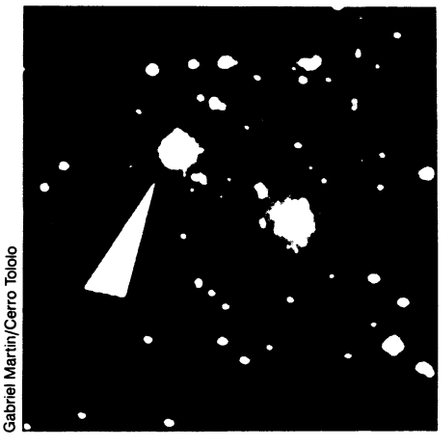
Supernova 1987A: Astronomers' Luck

"It's like Christmas," says astronomer Stanford Woosley of the University of California at Santa Cruz, speaking of the supernova in the Large Magellanic Cloud, officially named supernova 1987A (SN: 2/28/87, p.132).

The flow of information from this, the nearest supernova visible to us since 1604, continues to roll in, each piece of it a welcome gift to astronomers who specialize in these violent explosions of stars. Apparently the supernova exploded on Feb. 23. It was first noticed on Feb. 24 by Ian Shelton of the University of Toronto, who was working at the Carnegie Insitution of Washington's Las Campanas observatory in Chile, confirmed by nearby Cerro Tololo Interamerican Observatory and seen the same day by an amateur astronomer, Albert Jones of Nelson, New Zealand.



Gabriel Martin/Cerro Tololo

Arrow points to supernova 1987A.

By the first weekend after the discovery, the supernova's brightness in visible light had apparently peaked at a magnitude between 4 and 4.5, characteristic of a dimmer, type II supernova, rather than the type I originally predicted. On the weekend also came a report of bursts of neutrinos from the supernova. And on March 2, Brian Marsden of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., who is director of the Central Bureau for Astronomical Telegrams, told SCIENCE NEWS that astronomers observing with the International Ultraviolet Explorer (IUE) satellite had evidence "just today" indicating that the star that exploded was *not* the one most observers had thought it was — the blue giant star Sanduleak -69°202 — but a close companion to it.

Nevertheless, according to Woosley, if the supernova *was* the blue giant star, the timing of the neutrino bursts, which Carlo Castagnoli of the Istituto di Cosmogeofisica in Turin, Italy, reported from the neutrino observatory under Mt. Blanc, fits well into a theory of supernovas that Woosley and some colleagues have been developing for years.

In this model a supernova starts with the collapse of the core of a star. This collapse generates a shock wave that moves through the outer layers of the star, and when the shock reaches the surface, the explosion of bright light and expulsion of matter begin. Neutrinos, however, come from the core collapse, and the difference between their arrival time and that of the first bright light should reveal the time it took for the shock wave to traverse the star.

The neutrino bursts were recorded at Mt. Blanc at 2:58 a.m., universal time, on Feb. 23 — or, in decimal fractions of a day, as astronomers like to time things, on Feb. 23.124. It happens that on Feb. 23.442, R.H. McNaught of the Siding Spring Observatory in Australia took a picture of the blue giant, which would then have been just at the point of explosion. The difference between those two times is about 30,000 seconds, too quick for the shock wave to traverse a red giant — the sort of star expected to produce a type II supernova — but right for a blue giant.

However, the IUE data of March 2 were showing a spectrum characteristic of a B3 blue giant, which seems to indicate that that star was still there, and that the supernova would then have to be some close companion to it. If that's so, it changes everything, Woosley says.

Other avenues of observation remain to be heard from. This supernova should emit powerful gamma rays, and they should last for years, says Woosley. Bursts of gravitational waves might have come from the supernova, but so far there is no report of such an observation.

— D. E. Thomsen

Drug resistance: Malaria-cancer similarity?

Researchers at the Walter Reed Army Institute of Research in Washington, D.C., report experimental evidence that malaria-causing parasites may be using the same defense against antimalaria drugs as cancer cells do against certain anticancer drugs.

If both cancer cells and malaria parasites use the same protective device, researchers might be able to apply what they know about the cancer system to the problem of malaria drug resistance, according to the investigators.

Resistance to antimalaria drugs is a growing problem. Nearly all malaria infections in Indochina are now caused by parasites resistant to the most effective and least dangerous antimalaria agent, chloroquine. Cases that fail to respond to chloroquine have also been reported in South America and Africa.

After a meeting on cancer drug resistance recently at the National Institutes of Health, Samuel K. Martin of Walter Reed proposed that *Plasmodium falciparum*, which causes most of the 2 million to 3 million malaria deaths a year, sometimes uses a mechanism similar to that of tumor cells. If so, he theorizes, it could be thwarted in the same way resistance in cultured cancer cells can be reversed.

In the Feb. 20 SCIENCE, Martin, Ayo M. J. Oduola and Wilbur Milhous report they exposed drug-resistant *P. falciparum* to verapamil, one of several drugs that can prevent cancer cells from ridding themselves of chemotherapy. The verapamil made the malaria parasites sensitive to chloroquine.

Some cancer cells resist chemotherapy with the help of a cell-membrane protein that grabs and ejects toxic drugs that have gotten into the cell (SN: 1/3/87, p.12; 1/24/87, p.57). Verapamil is believed to inhibit this action. Donald Krogstad at Washington University in St. Louis is investigating whether the same protein used by cancer cells is present and functional in the parasites.

According to U.S. cancer researchers, testing of verapamil in cancer patients has begun in Japan, but there have evidently been problems with side effects. The National Cancer Institute plans to try drug-resistance-reversing agents in cancer patients soon, and one researcher involved says they will try several different reversing drugs at low doses to minimize side effects. Human trials of agents that reverse malaria drug resistance will have to wait until that system is better understood, says Milhous. In the meantime, the Walter Reed researchers are watching what happens in the cancer field, and searching for agents with fewer side effects than verapamil.

Michael M. Gottesman of the National Cancer Institute, who with Ira Pastan and other colleagues is working on cancer drug resistance, says the malaria findings "are interesting and may turn out to be very important." They may, he says, boost the understanding of the general drug resistance process and speed the development of new drugs for both cancer and malaria. *— J. Silberner*