Medical Xpress

Earlier surgical intervention could stave off fibrosis in people with mitral valve prolapse

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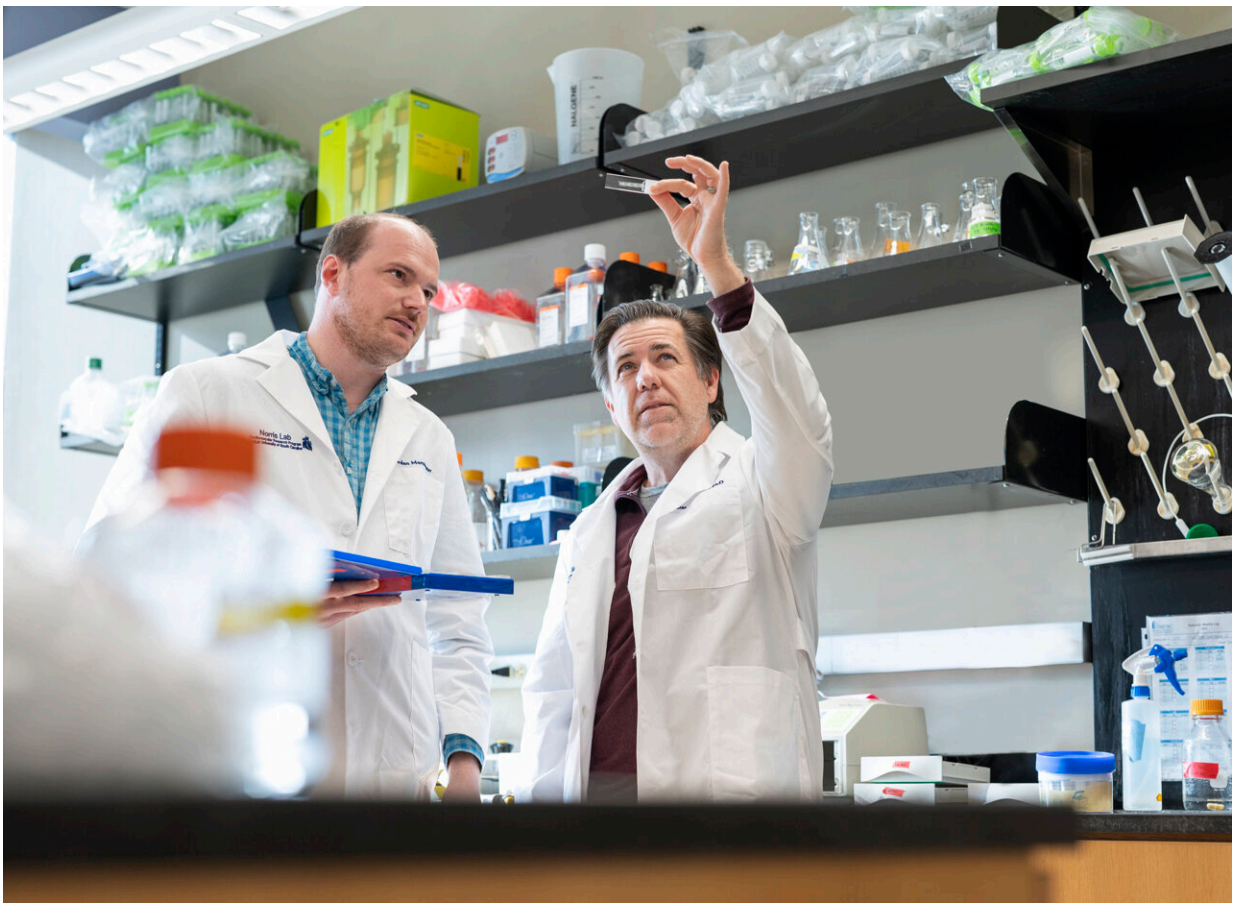

Dr. Chip Norris and medical student Jordan Morningstar in the lab studying mitral valve prolapse. Credit: Brennan Wesley

The larger the sail, the more wind it catches. But as that size increases,

so does the stress experienced by each sheet and line connected to the canvas.

The same thing can be said for a heart with mitral valve prolapse (MVP); as the diseased valve enlarges, the cords connected to the heart tissue feel stress and respond accordingly with stiffness and scarring, according to a recent paper in the *Journal of the American Heart Association*.

As a common degenerative heart disease, MVP affects 2-3% of the U.S. population, or 1 in 40 people. It's characterized by enlarged heart tissue and billowing of the mitral valve into the left atrium, according to the AHA. While it is often considered benign, physicians are careful to watch for mitral regurgitation, which is when the mitral valve doesn't close properly and allows blood to leak backwards through it. Once MVP progresses to that stage, surgery is often required. If left untreated, it can result in a heart attack, stroke or sudden cardiac death.

While only a tenth of patients with MVP are likely to need surgery, waiting until regurgitation symptoms occur might result in too much irreversible scarring, or fibrosis, in the heart, according to Chip Norris, Ph.D., principal investigator for the study and a professor in the regenerative medicine and cell biology department at MUSC. Too much fibrosis can result in stiff tissue and a heart that struggles to pump despite surgical intervention.

After examining tissue biopsies from both human and mouse MVP patients as well as applicable genetic data, Norris and his research team have now shown a relationship between regionalized fibrosis in the left ventricle and the disease. This relationship points to a potential need for guideline updates, according to Jordan Morningstar, a medical student at MUSC who was involved in Norris' study alongside fellow MUSC student Cortney Gensemer.

"It appears the fibrosis occurring in the myocardium is already established at the time of surgery," Morningstar said. "And it brings into question whether we need to be performing earlier surgical intervention in these patients in order to prevent the scarring from happening."

Norris suggests not only looking for mitral regurgitation in patients but also monitoring disease progression with imaging to track the amount of fibrosis in the heart over time. Advanced fibrosis could be added to the list of traditional markers for mitral valve repair, which include shortness of breath, mitral regurgitation and left ventricular dilation.

Norris says that this is the first study to provide direct evidence that MVP causes regionalized left ventricular fibrosis, which makes it impactful. "Understanding that the defective valve causes these changes in the left ventricle is significant," he said. "We need to start having better ways of imaging these patients and following them more closely."

He also suggests surgeons performing any kind of valve procedures should look for scarring in both the wall of the left ventricle. "These findings show us that mitral valve prolapse is a disease of both the valve as well as the ventricle," Norris said.

More research is needed to determine at what earlier point surgery would be most helpful and if it would indeed stop progressive fibrosis in that part of the heart.

Morningstar compares myocardial fibrosis to a cut in the skin. "When you injure yourself, you have a group of cells called fibroblasts which can proliferate into the cut and lay down the proteins that help resolve the wound," he said. "That wound gets smaller over time, but it eventually goes away, and a scar is left behind."

The same is true for left ventricular fibrosis—that scar persists and can

grow.

Through the study, Norris, Morningstar and Gensemer showed that mechanical stretching kicks off the fibrotic pathways in the heart. As the mitral valve gets bigger, it pulls—or yanks, as Morningstar puts it—on the tissues and cords connected to the heart. These cells respond with fibrosis and stiffening to lessen the stretching that occurs. But that stiffening only makes it harder to pump blood effectively throughout the body.

Norris plans to continue this line of research to determine if suggested changes to the surgical guidelines for MVP are needed, and he hopes these changes will provide fairer winds and easier blood circulation to patients with this degenerative heart disease.

More information: Jordan E. Morningstar et al, Mitral Valve Prolapse Induces Regionalized Myocardial Fibrosis, *Journal of the American Heart Association* (2021). DOI: 10.1161/JAHA.121.022332

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