of leaks that had plagued it for months, giving off the final few millipounds of gas that helped flight controllers maintain its orientation in space. With no way to keep its antennas pointed at the earth, the craft was effectively useless, and an on-board command was automatically triggered to silence its transmitter forever. (Its final weeks had been conducted in true Viking spirit: Knowing that the control gas was almost gone, Viking officials at Jet Propulsion Laboratory in Pasadena had hoped to acquire either some additional mid-latitude photo-mapping or a few more polar passes to provide water-vapor data. The spacecraft provided both.)

The other orbiter and both landers, meanwhile, are continuing to function. Orbiter 1 will now have to relay data to earth from both landers, probably "servicing" each landing craft about once a month. Present plans call for operating the orbiter through late February 1979, which will allow radio-propagation studies to run through the next solar conjunction.

Lander 1 may continue to operate for as long as two years, says mission director Kermit Watkins, since it has the capability of sending at least low-rate data directly to earth without the need for the orbiter relay. (Even after two years, the craft's location on the Martian surface continues to be refined. Merton Davies of the Rand Corp. says that matching of terrain features in pictures from the lander with high-resolution views from orbit [SN: 9/24/77, p. 199] now show the craft to be at longitude 47.968°W—about 8.2 kilometers west of its previously calculated site.)

Lander 2 will probably be the first of the remaining craft to be shut down, assuming that malfunctions do not change the sequence. It no longer has the capability of transmitting directly to earth, so it would be rendered effectively mute when the surviving orbiter is turned off, but the end will in fact come sooner—in early December—when the automatic data-gathering program now stored in its computer runs out. Meanwhile, the data continue to yield new results: It was lander 2, halfway to the Martian north pole, that photographed frost on the planet's surface (SN: 10/8/77, p. 228), and photos taken since the frost's disappearance look distinctly different from their "pre-frost" counterparts. Perhaps, suggests Ken Jones of JPL, the frost brought in dust particles that were then left behind, adding to Mars's many already known ways of modifying its surface complexion. □

Blood types deciphered

For fifty years, human blood has been classified as type M, type N and type MN, although nobody knew what the actual differences were. A rabbit's immune system makes the distinction—based on a human blood sample's reaction to rabbit antibody known to combine with M or N blood. Now several research groups have discovered the exact difference between M- and N-type blood. It is a variation in just two amino acids in a major membrane component of red blood cells. Furthermore, the rabbit antibody appears to react with both the sugar and amino acid subunits of those molecules, Elwira Lisowska of the Polish Academy of Sciences told a conference on Complex Carbohydrates in Biological Recognition at the National Institutes of Health.

The MN blood groups were discovered seven years after discovery of the better-known ABH (or ABO) blood types. Both groupings provided early examples of genetic codominance: A person with one M and one N gene, or one A and one B gene, expresses both genes. Different sugars on the key molecule of the ABH system are responsible for the antibody reaction. But the critical distinction between M- and N-type blood turned out to be a subtle difference in amino acid composition.

A few years ago amino acid sequences of the protein portion of MN-specifying molecules (called glycophoran A) revealed a mixture of two amino acids at each of two sites. Recent analyses showed amino acids serine and glycine at positions 1 and 5 in the M glycophoran A and leucine and glutamic acid at those positions in the N molecule. The blood of MN donors contained a mixture of those two types of glycophoran A, just as genetic codominance would predict. Lisowska, H. Furthmayr and V. T. Marchesi of Yale University, Olga Blumenfeld of Albert Einstein School of Medicine and W. Dahr and G. Uhlenbruck of the University of Cologne have all reported similar results.

A major surprise in this research is that although the amino acids seem to specify which antibody will react, the antibody reaction also requires carbohydrate close to the critical peptide sequence. Once researchers remove the sugar groups chemically, the antibody can no longer recognize the molecule. In other cases biologists have found either the sugars or the polypeptide, but not both, essential to an antibody's recognition. □

Stopping infection before it starts

Experiments show that flushing the bladders of mice with the sugar methyl alpha-D-mannoside prevents urinary tract infections by *Escherichia coli,* Nathan Sharon of the Weizmann Institute in Israel told the carbohydrate conference. This use of sugar may become an effective prophylactic procedure for persons prone to infection, such as catheterized patients, newborn infants in developing countries and burn victims.

The novel approach to blocking infections has arisen from investigations of bacterial attachment to cell surfaces. Adherence is a prerequisite of bacterial colonization and infection. The idea that surface sugars are the binding sites for bacteria has been hidden in the scientific literature since the 1950s, Sharon says. That role for sugars was rediscovered in 1977, following evidence that in many cases cell surface sugars play a central role in biological recognition.

Sharon and colleagues David Mirelman and Itzhak Ofek have identified both parts of the attachment mechanism. The cell surface site is a sugar, mannose or a related structure, present on nearly all cell membranes. Special proteins capable of grabbing onto mannose have been detected on *E. coli* and some other infectious bacteria. The investigators have isolated the grabbing protein, which appears to sit on the bacteria's filamentous appendages (pili).

Sharon and colleagues find they can interfere with the binding of a bacterium to a cell by flooding the cell surface with a solution containing mannose or certain mannose derivatives. Furthermore, mannose-containing solutions can remove bacteria already attached to cells. In those experiments, the bacterial protein attaches to the sugars in solution, instead of to the sugars on the cell surface.

Research in other laboratories indicates that different sugars may play a role in the binding of other pathogenic agents. Perhaps a battery of simple, nontoxic chemicals will soon prevent bacterial adherence to cells and ensure that the pathogens are sneezed, coughed or flushed out of the body. □

New education agency

A separate, Cabinet-level Department of Education moved one step closer to reality last week when the Senate Governmental Affairs Committee voted unanimously to create it. The Senate bill would engender the new agency with an initial budget of $18 billion and a staff of 24,000. The Department of Health, Education and Welfare would be stripped of most, but not all, of its education programs and responsibilities; several other agencies also stand to lose oversight and control of substantial education-related programs in the reorganizational shuffle. The House has begun hearings on a reciprocal bill. □