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. □