safety measure, they commanded the craft to blow open several pyro valves. With these rigid barriers out of the way, helium gas could flow though the check valves and pressurize the fuel and oxidizer tanks.

The absence of the pyro valves also meant that any NTO that had leaked through the check valves could mix with MMH, the panel conjectures. The enormous heat generated by the NTO-MMH mixture would have made titanium as soft as butter, rupturing the system and rendering the craft dead in space, Coffey says.

The panel charges that NASA's Jet Propulsion Laboratory (JPL) in Pasadena, Calif., which supervised the Mars Observer project, and its main contractor, Martin Marietta Astro Space, relied too heavily on designs for satellites orbiting Earth. Check valves function well, with little leakage, for short journeys in the warmer environment near Earth. But using them for a long interplanetary journey can prove problematic, says panelist Peter G. Wilhelm of the Naval Research Laboratory.

Indeed, NASA originally planned to rely on the check valves only for the first few days of the mission, says Mars Observer project manager Glenn Cunningham of JPL. The agency intended to pressurize the propulsion system five days into the flight rather than wait until the craft reached Mars 11 months later, when its thrusters had to be fired.

But those plans suddenly changed about six months before launch, Cunningham said. Scientists recalled that one of the Voyager spacecraft in 1976 had suffered a leaky regulator soon after its fuel system was pressurized, so they decided to hold off pressurizing Mars Observer until necessary.

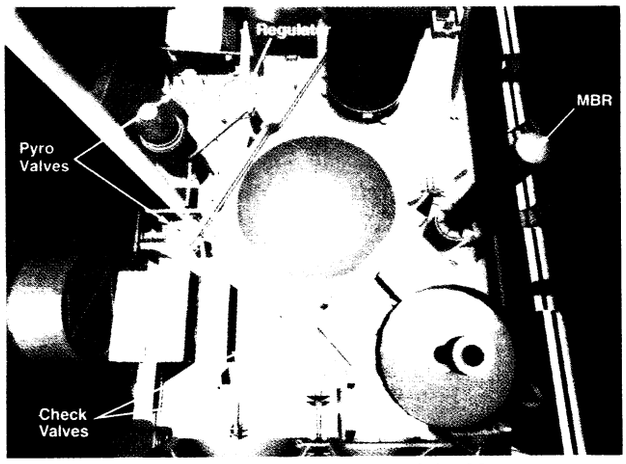

Illustration shows propulsion system of the Mars Observer, including fuel tank of monomethyl hydrazine (MMH). A small transmitter, the Mars Balloon Relay (MBR), lies at right.

Had JPL officials gone with their original plan, the NTO leak would have been far smaller (five days' worth of seepage rather than 11 months' worth), and the craft might have survived, Wilhelm says.

Wilhelm adds that in the long run, debating the cause of the Observer's failure has less significance than ensuring that NASA addresses the myriad other problems found by the panel. If NASA doesn't, he cautions, "then the next time [they launch a similar craft], it will be something else that gets them."

—R. Cowen

Immune therapy stems diabetes' progress

Diabetic mice treated with a particular monoclonal antibody have regained the ability to regulate their blood sugar, leading researchers to hope that a similar treatment may one day stop insulin-dependent diabetes in humans.

Monoclonal antibodies are proteins produced to seek out and attach to specific molecules. The anti-CD3 monoclonal antibody homes in on the CD3 molecule, which sits in the membrane of immune-system cells called T-cells and serves as a docking site that helps these cells recognize their targets. Physicians use this monoclonal antibody to prevent and treat the rejection of organ transplants, with mixed success.

Up to now, researchers had demonstrated that they could use substances that interact with the immune system — including this anti-CD3 molecule — to stop the development of diabetes, but only if given before the autoimmune attack destroyed most of the pancreas' insulin-producing beta cells (SN: 11/6/93, p.292). Treatment early in the disease process with different monoclonal antibodies, as well as the immune-system-suppressing drug cyclosporin, can keep these mice diabetes-free.

"This is really the first treatment that has shown any ability to cause remission," says Joan T. Harmon of the National Institute of Diabetes and Digestive and Kidney Diseases in Bethesda, Md.

Lucienne Chatenoud and her colleagues at Necker Hospital in Paris tried the approach in adult, nonobese diabetic (NOD) mice. Many such mice lose the ability to regulate their blood sugar as they mature because their T-cells attack their insulin-producing cells.

Within a week of developing high blood sugar or signs of T-cell attack, the NOD mice were given either low doses of the anti-CD3 antibody for five days or hamster immunoglobulin as a control injection. In some experiments, the researchers also gave the mice the drug cyclophosphamide, which speeds the development of diabetic symptoms.

Researchers then monitored the distribution of different T-cells in these mice. They also implanted bits of NOD-mouse pancreas into the kidneys of treated mice or attached pieces of tail skin from a different mouse strain to see what transplants survived. Within a month, 64 to 80 percent of the anti-CD3-treated mice regained the ability to regulate their blood sugar, the team reports in the Jan. 4 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

T-cells still seemed to infiltrate the pancreas, but they stopped attacking beta cells. Also, these mice rejected the skin grafts but not the pancreas implants, indicating that the treatment quelled the T-cell attack but did not totally destroy immune-system function, says Chatenoud. The anti-CD3-treated mice remained diabetes-free for at least four months.

A study by Chatenoud's colleague Jean-Francois Bach had shown that this same anti-CD3 monoclonal antibody causes side effects too severe to make it a useful treatment in people, she notes. But she hopes that an anti-CD3 antibody fragment, which also halted diabetes in mice, will prove beneficial to people without causing side effects.

However, "it's a long way before we know whether a similar method will work in humans," Harmon cautions.

Important differences exist between human and mouse diabetes and between the immune systems of these two organisms.

"It's easier to manipulate the disease in the NOD mouse than in humans," says George S. Eisenbarth, an endocrinologist at the University of Colorado Health Sciences Center in Denver. "In man, it might be too late to intervene when [we] see the high blood sugar."

Nevertheless, the French work "suggests there is an injury mechanism that is reparable," says C. Garrison Fathman of Stanford University School of Medicine. Autoimmune diseases often progress in fits and starts: Many diabetics go through a "honeymoon" period after their first signs of high blood sugar and seem to recover temporarily.

This study indicates that a one-time intervention with some immune-system regulator during this period may prevent the development of full-blown diabetes, he adds. *—E. Pennisi*

Science Service, Inc. is collaborating with JSTOR to digitize, preserve, and extend access to *Science News*. www.jstor.org