STROKE BUSTERS in TURBULENT BLOOD

THE TOOLS ARE IN PLACE TO ZERO-IN ON THE CORRELATIONS BETWEEN TURBULENT FLOW AND STROKE.

If the brain is a light bulb and blood to the brain is electricity, then a stroke is lights out, what happens when you cut off the flow. Strokes kill about 150,000 Americans each year. They’re the third-leading cause of death in this country, after heart disease and cancer, and the leading cause of adult disability, affecting two to three million American stroke survivors.
Most strokes — about 80 percent of 700,000 a year in the U.S. — happen because an artery that carries blood uphill from the heart to the head gets clogged. Most of the time, as with heart attacks, the problem is atherosclerosis, hardening of the arteries, calcified buildup of fatty deposits on the vessel wall. The primary troublemaker is the carotid artery, one on each side of the neck, the main thoroughfare for blood to the brain.

Awareness of a relation between strokes and the carotid artery is at least as old as the name — from the Greek verb karoun, to plunge into deep sleep or stupor. Only within the last 25 years, though, have researchers been able to put their finger on why the carotid is especially susceptible to atherosclerosis.

"Blood has the same level of cholesterol in our toes as in our coronary arteries," says Frank Loth, a biomechanics professor at the University of Illinois, Chicago, "so you might expect that atherosclerosis would be a diffuse disease, that we’d get it anywhere. But we don’t. There are particular sites — coronary arteries, abdominal aorta, carotid arteries and others."

Loth’s specialty is hemodynamics, fluid dynamics of the blood — a relatively recent and growing field of work that has produced some answers about atherosclerosis. For the past 15 years, Loth has teamed with University of Chicago vascular surgeon Hisham Bassiouny to study vascular hemodynamics. Among other things, they’ve worked on understanding flow in the carotid artery, both healthy and with arterial narrowing — called stenosis — due to the plaque buildup of atherosclerosis.

“We’re trying to define the hemodynamics for different degrees of carotid stenosis,” says Bassiouny, who specializes in carotid artery disease and in endarterectomy, a life-saving surgical procedure to remove plaque from the carotid artery.

“Our hypothesis is that there are specific flow patterns, turbulent and non-turbulent, that may predispose to plaque progression or plaque breakdown.”

Four years ago, Loth and Bassiouny joined forces with two computer scientists, Paul Fischer of Argonne National Laboratory and his associate Henry Tufo, experts in the numerical methods of flow modeling. The objective: Develop the ability, with computational modeling, to provide a detailed readout of the flow patterns and forces in the carotid arteries of patients, information that doctors can then use to help identify who’s at high risk for stroke.

With a mix of disciplines to fit the job — vascular surgery, fluid mechanics, advanced numerical methods — the Chicago-based team has made rapid strides. This year, with availability of LeMieux, PSC’s terascale system, they’ve done what hasn’t been done before. Starting with a CT scan from a patient’s severely clogged carotid artery, they’ve simulated the transition from smooth to turbulent flow that occurs in that artery over the course of one heartbeat. Just as importantly, they’ve demonstrated that it’s feasible to produce this kind of information quickly, within 24 hours, so it can be used in treatment planning.

SHEAR STRESS

Over the last 20 years, hemodynamics has established a relation between flow patterns and the likelihood of atherosclerosis. The vessel sites most susceptible to disease are like the outside bank of a stream where there’s a sharp turn. “You might have a region where water is slow,” says Loth, “and you’ll see leaves and branches in a recirculation area with a little sandy beach. The same thing happens in arteries.”

When there’s low flow velocity and recirculation, the vessel wall feels “low shear stress.” Like the force you exert on a desktop as you slide your hand across it, shear stress is force in the direction of flow. Low shear stress, research has shown, is one of the key factors in predicting whether someone with healthy arteries will develop atherosclerosis.

In the carotid artery, low shear stress tends to happen near a particular site — the carotid bifurcation — where the artery splits in two. In one branch, just past the fork, a healthy artery is spacious and then narrows as it turns inward toward the brain. In the spacious region, flow along the outer wall is often slow with recirculation, prime territory for trouble.

Over time, as plaque builds up, the flow patterns at this site change. In a healthy, spacious artery, the flow is smooth. In a stenosed, narrow artery, flow into the bend is faster and, with enough narrowing,
becomes turbulent. The increased force of this flow can disrupt plaque, a potentially fatal problem. “The mechanism of a stroke in half the cases,” says Bassiouny, “is plaque in the carotid artery that breaks apart. As fragments travel upstream, they can block a vital artery.”

The choice of treatment for carotid atherosclerosis — blood-thinning medication, surgery, or no treatment — depends not only on the degree of narrowing, but also on whether the plaque is likely to fragment. Knowing the flow patterns and forces, says Bassiouny, would lead to better decisions. “Not every patient who has plaque has a stroke. For someone with 60 percent stenosis, we could decide the case is non-conducive to progression and instability. Another patient with 60 percent stenosis but with different plaque configuration and flow dynamics might need an endarterectomy.”

Fischer is a pioneer in an advanced numerical approach called “the spectral element method,” having worked on his dissertation at MIT with its originator, Tony Patera. The advantage is high accuracy with efficient use of computing resources. In 1999, a major computing award, the Gordon Bell Prize, recognized Fischer and Tufo for the quality algorithm and fast performance of Nek5000, their spectral element program.

Loth and Fischer spent the first two years of their collaboration adapting Nek5000 to simulate vascular hemodynamics. Tufo is a specialist in “scaling,” software techniques to maximize the teamwork among hundreds or thousands of processors in massively parallel systems, and he took charge of getting Nek5000 up and running efficiently on LeMieux. With his fine tuning and using 2,048 processors in test runs, Nek5000 steps through its paces at 1.25 trillion calculations per second.

Bassiouny provided CT scans from a 55-year-old man with a 70 percent stenosed carotid artery. With the scan data as input, Seung Lee, a University of Illinois, Chicago graduate student who works with Loth, used a series of programs to construct a mesh-like computational grid. Doppler ultrasound measurements from the patient provided the initial flow velocity.

Using 256 processors for this first real-case simulation, the researchers were able to simulate a full cardiac cycle — one heartbeat — in 11 hours of wall-clock time. An animation depicts the results at a cross-sectional slice through the artery, as if looking down at a river in which the flow alternately rushes forward and then slows as the heart relaxes. Just around the bend from where the carotid turns inward toward the brain, as the flow feels the force of the heart’s contraction, a slow, lazy river transforms to a torrent with violent swirls of turbulence.

“This is somewhat surprising,” says Fischer, “since people generally expect turbulence to result from separation past the stenosis, rather than due to transition inside the stenosis. The strong three-dimensionality of the structures shows the importance of full three-dimensional modeling of these flows.”

It’s the first time this transition has been captured by simulation of an actual patient’s carotid artery. Fischer is pleased not only with the flow results but also with the computing turnaround. “I didn’t think we’d be in this 24-hour range on our first shot. We’re ahead of the curve for two reasons: the numerical methods we use and having access to a machine like Pittsburgh’s.”

Part of the objective for this first round of simulation was to measure how rapidly flow velocity fluctuates with time, from which the researchers can judge how thinly to slice the calculations in the next round of

TRANSITION TO TURBULENCE IN A STENOSED CAROTID ARTERY

These snapshots from the simulation show increasing turbulence (left to right) as the flow approaches the systolic peak, when it feels the force of the heart’s contraction. The complex twisted structures (seen in closeup of the last snapshot) are vortex surfaces, a way to visualize the structure of turbulent flow, with color (red to blue) indicating decrease in pressure. The pressure drops markedly, corresponding to increased flow rate, in the stenosed region (before the turn), and the high flow rate and low pressure continue around the bend.

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10 cardiac cycles. At the turbulence peak, the midstream flow velocity fluctuates at about 350 cycles per second, which means they’ll need to take a computational snapshot every thousandth of a second to capture details of the flow.

It’s the first good look at the transition to turbulence in a carotid artery, and along with new information, it demonstrates that tools are in place to zero in on the correlations between turbulent flow and stroke. “Within five years,” says Fischer, “it should be possible to routinely simulate weakly turbulent hemodynamic flows.” For medical researchers, this means it’s feasible to gather flow data on a range of patients with diseased carotid arteries and carry out long-term studies. What degree of turbulence and high shear stress under what conditions means serious risk of stroke? Getting the answers is now within sight.

**A Detailed Readout of the Flow Patterns in Carotid Arteries Will Help Doctors Identify Who’s at High Risk for Stroke.**