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.

Their new data indicate that the *tat* gene from HIV-1, which increases the production of viral proteins in cultured cells, may somehow trigger the growth of Kaposi's tumors. Both male and female transgenic mice that carry the HIV-1 *tat* gene mice express it in their skin cells and about 15% of the males develop Kaposi's-like tumors on their backs. Jay does not know why female mice fail to develop the tumors nor why only a small fraction of male mice do.

Jay and his collaborators interpret their new data to mean that the *tat* gene from the AIDS virus has the potential to cause cancer, in this case Kaposi's sarcoma. If this is true, it remains to be explained why all HIV-infected people do not develop Kaposi's. The researchers have not been able to show that the Kaposi's-like cells in the mice express the HIV-1 *tat* gene, nor can they detect *tat* expression in cultures of the tumors.

Both research groups propose models for the growth of Kaposi's. Gallo and Salahuddin agree with Jay that the cause of Kaposi's in HIV-infected individuals is HIV, "but it is indirect," says Gallo. When HIV infects T lymphocytes, it stimulates the cells to release the new growth factor. This factor may activate endothelial cells to become spindle-shaped cells. Once activated, Kaposi's cells make many factors, including interleukin-1, basic fibroblast growth factor, plasma-derived growth factor, chemoattractants and chemosuppressors that are not yet identified, and perhaps the new factor, says Gallo. Collectively, the factors recruit or stimulate other cells types such as granulocytes and macrophages from the blood and fibroblasts from the skin. Additionally, some of the factors may stimulate blood vessel formation. Jay thinks that growth factors may be involved in these processes but he sees a more direct role for the *tat* gene.

Many questions about Kaposi's remain to be answered. For example, no one can explain why it is predominantly a male disease. In the United States it occurs primarily in homosexual men with HIV infections. In Africa, it is widespread among young men and women, many of whom are not infected with HIV-1. Some older men in Mediterranean countries often have a milder form of Kaposi's. It is not clear what causes the disease, or if it has the same cause in these various populations.

The new work should allow researchers to study cellular and molecular mechanisms that trigger Kaposi's growth in vitro and may also help to identify its origins in vivo. "We can also ask whether other tumors are caused by chemical factors," says Salahuddin. If so, the new information may lead to different ideas about possible causes of tumor formation. ■ **DEBORAH M. BARNES**

Does Earth Fill Its Own Magnetosphere with Ions?

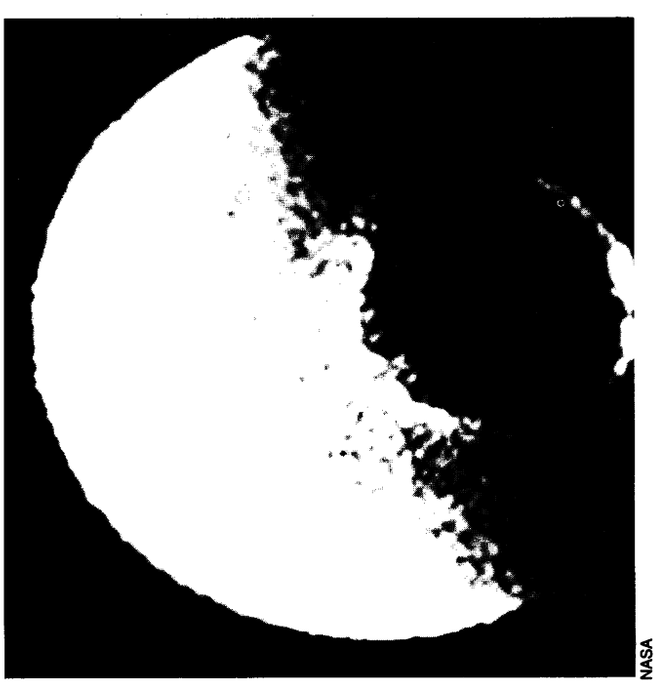
The ionosphere went from being an insignificant to a major source of magnetospheric ions; could it be the dominant source?

WHEN CHARLES CHAPPELL went to graduate school in the mid-1960s, he was taught that the ions filling the teardrop-shaped magnetic field enveloping Earth, including those trapped in the Van Allen radiation belt, came from the sun. The ions in the nearby ionosphere of the upper atmosphere, being 100 to 1000 times less energetic than those carried from the sun by the solar wind, seemed an unlikely source.

That has all changed as improved instrumentation has broadened the range of ion energies and densities that can be measured. Now Chappell, who is at the Marshall Space Flight Center in Huntsville, is arguing that the ionosphere is so leaky that it could be the sole source of observed ions found in the magnetosphere above it. If the sun also contributes a significant share, that could be a further indication that space physicists are still failing to detect a significant portion of magnetospheric ions. Because ions give the magnetosphere its physical substance and carry energy to fuel phenomena from the aurora to radio interference, sorting out ion sources is of major interest.

Chappell points out in a recent *Reviews of Geophysics* paper that researchers' thinking on the source of ions filling Earth's magnetic field has been shaped by the limitations of the available instrumentation. For two decades the detectors carried aloft by rockets and satellites tended to measure either high-energy charged particles high in the magnetosphere, where there are a few particles per cubic centimeter, or low-energy particles found at high densities of thousands of particles per cubic centimeter in the innermost magnetosphere and ionosphere. (Air at sea level has more than a billion molecules per cubic centimeter.) In fact, says Chappell, the innermost magnetosphere, called the plasmasphere, for years fell in a crack—it was at such a high altitude that ionospheric specialists left it to those studying the magnetosphere, but the magnetospheric community neglected it because the low energies of its ions linked it so closely to the ionosphere. Any upward flow of relatively low-energy ions went unnoticed.

Improved technology has gone far in closing this gap. Earth's ionosphere is now seen as a "multifaceted fountain," in Chappell's words, that spews low-energy ions from a



NASA

Earth in UV. *This ultraviolet image made by Dynamics Explorer 1 shows the dayglow of the dayside and the emissions of the auroral oval around the north pole. In addition to the downward flow of energy to the upper atmosphere implied by this emission, the Dynamics Explorer helped elucidate the upward flow of ions.*