the experiments, the abundances of gases trapped in water-ice chilled to 50 kelvins most closely approximated terrestrial conditions. That temperature has special significance, Owen says, because it corresponds to the temperature found in the region between Uranus and Neptune, where scientists believe comets first formed.

On the basis of this work, Owen suggests that the present noble gas composition on Earth, Mars, and Venus may represent the mixture of two reservoirs: trapped gas released when comets first rained down on the planets, and gas released in an entirely different pattern from rocky material inside the planets. Different planets – and different regions within those planets – draw from these two reservoirs in different proportions. For example, says Owen, material from deep within Earth's mantle, shielded from cometary bombardment, exhibits a noble gas composition more akin to that of rocky compounds such as meteorites. He adds that the higher abundance of argon on Venus indicates that the most recent series of comets striking that planet formed at temperatures below 50 kelvins, since the Tel Aviv experiments indicate that chillier water-ice traps more of the noble gases.

Owen emphasizes that his conclusions remain speculative because researchers have yet to measure the actual abundances of noble gases inside "new" comets – icy bodies that are visiting the inner solar system for the first time and haven't had a chance to warm up and expel most of the gases trapped within them. If future observations confirm the findings, he says, the recent work may provide one of the first direct links between comets and planetary evolution.

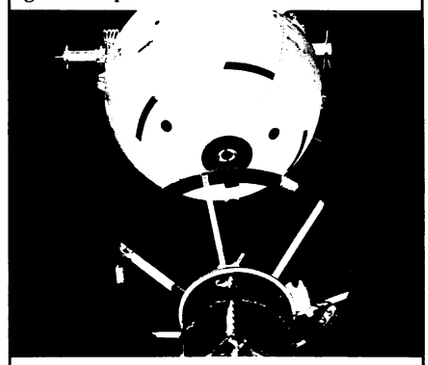
The new findings also strengthen the argument that comets played a crucial role in transporting biochemical compounds to the planets, says Oró, who spoke at this week's conference. Adds Owen: "The fact that you have to have comets coming in [to account for the noble gas abundances] means you're also bringing in carbon and nitrogen."

– R. Cowen

Bolt jammed tether's reel

Investigators have attacked the nuts and bolts of the problems encountered during deployment of the Tethered Satellite System (SN: 8/15/92, p.101) and found a bolt at fault.

Last-minute modifications to the satellite's tether-reel assembly included the addition of a quarter-inch-diameter securing bolt, which apparently jammed the mechanism in-flight, a board of international investigators reported last week.



NASA

The tethered satellite moves away from its boom during initial deployment.

The board's preliminary analysis indicates that this bolt prevented the free movement of a device called the level wind mechanism, which feeds out tether much as a fishing reel feeds out line. This is believed to have caused the tether to jam first at 179 meters and again at 256 meters.

Although shuttle astronauts managed to clear both snags, this problem would have prevented the full deployment of the satellite even without the other failures that occurred during the mission, the board said.

Investigators continue to examine all of the major problems encountered during the mission, including an initial failure to deploy the satellite and the final tether snag, which developed at the far end of the deployment boom. □

Blood-vessel growth genes stop making sense

Using a new genetic technology called antisense, researchers have completely shut down the operation of a gene that can cause the walls of arteries to thicken, reducing blood flow to a trickle. The scientists hope their strategy will one day benefit patients undergoing balloon angioplasty, a vessel-widening procedure that sometimes backfires, prompting the growth of cells within arterial walls.

The research team, led by Robert D. Rosenberg of the Massachusetts Institute of Technology in Cambridge, used antisense to prevent arterial thickening among rats whose neck arteries had been reamed by balloon angioplasty. Rosenberg, who also holds a post at the Harvard Medical School's Beth Israel Hospital in Boston, and his colleagues report their results in the Sept. 3 NATURE.

Each year, roughly 260,000 people in the United States undergo balloon angioplasty. Surgeons snake a catheter tipped with an uninflated balloon through the fat-clogged arteries of atherosclerosis patients. By inflating the balloon, the surgeons compress the deposits, widening the arteries.

However, balloon angioplasty has an undesired effect on an estimated one-third of those who receive the therapy. In these patients, the friction of the inflating balloon spurs smooth-muscle cells lining the arteries to grow and divide, causing the arteries to narrow again.

Earlier this year, Rosenberg's team and a separate research group reported that smooth-muscle cells grown in laboratory culture switch on a gene named c-myb before they begin dividing. This "proto-oncogene" spurs normal cell growth and differentiation in a wide range of tissues. When it becomes damaged through mutation, it can cause the uncontrolled growth characteristic of cancer.

Rosenberg and his co-workers set out to determine whether they could prevent arterial thickening by inactivating this growth-promoting gene. They applied a gel containing short oligonucleotides – the chemical building blocks of genetic material – to the neck arteries of angioplasty-treated rats. These "antisense oligonucleotides" were designed to bind to and block the "sense" of a chemical message called RNA, which genes use to direct a cell to make proteins. By specifically blocking the RNA of the c-myb gene, Rosenberg's team hoped to prevent it from prompting the inappropriate growth of smooth-muscle cells in the rats' arteries.

The researchers found that the arteries of rats treated with the antisense therapy contained no detectable levels of c-myb RNA, while the arteries of rats treated with a control compound had high levels of the substance. Moreover, the arteries of the antisense-treated rats showed virtually no muscle-cell growth after two weeks, whereas those of the control rats thickened considerably.

Rosenberg says he is "enthusiastic but cautious" about the prospects of using a similar approach in humans who undergo balloon angioplasty. He has applied for a U.S. patent on the technique, and is helping to form a company to commercialize the therapy.

"This is a very flexible approach to turning off [artery-thickening] genes," Rosenberg asserts. "[The c-myb gene] is one target, but there could be many other targets." He hopes to begin human clinical trials of antisense compounds within two years.

Volkhard Lindner, a vascular biologist at the University of Washington in Seattle, calls antisense "an interesting concept" for preventing arterial thickening after balloon angioplasty. But he cautions that Rosenberg's team has not proved that the antisense compound specifically blocks c-myb without interfering with other genes. He also suggests that the group may have jumped the gun by looking for arterial thickening among the rats after waiting only two weeks. Such thickening sometimes takes six weeks to develop, Lindner says.

– C. Ezzell