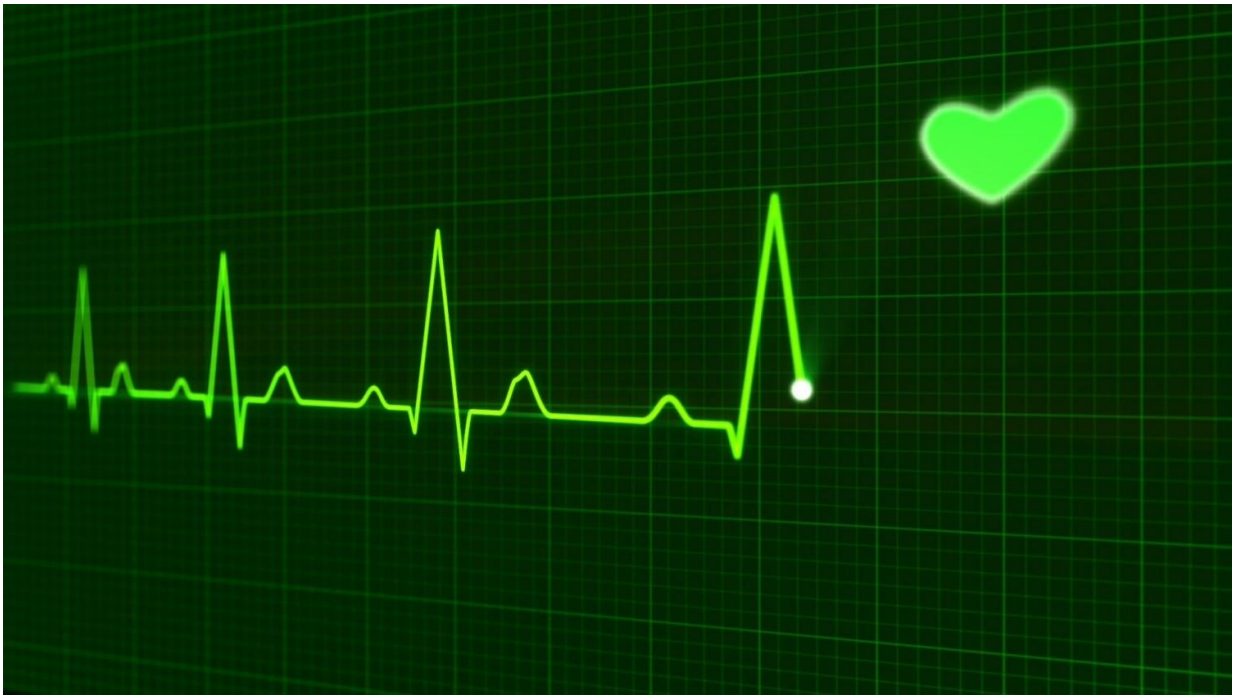
A paradigm shift in heart failure treatment?

May 28 2018



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A small, preliminary study could trigger a paradigm shift in the treatment of heart failure. The late-breaking research is published today in *Circulation* and presented at Heart Failure 2018 and the World Congress on Acute Heart Failure, a European Society of Cardiology congress. The study suggests that heart failure may be caused by inappropriate fluid shifts in some patients rather than an excess of fluid.

Heart failure is a global pandemic affecting at least 26 million people worldwide and is increasing in prevalence. Despite the significant advances in therapies and prevention, mortality and morbidity are still high and quality of life poor.

It is currently believed that heart failure is caused by excess salt and fluid in the heart and lungs. However, 50% of patients with acute heart failure do not have fluid overload when they present to hospital.

In these patients, an alternative mechanism could be that fluid has been inappropriately redistributed from the abdominal (splanchnic) compartment to the heart and lungs. It is thought that this leads to increased pressure in the heart, resulting in impaired heart function (decompensation). It has been proposed that this redistribution is partly caused by overactivity of the splanchnic nerves, which cause the vasculature in the belly to constrict.

This first proof of concept study in humans sought to prove that this fluid redistribution exists in patients with acute heart failure and could be a target for treatment. The study included five patients with acute heart failure admitted to Duke University Hospital in Durham, North Carolina, US.

Splanchnic nerve block was performed by injecting lidocaine on either side of the spine under X-ray guidance. This is an established procedure that is used to relieve abdominal pain in patients with pancreatic and duodenal cancer. Right heart catheterisation was conducted to measure pressures in the heart and pulmonary artery.

Following lidocaine injection, pressures in the right and left side of the heart and pulmonary artery reduced rapidly, suggesting that blood shifted from the heart and lungs to the belly. The reduction in pressures began 15–20 minutes after the nerve block and reached the maximum

effect at 30 minutes. Pressures mostly returned to starting levels at 90 minutes, which is the expected duration of lidocaine action. There were no adverse events.

Before and after the procedure, patients performed a six-minute walk test and were asked about shortness of breath, a typical heart failure symptom. Both measures improved, but the differences were only statistically significant for the shortness of breath questionnaires, likely due to the small number of patients.

Principal investigator Dr. Marat Fudim, of Duke Clinical Research Institute, said: "This study supports the notion that volume redistribution is a cause of heart failure. When the splanchnic nerve is blocked, the belly vasculature is no longer constricted and space is created. Fluid then shifts from the heart and lungs to the abdomen, where it should be. This normalises pressures in the heart and lungs and patients feel better."

Diuretics are a cornerstone of treatment in acute heart failure but often cause kidney damage. "This may be because these patients do not actually have extra fluid," said Dr. Fudim. "Our study suggests that shifting fluid from the chest to the belly, instead of getting rid of fluid with diuretics, may be the appropriate treatment for some patients with acute heart failure."

Dr. Fudim noted that the nerve block in this study was temporary. Future studies will need to examine the impact of longer-acting medications or even killing the splanchnic nerves. The latter has been used to alleviate pain in cancer patients. The nerve block will also need to be tested in patients with chronic heart failure.

More information: Marat Fudim et al. Splanchnic Nerve Block for Acute Heart Failure, *Circulation* (2018). DOI: 10.1161/CIRCULATIONAHA.118.035260

Provided by European Society of Cardiology