Planetology: Mercury and magnetism

Martians and Mercurians met on Monday, along with a few representatives of earth's moon. On Tuesday, the Venusians appeared, as did the asteroid and meteorite people. The Jovians had the next day to themselves, followed on Thursday by the Saturn, Uranus, Neptune and satellite delegations, leaving Friday for factions otherwise unheard from.

For the planet people it was the event of the year: the annual meeting of the American Astronomical Society's Division for Planetary Sciences. From as far away as Hawaii they came, to congregate at Columbia, Md., for the one occasion at which ranking U.S. experts on the entire solar system (except for the sun itself—that's another division) can compare results, propound and shoot down theories, and generally talk the shop of other worlds.

In 1969 when the division was established by a committee including such noted researchers as Lewis Branscomb, Gordon Pettengill and Carl Sagan, only two planets—Mars and Venus—and earth's moon had been visited by man-made spacecraft. Most studies were, and still are, done from earth. Even so, planetologists are fully aware of the unique value of a close look, and last year's meeting in California was a standing-room-only affair, thanks largely to hot-off-the-computer data on Venus and Mercury from Mariner 10 and on Jupiter from Pioneer 10 (as well as Skylab's observations of Comet Kohoutek). Interest in the planets has grown so rapidly, according to one attendee, that the present 260 members of the Division for Planetary Sciences outnumbers the entire AAS at the time the division was formed.

One of the most interested these days is Norman Ness of the Laboratory for Extraterrestrial Physics at the NASA Goddard Space Flight Center in Greenbelt, Md. In charge of the twin magnetometers aboard Mariner 10, he was amazed as were most of his colleagues when the spacecraft's first encounter with Mercury last March 29 revealed the "absolutely unanticipated" presence of a substantial magnetic field. The second encounter, on Sept. 24, was primarily for photography and was too far away for good magnetic field measurements. But there's one more chance: a third and final pass coming up on March 16—even closer than the first one. Although other instruments will be operating in addition to Ness' magnetometers, "this," says an official at the Jet Propulsion Laboratory in California, from which the spacecraft is being controlled, "is Norman's show."

"Mercury-3," as it is called, could be the last visit to the solar system's innermost planet in many years. So intriguing are the possibilities of this final flyby, and so high is the interest in the data already in hand from the first two, that the First International Colloquium on Mercury has been scheduled to meet at Pasadena in June.

Since Mariner 10's initial revelation, Ness has been at work making what he could out of his surprising data. His basic conclusion, and the most significant one for future studies of Mercury, is that the magnetic field is a real one, internal to the planet, rather than a vague interloper poured on from the outside by the solar wind. The most compelling evidence for this is that while both approaching and leaving the planet, Mariner 10 reported the presence of a clear "bow shock," like the wake around the bow of a boat, where the million-mile-an-hour flow of the solar wind was being pushed aside not by Mercury itself, but by its magnetic influence. Furthermore, says Ness, it seems likely that the field is caused by an "active dynamo mechanism"—electric currents moving around the planet within its metallic core. Previously it was assumed that such a dynamo requires a rapidly rotating planet, which Mercury is decidedly not.

The heavy metallic core, however, seems decidedly present. Mercury seems to be made of such lightweight, moon-like stuff on the outside that its large mass (confirmed by its bending effect on Mariner's trajectory) must signal a truly ponderous interior. Ness estimates, in fact, that the core may be as much as 70 percent of the radius of the planet and that 60 to 70 percent of its mass is iron—fine stuff for a dynamo.

The strength of the field seems to be about 350 gammas at the equator. The

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field is, not surprisingly, dipole shaped, like the doughnut-shaped field around a bar magnet. However, Ness says, it is either tilted by about 7 degrees from the axis of rotation of the planet (less than the field of either earth or Jupiter), or it is something more complicated than a simple dipole. The upcoming encounter with Mariner 10 ought to help determine that. Besides simply providing more "data points," the final flyby should occur barely 500 kilometers from the planet, about 200 kilometers closer than the one that revealed the field in the first place. This should put the probe in a region where the field is about twice as strong as it was where it was first measured, so that fine details should be more visible.

The biggest question for "Mercury-3" will be whether there is really a dynamo effect in Mercury, or whether instead the field is due to a large mass of permanently magnetized materials. If there is a dynamo, what makes the currents that run it—convection currents from internal heating or precession of the core's axis within the planet? If, as seems likely, there is such a core, how did it evolve?

There are many such questions at the gathering of the planet people. Norman Ness, however, may be closer than most of his colleagues to a few of his answers. □

Marine models will aid future research

"Boy, have we got models for you!" This statement, made last week by a marine biologist, sums up the feelings of many researchers in marine biology and predicts what they see as a changing emphasis in biomedical research. Researchers from several fields met last week in Washington at a marine biomedical conference sponsored by the National Institute of Environmental Health Sciences to discuss this changing emphasis, and agreed that marine animals will play an increasing role in environmental and biomedical research.

Many researchers have in recent years focused on complex mammalian systems in their study of disease mechanisms. They reason that the closer their research animals are to humans, the more meaningful will be the application of their results. But, says Stewart G. Wolf of the University of Texas at Galveston, a phylogenetically simpler organism might yield more information than a complex mammal. "We now know that understanding the body's regulation of chemicals is the key to the secret of understanding disease. The question has gone from what happens to how does it happen." We must consider, he says, "the possibility of looking at human disease by turning to phylogenetically earlier periods where systems are simpler, but the biochemical functions are similar. Our exploitation

of marine models has only just begun."

One such model, the sluglike mollusc *Tritonia*, is beginning to yield important information on the mechanisms involved in epilepsy. Marine biologist A. O. Dennis Willows of the University of Washington's Friday Harbor Laboratories has developed a hypothesis on brain neuron behavior during epileptic seizures based on his work with *Tritonia*.

Willows discovered about 10 years ago that *Tritonia* has easily accessible brain neurons more than a million times larger than the average mammalian neuron. Where it is difficult to insert one microelectrode into a mammalian neuron, Willows can insert four or five into a *Tritonia* neuron without killing the cell. His epilepsy studies grew from basic studies on brain function and behavior. He suspected *Tritonia* might be a good model for studying epilepsy and found that after administration of the drug pentylenetetrazol, the neurons exhibited epileptic-like behavior. Neurons that were previously inactive became active, neurons acting as individuals became synchronous and neurons that usually produced single impulses fired bursts of impulses.

Willows now hypothesizes that pentylenetetrazol produces a sodium conductance across the neuron membranes and this initiates nerve firing. He also suggests that where nerve axons touch, there are weak electrical interactions that "communicate" and initiate synchronous firing.

"For decades," Willows says, "epilepsy has been studied in mammals. Millions of dollars have been spent and the problem still remains." Researchers could have gotten further "in cellular terms" if they had used a neuron system where it is possible to control and measure the electrical and drug environment, he says. The nerve activity during epilepsy appears to be the same in *Tritonia* and in higher animals, Willows says, and researchers soon may be in a position to explain epilepsy and choose more appropriate anticonvulsants.

Other marine animals being used as models for biomedical and environmental research also were described. Japanese carp are being used in diabetes mellitus studies, Pacific salmon in arteriosclerosis studies, shark livers for various diseases including cirrhosis, lamprey and hagfish for basic endocrine studies and deep sea angler fish for transplantation studies. Tumors have been found in marine animals of all phyla and have been linked to oil spills and other chemical sources. Some think these tumors could be monitored and serve as "early warning devices" of carcinogenic water conditions. □

Protein synthesis decreases with age

No process is more fundamental to understanding the growth and functioning of the human body than protein synthesis, but measuring even the overall rate of this process is a tricky business. Individual biochemical mechanisms by which protein synthesis proceeds can be traced in the workings of body subsystems, but their efficiency may differ by as much as a factor of 100 from that of the whole-body average and their rates cannot be extrapolated. Now a team of five researchers in the Department of Nutrition and Food Science and Clinical Research Center of MIT believe they have found a way to measure the rate of whole-body protein synthesis—at least closely enough to study accurately how it changes during aging. They report their results in NATURE (Vol. 253, p. 192).

Not surprisingly, the rate decreases sharply during the first years of life, from 17.4 grams of protein synthesized per kilogram of body weight per day in the newborn, to 6.9 in older infants. The rate in young adults is 3.0, only one-sixth that of the newborn. In the elderly the rate drops to only 1.9.

To explain this decrease, the researchers recalculated the synthesis rates on the basis of energy expenditure for each age group, and found that the amount of protein synthesized for each calorie of energy expended is nearly constant, around 0.11 grams per calorie. They also found that the efficiency of protein synthesis is also nearly constant throughout life—one gram of dietary protein suffices as raw material for the synthesis of 4 to 5 grams of body protein in either youth or age. Thus the decrease in dietary protein needed to sustain bodily functions as one grows older (down from 3.2 grams protein per kilogram body weight per day as a newborn to 0.57 as an adult) appears to result almost entirely from the changing rate of whole-body protein synthesis, in turn a function of decreased energy needs.

In an analysis of this research in the same issue of NATURE (p. 157), J. C. Waterlow presents other, previously unpublished data, showing that much of the energy expenditure for protein synthesis in young rats goes into producing muscle growth. As growth slows, so does the rate of protein synthesis and the proportion of synthesized protein that goes into new tissue. The next problem, Waterlow concludes, is to use these new data on synthesis rates to better understand the means by which the rates are controlled. □

Stroke: Humoral connection

Autopsies show that around the ulcerated fatty deposits caking the arteries of patients with severe atherosclerosis are accumulations of platelets, the so-called "third corpuscle" of the blood stream. This discovery has led to the suggestion that such accumulations of platelets may contribute to shutting off the brain's blood supply in stroke and that some local humoral agent causes the platelets to aggregate. When arachidoric acid—a common fatty acid essential in nutrition—was found to cause platelet aggregation in vitro, University of Virginia Medical School neurologists Thomas W. Furlow Jr. and Norman H. Bass tried injecting a derivative of the acid, sodium arachidonate, into rats to see if it would cause them to suffer strokes.

The results, reported in Feb. 21 SCIENCE, show that all injected rats died of stroke within minutes and that their small cerebral blood vessels were later found to be clogged with platelet aggregations. Possibly complicating effects of clotting through formation of fibrin were suppressed through administration of an anticoagulent drug.

The researchers suggest that transient elevation of such humoral compounds as arachidonate may trigger human strokes, though the connection has yet to be proven. One way or the other, they conclude, this model promises to contribute to better understanding of stroke and might lead to preventative treatment. □