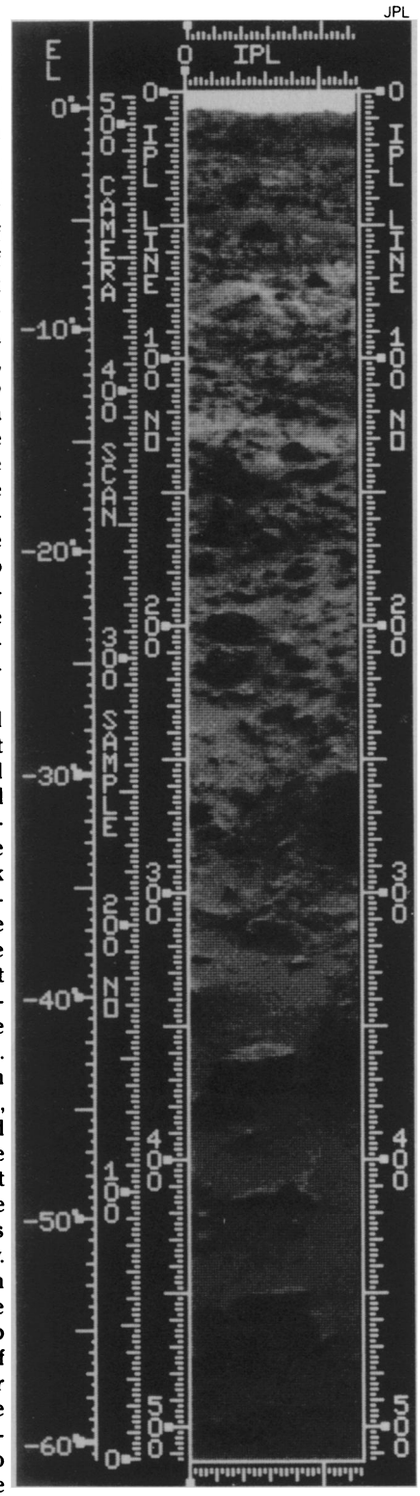
SCIENCE NEWS of the week
Viking's Odyssey: End of the Ending

A thin white line extended across the black monitor screen, as flat as the electrocardiogram of a corpse. On the evening of Jan. 21, as the appointed time drew near, a group of people in a room of building 230 at Jet Propulsion Laboratory in Pasadena stared at the screen, willing the line to suddenly develop a sharp vertical spike that would signal the return to life. There was little hope, however, and when the line failed even to shudder as the moments passed, the emotions of the onlookers did not include surprise.

The spike would have signaled that the National Aeronautics and Space Administration's Deep Space Tracking Network had picked up a message from Mars — the first report from the Viking 1 landing craft since it fell mysteriously silent more than five months ago. Reaching the Martian surface on July 20, 1976, it was designed to operate for three months but kept working for more than six years. Its message of last Nov. 13 appeared on schedule, complete with a typically sharp photo taken as part of a series to monitor the scene over time under identical lighting conditions to seek changes in the ground and atmosphere. By then under the automatic control of its own computer, it was programmed to operate through 1994. But its next message, expected on Nov. 19, never came. With no incoming data to aid in a diagnosis, engineers at JPL and elsewhere struggled to understand the problem and transmitted numerous attempts at remedial computer commands, but to no avail. An already programmed instruction for the craft to start sending on its own if it did not hear from earth for nine consecutive weeks produced nothing when that message was due on May 5, nor did two final efforts to trigger a response by ordering the craft to reconfigure components of its transmitter.



Viking's last photo of Mars was taken by Lander 1 on Nov. 5, 1982, after 2,238 Martian days (2,299 earth days) on the surface, and radioed to earth on Nov. 13. The dark-looking ground and diffuse-edged shadows suggest that a dust storm first spotted weeks earlier was still underway.

The obvious things had all been tried, and several less so (members of the now-tiny Viking team once sought out the computer in the Viking lander at the National Air and Space Museum for comparison tests), but VL-1 on Mars stood mute, after the expenditure of five months of time, effort and money. "It will be my recommendation," said project manager George Gianopulos of JPL, drafting a status report on the May 21 events for NASA headquarters, "that further attempts be terminated, and that the Viking Lander Monitor Mission be declared ended."

The other Viking lander and the two orbiters had already stopped working. The vast project, begun a decade and a half ago, has wound down. "A piece of history," says Geoffrey Briggs of NASA, "is now over."

—*J. Eberhart*

Hormone aberration-anorexia link found

Most of the symptoms of anorexia nervosa are by now well known: the obsession with thinness, the tendency to exercise excessively, amenorrhea. But there are other less well known symptoms, including urinary defects: anorectics seem to urinate too much, and even when extremely dehydrated they seem unable to hold normal amounts of water in their kidneys. Government scientists have now linked this symptom to abnormal hormonal activity — an abnormality that may help to explain the mental aberration associated with self-starvation.

According to psychiatrist Philip W. Gold and his colleagues at the National Institute of Mental Health in Bethesda, Md., anorectics appear to have a defect in the so-called "osmoreceptor" in the brain's hypothalamus — the receptor that signals the pituitary gland to secrete the anti-diuretic hormone called vasopressin. Normally when a person eats a lot of food that is heavily laced with salt, the high level of sodium in the body fluid causes the osmoregulatory cells to shrink, which in turn triggers a rise in vasopressin; vasopressin tells the kidney to hold on to water, so that the concentrated sodium can be diluted and excreted. In anorectics this regulatory mechanism appears to have gone awry.

As reported in the May 12 NEW ENGLAND JOURNAL OF MEDICINE, Gold gave heavy doses of a salty solution to normals and anorectics (while extremely underweight, during and following recovery). While the normal subjects (and depressed and schizophrenic patients as well) responded to the saline solution with a direct increase of vasopressin in their blood, the anorectic patients did not: they had either a deficient vasopressin response or, more commonly, a completely random response.

Gold notes that the anorectics also tended to have abnormal levels of vasopressin in the cerebrospinal fluid; but instead of being random, the vasopressin in the CSF appeared to be high. Vasopressin has been shown to improve memory in laboratory animals by strengthening the coding of information, and the excess vasopressin in the CSF (which bathes the brain cells) could explain the abnormally strong coding of certain thoughts in anorectics — notably the persistent thoughts about weight.

The abnormal vasopressin secretion disappeared as the anorectic patients recovered — but very slowly, Gold says. What may happen, he speculates, is that anorectics become preoccupied with slenderness for psychological reasons, but as they lose weight they trigger hormonal abnormalities. These abnormalities