
The last Viking: Triage

Designed to work for 90 days, the Viking 1 landing craft reached the surface of Mars on July 20, 1976, and operated for nearly six and a half years before falling mysteriously silent last November (SN: 1/8/83, p. 20). Since then, a small group of engineers at Jet Propulsion Laboratory in Pasadena has been struggling to reestablish communications, aided by consultants reennlisted from among the old Viking hands at Martin Marietta Corp. (the spacecraft's builder) in Denver. With the project down to a skeleton part-time crew and a minuscule budget, there have nonetheless been long hours, plenty of midnight oil, "tiger teams" assembled to check out particular possibilities for resurrection (sometimes requiring a search to locate people who can still understand the original computer programming) — but all to no avail. This week, National Aeronautics and Space Administration officials decided that even Viking's version of a full-scale effort has reached the point of diminishing returns.

"Basically," says Geoffrey Briggs, deputy director of the agency's Earth and Planetary Exploration Division, "we are abandoning all efforts other than the very last and simplest things we can do." The lander's computer is programmed to start transmitting automatically if it hasn't heard from JPL in nine weeks (the last attempt to signal the craft was on Feb. 25), and tracking stations will be listening in early May, but hope is dim. Commands will also be sent to try to reconfigure components in the Viking transmitter, in case it is alive but mute. "After that," says Briggs, "one just says that we simply can't afford to make the kind of heroic effort of bringing extra people in and so on — taking them away from their jobs — to pursue something which appears to be futile." Viking veterans have a few other "low-level fixes" in mind, and some may be tried, but they can't go on forever. —*J. Eberhart*

Polio still problem in developing world

Although polio has been largely eliminated from developed countries, it is still a problem in developing ones where fewer than one-fifth of children are vaccinated against polio, reported scientists at an International Symposium on Poliomyelitis Control held recently in Washington, D.C. Still, progress is being made toward getting more children in these countries vaccinated against polio, the scientist concurred.

For instance, as Albert B. Sabin, developer of the live polio virus vaccine and currently visiting professor at Georgetown University School of Medicine in Washing-

ton, D.C., pointed out, massive polio vaccination campaigns have been successful in Brazil, Mexico and Cuba because non-medical personnel have aided medical professionals in vaccinating children.

The oral live polio virus vaccine rather than its alternative, an injectable killed polio virus vaccine, is mostly used in developing countries because of cost, effectiveness, ease of administration and some other factors. Yet the live vaccine has had only limited success in reducing polio in the Gaza Strip of Israel, presumably because viruses in children's intestinal tracts were killing virus in the vaccine. So children in the area, reported Eli E. Lasch of the Department of Public Health in Gaza, continued to get the live vaccine, but in conjunction with the killed one, in order that some virus material could bypass the intestinal tract and immunize those children whom the live vaccine failed to protect. It worked.

On the other hand, a more potent killed vaccine than the one now available may eventually benefit children in some developing countries that have had limited success with the live vaccine, predicted Alan R. Hinman, director of the Division of Immunization at the Centers for Disease Control in Atlanta. Such a vaccine has been developed by A.L. van Wezel and colleagues at the Riks Institute for Community Health in Bilthoven, Holland. It is made from virus growth in cells that are cultured on tiny beads. —*J.A. Treichel*

Clark's heart switched off

On March 23 the world's first recipient of an artificial heart — Barney Clark of Seattle — died. It was almost 16 weeks to the minute after he had received the heart on Dec. 2 and after the heart had beat in his chest nearly 13 million times.

Clark's death was attributed to complications related to the degenerative heart disease he had experienced before his natural heart was replaced by the artificial one, not to the artificial heart. In fact, the artificial heart was switched off only after his brain and other organs had failed, and at autopsy the artificial heart was found to be in good condition. Only one of Clark's numerous medical setbacks after he had gotten the artificial heart — when a valve in the heart broke and had to be replaced (SN: 12/18 & 25/82, p. 388) — was due to the heart itself.

As people throughout the world, including President Reagan, hailed Clark as a medical pioneer and highly courageous individual, doctors at the University of Utah Medical School in Salt Lake City, who had given Clark his artificial heart, said another artificial heart implant within the next few months was possible. But this time, they said, they hoped to put an artificial heart into a patient not so desperately ill as Clark had been, so that the patient would have a better chance of survival. □

In a capsule: Human antibody production

A technique for growing cells in porous, small spheres has boosted the quantities of human monoclonal antibodies that can be produced. This achievement could make it practical to use human monoclonal antibodies in the treatment of human diseases, according to the Damon Corp. of Needham Heights, Mass.

Antibody against a protein made by human pancreatic cells was produced in work at Damon Biotech, a subsidiary of Damon Corp. The human cell line making the antibody had been developed jointly by scientists at Joslin Diabetes Center in Boston and at the Wistar Institute in Philadelphia. They expect the antibody to be useful in diabetes diagnosis by detecting damage to pancreatic cells.

Using the encapsulation technique, scientists were able to produce 30 to 100 times the concentration of the antibody possible with conventional tissue culture methods. They had been unable to grow the antibody-making cells (called hybridomas) in the abdominal cavity of mice — the other conventional means of making monoclonal antibodies. Damon has also used the encapsulation technique for growing a variety of hybridomas, including one making antibody used in interferon purification (SN: 1/9/82, p. 24).

Although human monoclonal antibodies have been difficult to produce, they are expected to have important clinical advantages over more easily obtained mouse antibodies. Clinicians are concerned that mouse antibodies may provoke immune system attack and allergic reactions in patients. Animal monoclonal antibodies have been used to treat immune deficiencies (SN: 10/16/82, p. 244) and some cancers (SN: 8/22/81, p. 117), as well as to aid in kidney transplantation. "This new generation of human monoclonal antibodies may considerably expand the range of useful applications," says Allan P. Jarvis of Damon Biotech.

In the encapsulation procedure, which allows rapid cell growth and simplified purification, antibody-producing cells are mixed with a liquid containing a seaweed-derived gelling agent. The liquid is pumped through a small hole so it forms spherical droplets, which fall into a chemical solution where each drop gels into a solid sphere. For the human hybridomas, each sphere is 0.5 millimeter in diameter. The spheres are covered with a porous membrane of polymer, and the gel is then liquefied. The gelling agent flows out the pores, leaving the hybridoma cells and the antibodies they produce. After a period of cell growth, the capsules are broken open and the antibody is purified. A test-tube-sized container would give about 2 milligrams of antibody, the Damon scientists say. —*J.A. Miller*