ficult, and as factorization algorithms improve, such cryptosystems are placed in jeopardy.

"If you had a sufficiently dedicated adversary, then even numbers as large as 512 bits (154 digits) probably will not be safe for more than another 3 to 4 years," Manasse says. In the absence of radically new factorization methods, though, cryptographers can respond simply by keeping ahead in the digit race. The only real danger is to secrets that must remain secret for more than a few years.

In 1977, Rivest and his co-workers published an "unbreakable" cipher based on a 129-digit number, with a $100 prize offered for its solution. At the time, factoring a 40-digit number was an accomplishment. But 129-digit numbers are only about 100 times harder to factor than 100-digit numbers. Manasse expects to be able to upgrade the current network easily by a factor of 10, and possibly by a factor of 100.

Asked if he thinks he can win the $100, Manasse says, "If I do I'll have to split it a whole bunch of ways." ■ **BARRY A. CIPRA**

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Discovery Gets a Clean Bill of Health

NASA has made hundreds of fixes to the shuttle; the evidence from Discovery is that virtually everything worked

WHENEVER ENGINEERS have to go into an already complex piece of equipment and make hundreds of modifications, as the National Aeronautics and Space Administration (NASA) did to the space shuttle system in the aftermath of the 1986 Challenger accident, a wise person has to be concerned: there is always a chance that the fixes will create more problems than they solve.

Thus the institutional sigh of relief at the dramatically successful flight of the space shuttle Discovery on 29 September to 3 October, a flight that marked the nation's return to manned space flight after a 32-month hiatus. Not only was the mission itself nearly flawless, but all the post-flight indications are that every piece of modified equipment performed as expected.

"With all the changes it's incredible how few problems we've had," says Joseph E. Mechelay, manager of the Flight Data and Evaluation Office at NASA's Johnson Space Center near Houston. "The results of this flight indicate that we haven't screwed anything up."

Indeed, NASA treated this mission, the 26th launch of the shuttle since flights began in 1981, as if it were the test flight of a brand new vehicle. And now that Discovery is back on the ground, it is being examined in minute detail. Some highlights:

■ **The solid rocket boosters.** These are the huge white crayons on either side of the shuttle's rust-colored external fuel tank. Rather like skyrockets in a fireworks display, they fire for the first 2 minutes after liftoff and then fall away into the ocean, where they are retrieved for reuse.

In the last, fatal flight of Challenger on 28 January 1986, however, a design flaw allowed exhaust flame to burn through a rubber O-ring gasket in one of the boosters, and then to escape from the side through a joint in its metal casing. As a result the booster tore loose from its mount 73 seconds after launch and destroyed the whole vehicle. Much of the 2½ years since then has been spent in testing and validating a highly modified design for the boosters. In fact, says one manager at Morton Thiokol, the Utah-based company that builds the boosters, about four to five times as much work went into this effort as went into the original development in the mid-1970s.

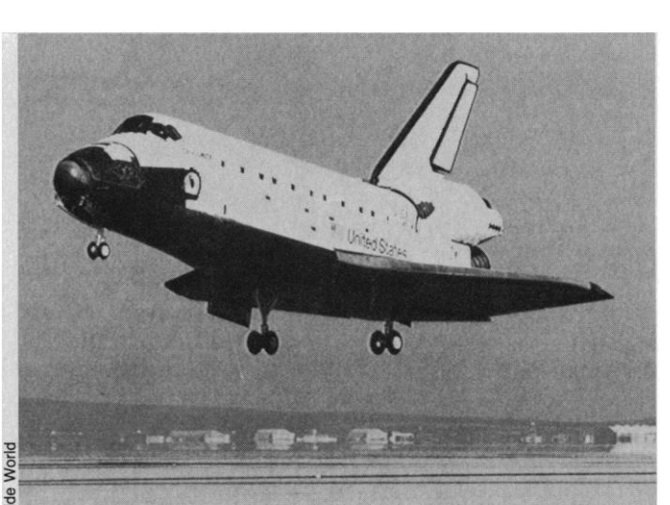
Discovery's boosters are now undergoing a preliminary examination at Florida's Kennedy Space Flight Center. "All indications are that they worked as planned," says Russell Bardos, head of NASA's shuttle propulsion office. "A cursory look shows no anomaly at all."

"It doesn't appear that any gas got to any O-rings," agrees Myron Uman, executive director of an ad hoc committee set up by the National Research Council to provide independent oversight of the booster redesign.

Once the preliminary inspection is completed in Florida the boosters will be sent back to Morton Thiokol in Utah, where they will be torn down, examined in detail, and then refueled for another flight.

■ **The main engines.** These are the three large cones located in the tail of the orbiter. During launch they burn some 780 tons of liquid hydrogen and liquid oxygen fuel located in the external tank. They produce more thrust per kilogram of weight than any engines ever built. They have been plagued with problems from the beginning, notably with bearing wear and cracks in the engines' many weld joints. During the hiatus, NASA therefore made some 35 upgrades to the engines. The agency also tightened up on inspection and certification procedures, and inaugurated the most aggressive ground testing program in the history of the main engines. Engines under test are routinely fired for more than 2000 seconds at a stretch, for example, about five times as long as they will be fired during an actual flight.

The data from Discovery suggest that the rigor paid off. Telemetry showed no indication of any problem with the engines during ascent, and a preliminary inspection on the ground shows none of the problems that



Touchdown. *After 4 days in orbit, the space shuttle Discovery makes its final approach for a landing at Edwards Air Force Base in California.*

had appeared previously.

■ **The orbiter.** During the standdown period NASA made 210 major and minor changes to the shuttle orbiter itself, not counting some 100 changes to the spacecraft's software. On previous missions, for example, the beryllium brakes on the shuttle's landing gear had shown a distressing tendency to shear and crack. Discovery's new carbon brakes seem to have worked much better: no damage was reported.

Meanwhile, there was the orbiter's new crew escape system—clearly not something that one would want to test for real. During Discovery's 4 days in orbit, however, the crew did practice setting up the curved, telescoping pole that they would use if they ever had to bail out. The idea is that they would slide through the hatch and along the pole while the orbiter is in a more or less level glide; the pole would then carry them outward far enough that they would not collide with the wing. In any case, the practice session went very smoothly, even easier than on the ground.

The mission was not without its little glitches, of course. But the three most significant problems—a balky antenna dish, a failed backup circuit on one of the shuttle's on-orbit thrusters, and an iced-over cooling system—were decidedly minor league.

■ **Weather forecasting.** The decision to launch Discovery on 29 September despite abnormally slow high-altitude wind speeds was not taken lightly. The orbiter's computers had been programmed for flight through the much brisker winds expected at that time of year, so that the milder conditions would have paradoxically resulted in unacceptably high levels of stress on the vehicle. Moreover, reprogramming the computers would have taken so long that the winds would have probably changed again anyway.

However, the weather forecasting facilities at Cape Canaveral have been greatly upgraded during the hiatus, so that they are now as good as any in the country. In particular, a series of radar-tracked weather balloons allowed shuttle engineers to monitor wind speeds in real time. (The data had previously been delayed by 3 hours.) These data, in turn, allowed the engineers to refine their structural models and to show that the shuttle's margin of safety would not, in fact, be exceeded. The upshot was a go-ahead for launch at 11:37 a.m. EDT, 98 minutes later than scheduled, and just 20 minutes before the arrival of a rainstorm that would have canceled the launch.

For future missions, NASA may try to circumvent this kind of problem by having different versions of the shuttle software available for a variety of wind conditions.

■ M. MITCHELL WALDROP

New Clues About Kaposi's Sarcoma

Several growth factors, including a novel one, and infection with the AIDS virus may promote the growth of Kaposi's tumors in patients with AIDS

TWO LABORATORY GROUPS, working independently, report new information about Kaposi's sarcoma, best known in the United States today as a skin tumor that occurs in some AIDS patients. Data from Robert Gallo's research group at the National Cancer Institute (NCI) and their collaborators indicate that Kaposi's is not really a cancer and that chemical factors sustain the growth of Kaposi's cells in vitro. Gilbert Jay, also of NCI, and his colleagues say that Kaposi's is a cancer. They find that male, but not female, mice that carry a regulatory gene from the AIDS virus develop Kaposi's-like tumors. The Gallo group's papers appear on pages 426 and 430 in this issue and the report from Jay and his co-workers is in the 13 October issue of *Nature*.

None of the new work establishes a cause for Kaposi's, but it increases the understanding of how Kaposi's tumors may grow. "There are some obvious similarities in the data from the two laboratories," says Zaki Salahuddin of NCI. He notes that both groups have a mouse model for the disease, both agree that spindle-shaped cells are important in the growth of the lesions, and both propose that the human immunodeficiency virus type 1 (HIV-1) that causes AIDS may indirectly trigger Kaposi's in people with HIV-1 infection. Neither group can find an HIV-1 gene or its product in cells from a Kaposi's lesion, however.

About 20% of people who die from AIDS are also diagnosed with Kaposi's, according to the Centers for Disease Control in Atlanta, although more may actually have the disease. Kaposi's lesions often grow in the dermis of the skin and contain many different cell types. Spindle-shaped cells, which may arise from endothelial cells that line the blood vessels or ducts of the lymphatic system, predominate. The tumors are richly supplied with blood vessels and can appear in several parts of the body simultaneously. Kaposi's tumors can also occur in the lungs, liver, spleen, and digestive tract and can cause death.

The new work makes several contributions to the study of Kaposi's. First, it establishes a method by which researchers can grow large numbers of Kaposi's cells in tissue culture for a long time which, until now, had not been possible. Gallo, Salahuddin, Shuji Nakamura, and Barbara Ensoli of NCI, and Peter Biberfeld of the Karolinska Institute in Stockholm and their colleagues, like many others, looked for a virus that might cause Kaposi's and were unsuccessful. For their search, they had to be able to grow Kaposi's cells in vitro. After a year of effort,

"We can also ask whether other tumors are caused by chemical factors."

Salahuddin and Nakamura found that a T cell line infected with HTLV-II, a human retrovirus distantly related to the AIDS virus, secretes a factor that promotes the growth of Kaposi's cells in culture.

Second, the new results indicate that a complex series of chemical factors stimulates the growth of Kaposi's cells in vitro and that at least one may be novel. HTLV-II–infected T cells, those infected with the human retroviruses HIV-1, HIV-2, or HTLV-I, and possibly Kaposi's cells themselves, make the new factor.

"Don't confuse the novel factor with the signal needed to initiate the disease in vivo," says Salahuddin. Although it may exist and play a role in vivo, the researchers have not yet identified it and have only found it in cultured cells. They do have evidence of its biological activity in vivo, however. When *nude* mice (which lack immune system function and do not reject the foreign cells) are injected with Kaposi's cells that make the factor, the mice develop Kaposi's-like tumors. The tumors are clearly made of mouse cells, says Gallo, and, unlike most cancer cells, their chromosomes are normal.

A third finding that may help to explain how Kaposi's tumors develop in AIDS patients comes from Jay and Jonathan Vogel, also of NCI, Paul Luciw of the University of California at Davis, and their co-workers.