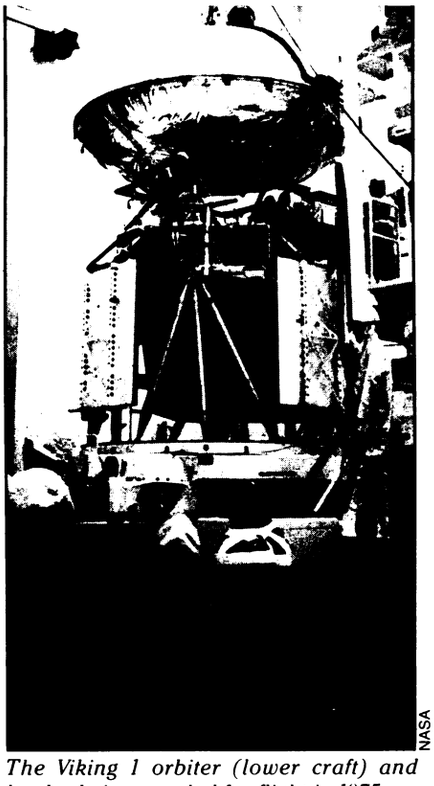
Silence comes to Mars orbit

Viking 1 left the earth for Mars on Aug. 20, 1975, nine days late, riding a powerful but touchy rocket whose maiden flight the previous year had ended in a brilliant explosion when the range safety officer destroyed it following the failure to ignite of its sophisticated upper stage. What actually rode the Mars-bound flight, in fact, was originally to have been Viking 2, but it was installed in place of its twin rather than delay the launch further by waiting for a repair. The National Aeronautics and Space Administration even pushed its luck a little more by bending a safety rule and swapping the two Vikings without first emptying the rocket of its more than 200,000 kilograms of fuel. Two spacecraft were sent to Mars by that booster—Viking 1 (like Viking 2, launched three weeks later) is the collective name for one vehicle that would orbit the planet and another that would land on it.

Both of the Viking 2 craft are now "dead" — a gas leak ended the orbiter's operations two years ago, and the lander succumbed in April of this year. The Viking 1 pair have proved tougher. The lander is programed to go on working until December of 1994, giving the mission an active presence on Mars for as long as 14 more years. But the Viking 1 orbiter, the last working spacecraft circling the planet, has come at last to the end of its line. Possibly before the end of this week, officials predicted at presstime (Aug. 5), "VO-1" was expected to be out of steering gas, leaving it mutely circling the planet it has studied for more than four years.

Reaching its circum-Martian orbit on June 19, 1976, VO-1 was intended first merely to carry its piggyback landing craft while photographing potential landing sites, and then to relay initial data from the lander to earth. The momentous landing occurred that July 20, and the orbiter stayed at its double assignment of support (for the lander) and science (on its own). Thanksgiving came, and with it solar conjunction, placing Mars and earth on opposite sides of the sun and temporarily shutting off communications. But after conjunction, the orbiter was still ticking, and NASA instituted its "Extended Mission," scheduled through April of 1978. Then came the "Continuation Mission," the "Survey I Mission," the "Mapping Completion Mission" and finally a "Survey II Mission" that began this April 24.

For two years, the Viking flight team has been down to barely two dozen people from its original 800 (which then included scores of scientists now back in their laboratories), and the overworked staff has sometimes had to argue for a little extra money to keep the still-operable orbiter going. But the end has been inevitable. The craft has been running low on fuel for its



NASA

The Viking 1 orbiter (lower craft) and lander being coupled for flight in 1975.

engine (used to change the height and shape of its orbit) as well as on the steering gas needed to keep its solar panels pointed at the sun, its antennas at earth and its scientific instruments at Mars. Finally, with the orbiter's infrared and water-vapor measurements behind it and with more pictures taken than the mission's planners ever dreamed of (the two orbiters together captured over 50,000), project officials decided on one last test: three final firings of the engine to use up the last of its fuel and see how efficiently the load of propellant carried from earth had been utilized (SN: 6/21/80, p. 389). The stabilization maneuvers needed during the firings were also expected to use up the steering gas, but when the "burns" were conducted last month, neither supply was exhausted. An additional burn was done, and it indeed used up the fuel, but a bit of steering gas—sufficient by itself for much photography — remained. "I think it's up there making its own gas supply," quipped Nancy Evans of the orbiter team at Jet Propulsion Laboratory in Pasadena. Not even an instability that showed up after the final firing seemed able to run out the gas. To ensure being able to conduct final maneuvers and play back a last few frames on the craft's tape recorder, engineers signaled open a valve to let in a tiny bit of leftover helium formerly used for pressurizing the engine fuel, but it would be only a matter of days.

At NASA's Langley Research Center in Virginia, where the mission plan was originally developed, officials plan a commemorative reunion of Viking staffers. At Jet Propulsion Laboratory, which guided the craft for half a decade, they're planning a wake. (See Viking photos p. 89.) □

Human antibody: Pure and simple

When the human body responds to an invading microorganism or chemical, it produces a complex and unpredictable mix of antibodies targeted to different parts of the intruder. These antibodies could be useful in studying and treating human disease, but scientists would need an abundant source of pure antibodies specific for a foreign or disease-related material. Five years ago British scientists discovered a method of combining two types of mouse cells into hybrid cells that grow indefinitely in the laboratory and produce quantities of specified "monoclonal" antibodies (SN: 12/30/78, p. 444). While these mouse antibodies have been tremendously useful in laboratories around the world, the human body's natural reaction against foreign substances is expected to limit the value of mouse antibodies in clinical diagnosis and treatment. Now, Stanford University researchers report success in creating hybrid human cells that in the laboratory produce a pure human antibody.

One source of human cells for the Stanford experiments is cancerous bone marrow cells selected for their ability to grow under laboratory conditions. These cells are fused with spleen cells obtained from patients with Hodgkins' disease. Spleens are routinely removed from such patients as part of the clinical evaluation, but only spleen cells that appear unaffected by the disease are used.

Lennart Olsson and Henry S. Kaplan fused the spleen cells and bone marrow cells and identified hybrids, called hybridomas, that make antibodies, as spleen cells do, and can propagate in the laboratory. Because the Hodgkin's disease patients are exposed to the chemical 2-dinitrochlorobenzene (DNCB) in the course of their diagnostic tests, some of the spleen cells make antibodies to that chemical. Olsson and Kaplan now report that some of the bone marrow-spleen hybrids, and their descendants, make pure antibodies to DNCB.

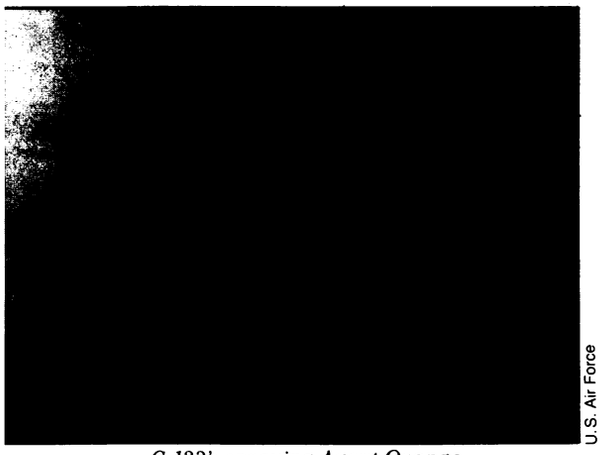
The value of the method will be limited, however, if patients must be exposed to a different substance, including disease-causing agents, for each antibody scientists want to obtain. Olsson and Kaplan, however, have already taken the first step in avoiding this problem and making their technique more widely applicable. They have exposed spleen cells to a foreign substance, sheep red blood cells, after the spleen had been removed from the patient. Kaplan explains that when they fuse spleen cells with cancerous bone marrow cells, some of the resulting hybridomas make antibodies to the sheep cells.

"These experiments are just to demonstrate feasibility," Kaplan says. "Antibody to DNCB and to sheep red blood cells is of

no earthly use to anybody." But if the technique works to produce any of a broad spectrum of antibodies, its applications will be numerous. The specific antibodies could be used to rapidly diagnose viral and bacterial infections, cancers and damage due to heart disease. They could also be used in making vaccines, bolstering the efforts of the immune system and carrying drugs to specific sites within the body.

In addition to determining the range of substances that will provoke specific human antibodies in laboratory cells, Olsson and Kaplan plan to investigate use of cells that can be obtained more easily than spleen cells. Human blood cells called beta-lymphocytes make antibodies, and the researchers suspect that such blood cells could be used to supply the antibody-making ability to hybrid cells. □

Reprieve for Agent Orange



U.S. Air Force

C-123's spraying Agent Orange.

The C-123's would swoop down low over the jungle, dispensing a fine mist of 2,4-dichlorophenoxyacetic acid (2,4-D), 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and trace amounts of a contaminant, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (dioxin). Within a few days, the lush jungle would be eerily nude, sometimes exposing vast warrens of hospitals, kitchens and warehouses snugly dug into the earth. The Viet Cong, if not already rousted by the loss of camouflage, could be sought and destroyed with less hazard to U.S. troops.

Agent Orange, the potent herbicide used to strip the Vietnamese countryside of protective coverage, has left its manufacturers and the U.S. government a legacy of lawsuits and countersuits (SN: 3/17/79, p. 166; 1/26/80, p. 59). Veterans' groups say that exposure to Agent Orange has led to birth defects in their children, loss of libido, cancer and various neurological problems.

One of the nagging scientific questions has been whether exposure to Agent Orange really does affect a male's offspring born years later. A study released late last week by the Department of Health and Human Services indicates that there are no significant effects on mating, fertility or health of offspring in male mice fed the components of Agent Orange. Researchers James Lamb, John Moore and Thomas Marks did find evidence of liver and thymus gland toxicity in the treated animals, but the problems disappeared when the mice returned to a normal diet.

"We saw no significant decrease in fertility or survival of offspring, and no increase in birth defects," Lamb told SCIENCE NEWS. The doses, he says, were somewhat higher than what humans may have been exposed to in Vietnam, but veterans' exposures have not been well established.

When told of the study, Samuel S. Epstein, a long-time researcher into the hazards of dioxin, said that while it appeared fairly solid, some of the data that showed an apparent inversion between dosage and response in mating frequency were bothersome.

"The findings are consistent with two things," he says. "The fact that in the literature there are fairly clear-cut indications of toxic effects, and that it's been known for some time that the effect of dioxin in general is not mediated by a dominant lethal effect." There are bacterial data indicating dioxin is mutagenic, he notes.

But whatever the problems from Agent Orange, they may be mixed in with other effects of the war. A report issued last week by a government task force set up to coordinate Agent Orange research called for a study to determine whether service in Vietnam, rather than solely Agent Orange exposure, places veterans at high risk of developing health problems. While noting that dioxin's carcinogenicity in animals has been confirmed, the report called for further study to define the health risk posed by Agent Orange.

Specifically, it recommended long-term studies, since the health effects may not show up for 15 to 20 years, and verification of five European studies (SN: 4/12/80, p. 230) that show a correlation between the components of Agent Orange and cancer in exposed workers.

The task force concluded that a significant increase in knowledge is not likely to be realized for several years, but important information may come from studies (due to be released this month) of U.S. workers accidentally exposed to 2,4,5-T and dioxin, from work on congenital malformations conducted by the Center for Disease Control, and from a proposed study of Air Force personnel involved in Agent Orange application.

But in view of all the planned studies, it may be some time before the effects of the fallout from one of the United States' more potent weapons is fully quantified. □

The machinery of depression

It is generally accepted among psychiatrists that serious, "endogenous" depression — in which depression seems to envelop the person, regardless of life events — is rooted, often genetically, in a person's biochemistry. At the other end of the spectrum is environmentally induced, "normal" depression — a finite episode triggered by the loss of a loved one or by other disturbing situations. Somewhere in between lie various combinations of depression of various origins and severities.

All this has been inferred theoretically from years of experience and numerous studies — primarily statistical ones, such as Seymour Kety's twin studies in Copenhagen (SN: 10/7/78, p. 244). Laboratory experiments have also suggested that the brain's natural opiates, enkephalins and endorphins, play significant roles in human depression, or lack of it (SN: 11/25/80, p. 364).

Now, University of Iowa researchers report biochemical confirmation of the existence of distinct forms of depression, each mediated by the body's hypothalamic, pituitary and adrenal systems. Reporting in the July ARCHIVES OF GENERAL PSYCHIATRY, George Winokur, Michael A. Schlesser and Barry M. Sherman utilized the "dexamethasone suppression test," which previously has been shown to distinguish clinically depressed from nondepressed persons (SN: 4/28/79, p. 285). The nondepressed person's response to the drug dexamethasone involves a lowering, or suppression, of the body's cortisol level. The depressed person, however, exhibits no such suppression.

Through the test, the researchers say they could distinguish not only among depressed persons and "normal" controls, but among patients with primary depressive illness — significant depression or manic-depression accompanied by no other psychiatric diagnosis — and secondary depressive illness; nonsuppression was found in nearly half the primary population but in *none* of the 151 persons with either secondary or no depression.

Moreover, the test was able to distinguish among "the three familial subtypes of primary unipolar depressive illness," the researchers report. Nonsuppression was found in 75 percent of those who had a first-degree relative with depression and no related disorder such as mania, alcoholism or antisocial behavior; in 44 percent of those depressed persons with no first-degree relative with a psychiatric illness; and in 7 percent of those with a first-degree relative with alcoholism and/or an antisocial personality disorder. The latter finding indicates that depression in a person from such a family may be the *result* of alcoholism or antisocial personality, rather than the cause of them. □