

# Chest pain prior to a heart attack can protect the heart

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Patients who experience chest pain in the 24 hours preceding a heart attack, also called preinfarction angina, have smaller heart attacks and improved cardiac function in the contemporary cardiac stenting era, researchers found in a study published Jan. 22 in *Circulation: Cardiovascular Interventions*.

"Even before we began treating [heart attack patients](#) with angioplasty and stenting, physicians recognized that patients with chest pain prior to their heart attack seem to have better outcomes," says the study's senior author, Jay H. Traverse, MD, a research cardiologist at the Minneapolis Heart Institute Foundation in Minneapolis. "The question 'Given faster treatment times for stents, would the protective benefit be maintained?' still remained."

Prior to this study, there have only been a few, small studies to assess this effect in the cardiac stenting era. The researchers retrospectively assessed 1,031 patients admitted with a first [acute heart attack](#), or ST-elevation [myocardial infarction](#) (STEMI). They then analyzed all patients who had ongoing chest pain duration between one and six hours who received a cardiac stent to treat their heart attack, with a procedure called primary [percutaneous coronary intervention](#).

They identified 245 patients at the Minneapolis Heart Institute® at Abbott Northwestern Hospital who had occluded arteries on presentation, of which 79 patients had documented preinfarction angina, defined as episodes of similar chest pain within 24 hours of the onset of

their heart attack. "Physicians may not realize that between 30 and 40 percent of all STEMI patients experience preinfarction angina," Traverse says.

In the study, the researchers found that the occurrence of preinfarction resulted in a 50 percent reduction in heart attack size compared with patients without preinfarction angina. This translated into improved [cardiac function](#) at the time of the patient's discharge.

"Preinfarction angina appears to be a trigger that activates endogenous protective mechanisms in the heart," he concludes. "Future research should be focused upon identifying what these protective mechanisms are, as there may be a method of pharmacologically activating them."

Provided by Minneapolis Heart Institute Foundation

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