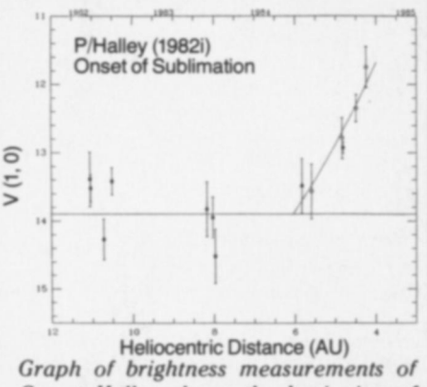
Watching Comet Halley come to life

For most of a comet's lifetime, it is just an inert ball of ice, dust and perhaps larger chunks of rock. Only when it nears the sun does the growing heat trigger the formation of the familiar, fuzzy "coma" and tail that make it stand out from other objects in the sky. Comet Halley is no exception, and when astronomers awaiting its 1910 "apparition" first saw it coming, about eight months earlier in 1909, its coma was already developing.

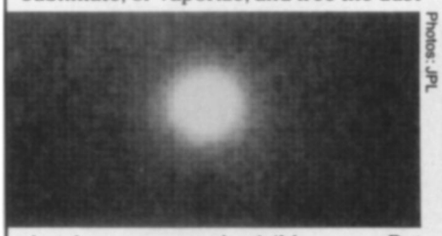
Now Halley is on its way again, due to make its closest pass to the sun next Feb. 9, but thanks in part to today's greatly improved instrumentation, it was "recovered" this time with more than three years to go, in October of 1982 (SN: 10/30/82, p. 277). An international fleet of spacecraft are on their way to study the comet when it gets near, but a unique advantage of the long period of ground-based observations has been the chance to study the coma as it begins to form — as the comet's icy nucleus first "comes to life."

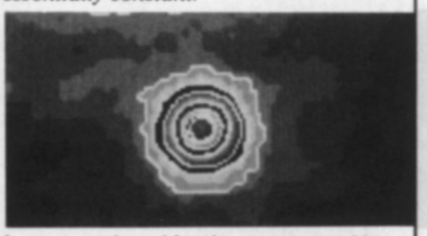
It is a difficult change to identify — the time at which the ice actually starts to sublimate, or vaporize, and free the dust that forms a comet's visible coma. But Susan Wyckoff, Mark Wagner and colleagues from Arizona State University in Tempe and the Institute of Astrophysics in Paris have now reported in the July 12 NATURE what they call, "to our knowledge... the first observation of the onset of sublimation in any comet."



Wyckoff et al./Nature

Graph of brightness measurements of Comet Halley shows the beginning of continuous sublimation of the comet's nucleus due to the heat of the sun — the first time, according to researchers, that the onset of sublimation has been observed in any comet. Observed magnitudes have been converted (V[1,0]) to correct for the comet's decreasing distance from the sun, so that if the nucleus were inactive — not forming a growing "coma" of dust and gas — the converted brightness measurements would remain essentially constant.





Photos: JPL

Changes in the apparent brightness of a comet's nucleus need not be due only to the formation of the coma, whose growth gives the nucleus a larger reflective surface. Another factor can be the rotation of the nucleus, exposing variously brighter and darker material to the sun.

A more exotic contributor, suggest Asoka Mendis, Harry Houpis and other researchers from the University of California at San Diego, could be the raising of dust from the surface of the nucleus by electrostatic charges due to the solar wind. One member of the UCSD group, for example, has found a correlation between some of Halley's brightenings and the position of coronal holes on the sun — possibly acting as openings to release high-speed solar-wind streams.

Whatever is freeing the dust — be it mere sublimation of the ice that traps it, electrostatic "levitation," or sudden bursts produced by the exposure of ices whose sublimation pressure is lower than that of water ice — the expansion of the area covered by dust provides a conspicuous signature.

The observations reported by Wyckoff's group were made last Nov. 26, Feb. 17, March 24 and April 2, using the Multiple Mirror Telescope near Tucson, and combined with other researchers' observations. The result (to which more data are being added) indicates that the dust coma began to grow sometime between early 1984 and 1985.

Other sensors, however, offer less clear clues to the subtle timing of the coma's birth. The photo shown (left), made last Dec. 30-31 with the 3.6-meter Canada-France-Hawaii Telescope on Mauna Kea, shows little if any indication of a developing coma, even when computer-divided into discrete brightness contours (right). These images, however, produced by Bruce Goldberg of Jet Propulsion Laboratory in Pasadena, Calif., and colleagues, will be part of a series documenting the comet's approach through standardized filters being made available to Halley-watchers at many different observatories.

— J. Eberhart

Something shared: Viruses, nerves

Certain viral encounters can turn a relatively innocuous situation into a potentially serious one. A measles virus infection, for example, can progress into encephalomyelitis, an acute inflammation of the brain and spinal cord. And the 1976 mass swine flu immunizations resulted in some of the recipients developing Guillain-Barré syndrome, an inflammation of the nerves that can sometimes lead to death.

University of Washington researchers offer a hypothesis to explain the relationship: similarities between viral and nervous system proteins turn an appropriate immune system response to a microbe into an attack on the nervous system. In the July 19 SCIENCE, the Seattle researchers detail their look at the order of amino acids — the basic components of proteins — in viruses and in myelin, which comprises the outer covering of nerve fibers.

Using a computer to compare the various sequences, they found similarities between sections of measles, Epstein-Barr, influenza A and B, and upper respiratory tract viruses and two myelin proteins that had previously been found capable of inducing neurological disease in laboratory animals.

Diane E. Griffin, a member of a Johns Hopkins University research team investigating measles and the immune system, comments, "It's interesting and it may be a clue, but I wonder if you wouldn't find similarities to other non-related proteins." Previous work at Hopkins, in Baltimore, has linked the encephalitis that sometimes follows measles to a more generalized immune disruption. The two possibilities are "not mutually exclusive, but neither one has given us the total answer," she says.

Based on their findings, the Washington group suggests that the protein coat of the virus or vaccine prompts a "specific sensitization" against myelin, which explains why only the nervous system is attacked.

The association would also cover the change in the rate of neurological complications from certain vaccines from year to year, says Ellsworth C. Alvord Jr., one of the researchers. Spontaneous mutations of the viruses can increase or decrease the protein similarities; identifying the culprit proteins, he notes, will enable manufacturers to eliminate them from their products.

Since most of the viral protein structures have yet to be determined, a lot of comparison remains to be done. Also on the agenda: injecting the suspect viral protein segments into animals to see if they do indeed spark neurological problems, and determining how similar the amino acid sequences have to be. *— J. Silberner*