonal factors caused by the activation of the angiotensin system. To test this hypothesis, I utilized a recently published computational model of the heart and blood circulation. First, I replicated HFpEF-induced adverse cardiac remodeling in the model based on experimental porcine data. Once I successfully replicated the HF state, I will aim to mimic the novel therapy in future by increasing the heart rate in the model and drive reverse growth by altering mechanical and/or hormonal factors.