Did Voyager 2 sense Jupiter at Saturn?

Jupiter's magnetic field had already been described as "the largest structure in the solar system," when, in 1976, scientists got a hint of just how big it might *really* be. The Pioneer 10 spacecraft, by then outside the orbit of Saturn (as far beyond Jupiter as Jupiter is from the sun), indicated that the ever-present solar wind had abruptly disappeared for a day. Since Saturn was around its orbit, far away from the spacecraft, the startling inference was that the probe had passed through Jupiter's magnetic tail, a region where the field lines are stretched out by the solar wind. The magnetic "walls" of the tail also keep out the solar wind's flow of charged particles, and it was the absence of such particles that the spacecraft had detected — nearly 700 million kilometers from Jupiter.

A few years later, it was noted that Voyager 2, which would fly past Saturn in August 1981, might be doing so just when the wide-ringed planet would be immersed in the Jovian tail, an alignment that occurs only about every 13 years. More than just representing a curiosity, the idea raised the rare chance of being able to study a planetary magnetic field that was, however briefly, free from the solar wind's influence. And indeed, while approaching Saturn last year, Voyager 2 several times took measurements consistent with brief passages through Jupiter's tail (SN: 5/23/81, p. 324), which might have been flexible enough, so far from its source, to have moved back and forth across the spacecraft's trajectory.

The Saturn flyby is now history, but was Jupiter's tail there at the same time? "I believe it," says William S. Kurth of the University of Iowa, but the evidence, he notes with frustration, is only circumstantial. The key elements in the case are two: Saturn, like Jupiter (and Earth, for that matter), is a source of radio waves, and some of its emissions, known as Saturn kilometric radiation (SKR), vanished for four days while the spacecraft was inside Saturn's magnetosphere (SN: 9/19/81, p. 182). It has been suggested that the SKR is driven by solar-wind particles pouring into the magnetosphere at its poles, so replacing the solar wind with the weaker flow of particles from Jupiter's tail could account for the effect. On the other hand, an extreme, temporary weakening of the solar wind — not involving Jupiter's tail — might produce the same result. The other ambiguous clue is the fact that Voyager 2 did not exit from the trailing skirt of Saturn's magnetosphere until it was more than twice as far from the planet as expected. Again, replacing the solar wind's pressure with that of the weaker tail might have let the skirt flare out, moving it away from the spacecraft, but again, so might a weakened solar wind.

For a while, Kurth and his colleagues had what they thought was the clincher: detection within Saturn's magnetosphere of a type of "continuum radiaton" thought to exist only at Earth and Jupiter — a characteristic Jovian "signature," in other words, transmitted down the tail. Last Friday, however, an example of the same signature turned up in a reexamination of data from Voyager 1, which passed Saturn when the Jovian tail was nowhere around. A Jovian component in Saturn's continuum radiation is still a possibility — its identifying frequency characteristics might simply have been masked by local effects at Saturn. The low end of its frequency spectrum could have been cut off by Saturn's local plasma frequency, and the high end by the plasma frequency of the solar wind, leaving the Jovian component indistinguishable from Saturn's own contribution. Detailed study of solar-wind conditions during the SKR dropout may help, such as by showing that the pressure was not abnormally low at the time, but Saturnian continuum radiation is a significant find in its own right. — *J. Eberhart*

Drug against surface herpes approved

Last week, a drug to treat initial genital herpes infections as well as localized herpes infections on the surface of the bodies of immunosuppressed patients was approved by the U.S. Food and Drug Administration. The drug is acyclovir (trade name Zovirax Ointment 5%). It is manufactured by the Burroughs Wellcome Co. in Research Triangle Park, N.C.

The approval is important for several reasons. Until now there has been no proven effective treatment for genital herpes infections, which currently afflict some 15 to 20 million Americans and which, according to the Centers for Disease Control in Atlanta, are spreading at epidemic proportions — some 400,000 new cases a year. The increase appears to be particularly rapid among white, educated, sexually active men and women between the ages of 25 and 35. Nor has there been any proven effective treatment for surface herpes infections in organ transplant or cancer patients whose immune systems have been suppressed with drugs.

In addition, acyclovir's sanction is one more indication that the FDA is serious about stepping up its approval of new drugs (SN: 12/5/81, p. 359). But perhaps most crucially, acyclovir's acceptance is one more sign that an era of antiviral medications has finally arrived — that scientists at last are learning how to design compounds that attack viruses selectively without impairing healthy cells (SN: 3/20/76, p. 186). To date, only a handful of antiviral drugs are on the American market (SN: 11/4/78, p. 309). In fact, in the opinion of Henry Balfour, chief of virology at the University of Minnesota Hospitals in Minneapolis, "Acyclovir is the most exciting antiviral drug which we have seen in the last decade."

Acyclovir was discovered in 1974 by Howard Schaeffer and Lilia Beauchamp of Burroughs Wellcome. Subsequent discoveries showed that it killed herpes viruses but spared healthy cells: healthy cells take up much less of it than herpes-infected cells do; less acyclovir is converted to an active form in healthy cells than in herpes-infected cells; acyclovir interferes with the herpes virus's DNA polymerase enzyme much more than it interferes with cells' DNA polymerase enzyme, thus inhibiting viral DNA replication but not healthy cells' DNA replication. Acyclovir's approval by the FDA was based not only on such *in vitro* discoveries but on several years of animal research and also on five clinical trials conducted between the spring of 1979 and the fall of 1980. Four of the trials tested acyclovir's effectiveness against genital herpes (essentially a herpes simplex II infection) and were conducted at the University of Washington in Seattle, Emory University in Atlanta, the Centers for Disease Control and the University of Vermont. The trials showed that acyclovir speeded the healing of initial genital herpes infections, reduced viral shedding (the length of time that live herpes viruses were present in sores and able to be transferred from person to person through sexual contact) and sometimes reduced pain from initial infections. The fifth trial, which tested acyclovir's effectiveness against localized herpes simplex I and herpes simplex II infections on the surface of the bodies of immunosuppressed patients, was conducted at the University of Alabama in Birmingham. Still other clinical studies carried out at other centers confirm and extend the above results (SN: 7/18/81, p. 37).

Scientists at about 100 medical centers throughout the United States and abroad are now undertaking studies to further explore acyclovir's effectiveness against herpes infections, Ronald E. Keeney, medical advisor in the department of clinical investigation at Burroughs Wellcome, told SCIENCE NEWS. For instance, acyclovir ointment is being tested in treating recurrent genital herpes infections. Recurrent infections are currently impossible to prevent because viruses retreat into nerve cells in between attacks in the genital skin area. Acyclovir ointment is also being tested against cold sores, which are due to herpes simplex I and which afflict some 25 million Americans. Injectable acyclovir is being investigated to see whether it can keep genital herpes infections from recurring. It is also being tested to see whether it is able to heal systemic herpes infections in immunosuppressed patients. — *J. A. Treichel*