Predicting the ups and downs of chest pain

Imagine coronary arteries choked with a bumpy layer of atherosclerotic plaque. Suddenly, the plaque ruptures and a swarm of platelets rushes to the scene in an attempt to heal the injured artery wall. A gelatinous blood clot forms at the site of the rupture.

This microdrama causes an unpredictable chest pain that can strike even while a person is resting. In medical parlance, such pain is called unstable angina. It's a dangerous condition that may signal an impending heart attack.

New findings suggest that people hospitalized with unstable angina face a higher risk of suffering a heart attack during their hospital stay if they have a telltale protein circulating in their bloodstream. Other new research demonstrates the down side of an anticoagulant drug commonly prescribed for unstable angina. Investigators describe both studies in the July 16 NEW ENGLAND JOURNAL OF MEDICINE.

In the first study, Christian W. Hamm of the University Hospital of Hamburg in Germany and his colleagues focused on cardiac troponin T, a protein that helps the heart contract. Although cardiac troponin T is not normally found in the bloodstream, it does escape into the circulation when heart cells are damaged. Thus, Hamm's group speculated that a blood test for the protein might help predict the outcome for people with unstable angina.

The investigators studied 109 people who were admitted to the hospital with unstable angina. Troponin T showed up in the blood serum of 33 (39 percent) of the 84 patients who had reported ongoing chest pains even while resting – the more serious form of unstable angina. Of these 33 people, 10 went on to have a heart attack, and five of the 10 died during hospitalization.

By contrast, the researchers found no troponin T in blood serum from the 25 patients with the less severe form of angina, in which pain subsided somewhat after hospitalization. None of these patients suffered a heart attack or died during hospitalization.

The presence of troponin T in the bloodstream suggests trouble ahead for patients with unstable angina, concludes James H. Chesebro of the Mayo Clinic in Rochester, Minn., in a commentary accompanying the research report. The test for troponin T "can detect small amounts of damage to heart muscle and thus is a warning sign," he told SCIENCE NEWS. Such patients may need greater protection from blood clots, which can block the coronary arteries, causing a heart attack, he adds.

In most cases, doctors treat unstable angina by giving patients anticoagulant drugs such as heparin. During an attack of unstable angina, blood clots may form in an attempt to heal the damaged artery. With heparin preventing the formation of new clots, the body's own enzymes can begin to dissolve any existing blood clots, Chesebro explains.

While heparin is considered the gold standard of unstable angina therapy, a second study hints that recurrent chest pain – and even heart attacks – may arise when patients stop taking the drug.

A team at the Montreal Heart Institute, led by cardiologist Pierre Théroux, made this discovery after conducting a clinical trial comparing the efficacy of heparin, aspirin, a combination of heparin and aspirin, and a placebo in the treatment of unstable angina. The researchers focused on 403 people who had been hospitalized with unstable chest pains and who received a six-day course of their assigned treatment. After therapy ended, the team monitored all patients closely for several days, recording any problems.

In analyzing the data, they were surprised to note a greater number of serious setbacks among people who had received heparin alone than among people assigned to any of the other treatment groups.

Of the 107 people who received heparin alone, 14 developed complications – such as another bout with chest pain or a heart attack – within hours after they were taken off the drug. Only five patients in each of the other three study groups developed such problems.

Furthermore, 11 of the 14 heparin patients with complications went on to require urgent intervention, such as cardiac bypass surgery. Only two other patients – one in the aspirin group, the other in the placebo group – needed such drastic care after their unstable angina flared up again.

No one knows why post-heparin patients are at heightened risk. However, Théroux speculates that once the drug is stopped, the body's clot-producing machinery may become hyperactive. The body metabolizes heparin quickly, he notes, and within hours new blood clots can start to form. In contrast, aspirin's effect lingers for days after treatment is stopped. Study participants who received aspirin along with the heparin may enjoy some residual protection, Théroux says, because aspirin discourages platelets from clumping together – a key step in the clotting process.

Chesebro advises cardiologists to monitor angina patients carefully after discontinuing heparin therapy and to consider giving them a second anticoagulant drug at that time.

– K.A. Fackelmann

'Baked Alaska' cooked up in liquid helium

Baked Alaska seems an unlikely term to encounter in physics, but this culinary surprise, consisting of meringue baked around ice cream, serves as an apt description of an exotic, theoretical model accounting for a curious aspect of liquid-helium behavior. The model, proposed in 1984 by Anthony J. Leggett of the University of Illinois at Urbana-Champaign, suggests that high-energy particles produced by cosmic rays can trigger the otherwise inexplicable formation of one form of superfluid helium-3 at the expense of another.

In the baked Alaska scenario, high-energy electrons, created by the passage of cosmic-ray-generated muons through the supercooled liquid, deposit significant amounts of energy in spots less than a micron in diameter. Each intensely heated microball of liquid helium expands into a hot shell, leaving behind a pocket of cold superfluid helium. Isolated from the rest of the liquid, this cold core provides a protected environment in which a bubble of a different type of superfluid helium-3 can nucleate and start to grow.

Now researchers have obtained experimental evidence establishing the plausibility of Leggett's scenario. "Our results are certainly consistent with the [baked Alaska] model, though there are still some unanswered questions," says Peter E. Schiffer of Stanford University.

"The Stanford results show that at least the idea of nucleation by high-energy particles isn't totally crazy," Leggett notes.

Schiffer, Douglas D. Osheroff, and co-workers report their findings in the July 6 PHYSICAL REVIEW LETTERS.

Helium-3, a rare isotope of helium, becomes a superfluid – a liquid that flows without friction – at temperatures below 2.5 millikelvins. In this chilly state, helium atoms tend to form pairs. Because these pairs can arrange themselves in two different ways, helium-3 has two distinct superfluid states. Depending on the pressure and the magnetic field applied to a sample, the so-called A phase is more stable than the B phase at higher temperatures, whereas the B phase takes over at lower temperatures.

In 1977, Osheroff (then at AT&T Bell Laboratories) and co-worker Michael Cross showed that the superfluids had characteristics implying that the A phase, even when supercooled well below the temperature at which a transition from the A to the B phase should occur, cannot by itself spontaneously make the change. Because such phase transitions actually do occur, this puzzling feature led to a search for a mechanism that would explain how the transition happens.