

# Atherosclerotic plaques' downstream spread linked to low shear stress

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In human coronary arteries, atherosclerotic plaques tend to spread downstream because of the changes in blood flow patterns the plaque causes, researchers have found.

This insight comes from a study of fluid dynamics in the arteries of people being treated for [coronary artery disease](#). The results are being presented Tuesday, Nov. 15 at the [American Heart Association](#) Scientific Sessions meeting in Orlando.

The study leader is Habib Samady, MD, professor of medicine and director of interventional cardiology at Emory University School of Medicine. Postdoctoral researcher Parham Eshtehardi is presenting the data.

Shear stress is a measure of how hard blood pulls on the walls of arteries, and is calculated based on intracoronary ultrasound and measurements of blood flow. Shear stress influences how sticky the cells lining the arterial walls are and how much [white blood cells](#) and cholesterol build up.

The researchers found that atherosclerotic plaques are often linked to a region of low shear stress immediately downstream, which in turn forms conditions favorable for additional [plaque buildup](#). In contrast, regions of high shear stress are most often found within plaques.

"Our findings confirm, for the first time in humans, some of the relationships between fluid dynamics and atherosclerosis that have been

predicted by laboratory studies," Eshtehardi says. "This may provide an insight to the role of shear stress in how plaques in human coronary arteries enlarge and progress."

The goal of Samady's research is to help doctors identify "[vulnerable plaque](#)," or plaque likely to spill open and form a blood clot, leading to a heart attack or stroke. Mature [atherosclerotic plaques](#) often have components such as a fibrous cap, dense calcium and a necrotic core of [dying cells](#) and fats. A thin cap and a necrotic core are features that may make the plaque more vulnerable to rupture.

A companion poster presentation (also Tuesday) has data showing that low shear stress is associated with greater necrotic core and dense calcium, while high shear stress is associated with greater plaque burden overall. Shear stress was not connected to the amount of fibrous tissue in the plaque.

"Our findings suggest that both low and high shear stress may be implicated in different stages of atherosclerosis and that for a given degree of plaque, low shear stress is associated with more [plaque](#) vulnerability, as defined by ultrasound," Eshtehardi says.

To collect the data, doctors examined 27 patients in Emory University Hospital's catheterization laboratory because they had abnormal exercise EKGs or stable chest pain. The patients' coronary arteries were examined by intracoronary ultrasound and Doppler guide wire before and after six months of therapy with atorvastatin (Lipitor).

To model shear stress, Samady and Eshtehardi teamed up with assistant professor Michael McDaniel, MD, and Jin Suo and Don Giddens, experts in fluid mechanics at Georgia Tech. The patients' arteries were divided into more than 100 segments each, and the shear stress was calculated for each one.

**More information:** More information on the abstracts here:

[bit.ly/sPBtRE](https://bit.ly/sPBtRE)

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Provided by Emory University

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