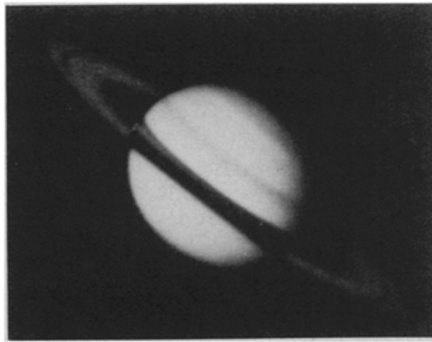


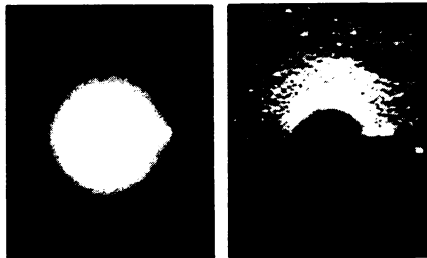
Voyager 1 approaching Saturn

Last September, the Pioneer 11 spacecraft's photos of Saturn, first close-ups of the planet ever taken, showed so few visible features in the atmosphere that they "scared the hell out of us," says Bradford Smith of the University of Arizona. Smith, however, is also the leader of the "imaging team" for the Voyager 1 spacecraft, due to pass by the planet on Nov. 12. Even with nearly 200 million kilometers to go, Voyager 1's photos are showing more pronounced banding around the planet, although Smith says it is too early to be sure whether this is due to changes in the clouds or merely a better camera system. Still, he adds, there are faint signs of possible structure within the bands.

The spacecraft will begin full-time observations of the planet on Aug. 22, providing data in part for the targeting and calibration of even more intensive measurements that begin Oct. 24, when Saturn will be too big to fit in one of the camera's narrow-angle frames. With 10 full days to go, even a 2-by-2-frame mosaic won't hold it. Meanwhile, Voyager's radio telescope has already determined the planet's 10 hour 39.9 minute rotational period, and charged particle sensors are seeking Saturn's effects on the space around it. Scientists are now working to decide where to aim the cameras to hunt for unknown or little-known satellites, what filters to use to best photograph cloud features over Titan, and other issues that must be resolved well before the flyby takes place. □



Voyager 1 photo of Saturn (above), taken June 24 from 187 million kilometers out, shows details as small as 3,500 km, already slightly exceeding earth-based resolution. The sun was barely above the ring plane when photo was taken, so rings appeared very dim; off-planet portions of the rings were brightened by computer, causing the discontinuity in apparent brightness where the rings cross the planet's disk. Earth-based photo (below left), taken Nov. 14, 1966, by W.A. Feibelman (now with NASA), enabled densitometer measurements indicating material outside main ring structure, extending to more than twice the then-known ring diameter. Recent computer processing of same image (below right — see July 11 SCIENCE) makes this wider, "E-ring" structure visible.



NASA

W.A. Feibelman, D.A. Kinglesmith III

antibodies to alpha-fetoprotein could keep amniotic fluid from inhibiting the binding of myasthenia antibodies to acetylcholine receptors. All these findings, they conclude, show that alpha-fetoprotein is the chemical in amniotic fluid, umbilical cord and maternal serum that inhibits the binding of myasthenia antibodies to acetylcholine receptors. They therefore suggest that alpha-fetoprotein might eventually provide a new treatment for myasthenia gravis.

In fact, the researchers speculate, the protein may eventually benefit patients with rheumatoid arthritis, lupus, multiple sclerosis and some other putative autoimmune diseases. The reason? These diseases, like myasthenia gravis, tend to disappear in latter pregnancy, suggesting that alpha-fetoprotein is combatting them. However, if the protein does inhibit these diseases, it is probably by altering autoimmune mechanisms rather than by binding antibodies to acetylcholine receptors. □

ESA comet-bound with or without NASA

With the National Aeronautics and Space Administration and various scientists around the country struggling to initiate some kind of U.S. spacecraft mission to comet Halley, the European Space Agency last week decided to go ahead and develop its own. NASA has been invited to participate, but, in a marked change from past NASA-ESA cooperative ventures, ESA this time is fully prepared to go it alone if the United States fails to come through.

The spacecraft, a modification of ESA's GEOS earth-orbiting research satellite, is to be launched between July 7 and 22, 1985, and to fly by the comet on March 13, 1986, a few weeks after the comet's closest encounter with the sun. The flyby will happen in a rush, at a relative velocity of 68 kilometers per second (244,800 km/hr), so all of the mission's observations will be made in a single four-hour period. The data will be radioed back to earth as they are collected, rather than stored on board for later playback (which would allow more intensive measurements), because of the possibility that dust close to the

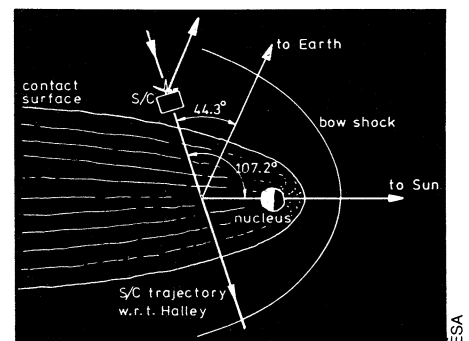
Myasthenia gravis: Another approach

Myasthenia gravis, a chronic disease that causes progressive weakness of the voluntary muscles, is no longer the mystery it once was. The cause appears to have been identified, and relatively successful treatments have been devised. Now there is a suggestion of a potentially more effective treatment.

Myasthenia gravis has been explained as an autoimmune disease in which the patient produces antibodies against the muscle cell receptors for acetylcholine, the nerve chemical that stimulates muscle cells. As a result, the cells cannot receive acetylcholine, and muscles fail to contract. Drugs that prolong acetylcholine's action and increase its ability to bind with muscle cell receptors have been used to treat the condition, but they do not always produce the desired effect. Research that may lead to a better treatment is reported in the JUNE PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES by Talma Brenner and colleagues at Hebrew University-Hadassah Medical School in Jerusalem.

Because myasthenia gravis often disappears during the latter stages of pregnancy, Brenner and associates suspected that something in pregnant women might be involved. Last year they reported that some chemical present in amniotic fluid, umbilical cord and maternal blood can inhibit the binding of myasthenia gravis antibodies to acetylcholine receptors. Since then they have worked to determine what that substance might be.

First they separated out various chemicals from amniotic fluid to see which of them might be able to inhibit, in the test-tube, the ability of myasthenia gravis antibodies to bind to acetylcholine receptors. They found that only amniotic fluid fractions containing alpha-fetoprotein had this ability. Then they put purified chemicals known to be present in human amniotic fluid in the presence of myasthenia antibodies and acetylcholine receptors. Only alpha-fetoprotein significantly inhibited the antibodies' binding to the receptors. Finally, they showed that



ESA flight planned through Halley's tail.

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