Bursts of Gamma Rays Baffle Astronomers

One burst may be linked with a supernova remnant, but that identification has not helped in unraveling the causes of bursts

On 5 March 1979, detectors on nine satellites scattered around the inner solar system observed an extraordinarily energetic and impulsive flash of gamma rays from space. By the end of April, researchers suspected that the source of the burst was in the remains of an exploded star in another galaxy. With this first identification of a source for a gamma-ray burst, astronomers were eager to figure out what high-energy processes produce such bursts. While a new theory has been proposed, overall the mystery of gamma-ray bursts has deepened. Now many experts doubt that the March event was associated with the supernova remnant. Moreover, they suspect that it may not be a typical burst, rather "an extremely interesting once-in-a-decade or once-in-a-century phenomenon."

The location of a burst source is calculated in much the same way that seismologists determine the location of an earthquake—from the time the signal arrives at each detector in the network. According to Doyle Evans of Los Alamos Scientific Laboratory and collaborators in Maryland, France, and the Soviet Union, the March burst came from the direction of N49, a supernova remnant in the Large Magellanic Cloud galaxy. Astronomers were elated that after a decade of puzzling about the bursts, they finally had a candidate for a source. But "we are very uneasy about the identification because of the great distance" to N49, says Ray Klebesadel of Los Alamos.

Many astronomers are leery of the identification for two reasons. They wonder why no brighter bursts have been observed from sources in our galaxy, if such a bright flash can come from an outside source. Moreover, if the burst came from N49, then a very large amount of energy—several billion times the sun's output—was emitted from a very small volume. The size of the emitting region is estimated from the length of time it took the flash to reach its peak brightness. According to Thomas Cline and Bonnard Teegarden of Goddard Space Flight Center in Greenbelt, Maryland, the full power of the burst turned on in less than 1 millisecond. Thus the source was small enough for light to cross it in that amount of time. "The energy density at the source was staggering," says Teegarden. (Although quasars are roughly a million times more powerful than the March burst, their energy is thought to be produced in a volume more than a trillion times larger.)

Many researchers think that the coincidence of the burst with the supernova remnant is no more than that. If the source were closer, it would need to be much less energetic to produce the same signature on the satellite records.

"I think that it is interesting that they [astronomers] have been waiting 10 years for an identification, and now that they have one, they are not willing to believe it," says Reuven Ramaty of Goddard. Nearly a decade ago Los Alamos researchers accidentally discovered gamma-ray bursts while looking for evidence of Soviet nuclear tests in data from Vela surveillance satellites. About 100 bursts, roughly one per month, have been detected to date. The sources are thought to be in our galaxy on the basis of their distribution in the sky. But now the only burst identified with a source appears to be linked with an object in another galaxy. While many experts would like to find fault with the identification, Cline points out that it "is as respectable as any other made in astronomy." N49 contains the small patch of sky assigned to the burst.

Searches with optical and x-ray telescopes to find closer and therefore more palatable candidates for the source have not panned out. According to David Helfand of Columbia University, the x-ray telescope on the second High Energy Astronomy Observatory ("Einstein") (see *Science*, 29 June 1979, p. 1399) found no pointlike x-ray sources in the direction of N49. Furthermore, of the stars in the area, only two hot ones are slightly suspect, explains Gerald Fishman of Marshall Space Flight Center in Alabama. But those stars are just as far away as N49, and therefore no more acceptable as sources.

As a theorist willing to consider that the burst might have come from as far away as the Large Magellanic Cloud, whether from the supernova remnant or from an unusual star, Ramaty felt spurred to devise a model to explain the phenomenal energy and impulsiveness of the burst. He speculates that the energy seen in the pulse could have come indirectly from a powerful nuclear detonation on the surface of an extremely hot neutron star. Such a detonation would release so many high energy photons (gamma rays) that they would collide with each other and produce pairs of electrons and their antimatter counterparts—positrons. In their brief lifetime, the energetic particles would slow down, and the energy lost from their motion would be radiated away as lower energy gamma rays. Finally, the electrons and positrons would collide and annihilate each other. Their mass would be converted into gamma radiation of a distinctive energy or wavelength, à la Einstein's $E = mc^2$.

The key to Ramaty's model is that this process goes on in a layer only 100 micrometers thick in the atmosphere of the neutron star. Closer to the surface of the star, the radiation from the nuclear explosion is too dense for any to escape. Ramaty explains that "the last thing the high-energy gamma rays do before they escape is create the thin layer," which essentially annihilates and regenerates itself 10,000 times in one-billionth of a second. Not only does Ramaty's model appear to account for the energy and abruptness of the burst, it also explains the gamma-ray spectrum of the event.

Last month Cline speculated that the March event could be a rare type of burst associated specifically with supernova remnants. He supposes that such bursts are energetic hiccups that can occur only once in the life of a remnant. Since stellar explosions occur only once per 30 years in a galaxy, he says "it is reasonable that we have seen only one [like the 5 March event] in 10 years." Cline's argument justifies why no similar but brighter bursts have been seen yet from sources within our galaxy.

Other astronomers agree that the March flash was unusual, but they remain unconvinced that the source must be in N49. As Walter Lewin of Massachusetts Institute of Technology says,

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"the source is anybody's guess." He thinks it is slightly more probable that the source is in our galaxy than in the Large Magellanic Cloud. "Although I hope it is in N49, because then the energetics is so boggling that we have something really exciting to worry about."

While astronomers have found a source for the extraordinary 5 March event, no candidates have been found for any of the 100 more "normal" bursts. Typical bursts are less than 1 percent as bright as the March burst, and shine erratically for several seconds instead of flashing nearly instantaneously. Only one typical burst has been located as precisely as the March event. From its spectrum, Ramaty surmises that its source is well within our galaxy—probably closer than 200 light-years to the earth. Yet nothing can be seen in the direction of that burst, according to Fishman. Einstein's x-ray telescope is scheduled to look at the area this spring.

With no visible source for one well-located burst and the other seemingly associated with an object in another galaxy, astronomers still have more questions than answers. Many experts now suspect that there might be two types of bursts—normal ones from sources within our galaxy, and extraordinarily rare, energetic, impulsive ones from supernova remnants or something else. All in all, gamma-ray bursts are not likely to be understood until more are located and improved spectra are obtained. While more locations should be determined soon with data from the present network of satellites, no better spectra will be forthcoming until the middle 1980's, when the problem-plagued Space Shuttle is scheduled to launch the newly proposed Gamma-Ray Observatory.

—BEVERLY KARPLUS HARTLINE

AMIS Negative on Aspirin and Heart Attacks

A large clinical trial shows that aspirin does not prevent cardiac deaths in patients who have already had a heart attack. But questions remain.

In 1975, the National Heart, Lung, and Blood Institute (NHLBI) began recruiting participants for a 3-year clinical trial to determine whether aspirin can prevent heart attacks. The results* are finally in. The answer, it seems, is that aspirin does not prevent additional heart attacks in men and women who have already had at least one. Moreover, trial participants who took aspirin experienced significantly more side effects, including bleeding from the stomach and intestines, ulcerlike pains, and stomach inflammation, than those on the placebo. "On the basis of these findings," says Robert I. Levy, director of the NHLBI, "the National Heart, Lung, and Blood Institute would advise physicians not to give aspirin on a sustained basis to heart attack patients as a means of preventing another myocardial infarction [heart attack]."

The trial in question, called the Aspirin Myocardial Infarction Study (AMIS), was the outgrowth of earlier studies that had suggested that aspirin might be of some benefit as a prophylactic against heart attacks. The results were not conclusive, however, and other studies had given negative results.

The possibility that an inexpensive, widely available drug, such as aspirin, might reduce the toll from the nation's number one killer was very appealing. The NHLBI thus decided to undertake, at a total cost of $17 million, a controlled clinical trial in the hope of settling the issue once and for all. That hope has not been realized, however, despite the apparently unequivocal nature of the AMIS results. Information from some other recent studies has raised questions about the AMIS design, leaving open the possibility that aspirin may still prove to be of benefit to heart patients.

The AMIS trial is the largest test of aspirin as a heart attack preventive ever done. It included 4021 men and 503 women, who were followed for 3 years. During this time the aspirin group took 1 gram of the drug per day (about the amount in three standard aspirin tablets) and the controls received placebos. Both groups were instructed not to take any aspirin on their own and were given a substitute pain-killer (acetaminophen) for minor aches or pains. Compliance with the drug regimens, which was carefully monitored, was good.

According to the AMIS results, aspirin did not reduce mortality in heart attack patients. In fact, the total mortality in the aspirin group (10.8 percent) was somewhat higher than that in the control group (9.7 percent), with 8.7 percent of the aspirin group and 8.0 percent of the controls dying from heart attacks and related heart disease.

There were fewer nonfatal heart attacks in the patients receiving aspirin than in the controls. This decrease is difficult to reconcile with the slight increase in fatal heart attacks in the aspirin group. One possible explanation suggested in the AMIS report is that people who suffer a heart attack while taking aspirin are, for some unknown reason, more likely to die than those who are not taking the drug. Whatever the explanation, the mortality findings, together with the high incidence of gastrointestinal problems in the aspirin group, would militate against routine use of the drug for prevention of heart attacks.

"We were disappointed by the results," says Levy. "We thought we had a potential winner." The initial optimism came about both because of the encouraging results of the earlier clinical trials and because aspirin has long been recognized as an inhibitor of the clumping of the small blood cells called platelets. Platelet clumping is a necessary step in the formation of blood clots. Because researchers think that the abnormal formation of clots in the arteries carrying blood to the heart muscle is one of the causes of heart attacks, the possibility that aspirin, by inhibiting clot formation, might prevent heart attacks was the rationale for testing the drug in heart patients in the first place.

On the bright side, the AMIS results did indicate that patients taking aspirin suffered fewer strokes and transient ischemic attacks (brief dizzy spells caused by reduced blood flow to the brain) than the controls. The difference was not quite statistically significant but did tend to confirm the results of two earlier studies. Why aspirin would work in the brain but not the heart is unclear.

Even though the AMIS results would seem to lay to rest the idea that aspirin might decrease mortality from heart attacks, enough questions have been raised about the design of the trial to sug-

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