SCIENCE NEWS of the week

Polio: Scars from an Old Foe

Three decades after defeating a disease that threatened to rob them of movement or even of life, some survivors of polio are coming down with mysterious new symptoms. For the victors, who fought for months to regain the use of affected muscles, sudden weakness or pain in a limb that has been strong for years can prompt a special anxiety. But scientists who gathered in Warm Springs, Ga., recently to discuss the new syndrome have news they hope will ease the tension: No matter what causes the new symptoms, they progress slowly if at all, and are almost certainly not linked to a reactivation of the paralyzing virus of childhood.

For Nancy Frick, an advocate for the disabled in Hackensack, N.J., the news sparked cautious relief, and she is intent on getting the word out to fellow polio survivors and doctors who often have been as baffled by the symptoms as their patients. "A lot of people have been told that they have anything from postpartum depression to muscular dystrophy," Frick told SCIENCE NEWS. Perhaps most damaging of all, many of the patients have been led to believe they have amyotrophic lateral sclerosis or ALS, the swiftly fatal neuromuscular disease that killed baseball player Lou Gehrig. But while the cause or causes of the symptoms is still unclear, "several lines of evidence indicate that there is not a link to ALS," says Lauro Halstead of Baylor College of Medicine in Houston.

Several viable theories might explain the new syndrome, says Halstead, who has surveyed more than 200 cases in an effort to define and classify symptoms. It is too soon to be certain how many of the 300,000 U.S. polio survivors are affected, he says, though several preliminary surveys put the estimate between 20 and 25 percent. There are probably at least half a dozen subgroups within the affected population, Halstead speculates, each with a different cause — still undetermined — of the same symptoms.

Richard R. Owen, who directs the Sister Kenny Institute in Minneapolis, believes that the fatigue and weakness many patients describe can be traced to an increasingly sedentary lifestyle as they age. "Muscles sink down to the lowest level of challenge that they're put to," Owen says. But Halstead argues that many post-polio patients are leading strong and active lives when the sudden weakness hits. Another theory proposes that the weakness and pain stem from overloaded anterior horn cells, nerve cells of the spinal cord that control muscle movement. Many of these cells are destroyed during the original invasion of the polio virus. If some of the cells are damaged, but not destroyed, the theory goes, they too might be expected to fail after years of compensating for lost nerve cells.

David O. Wiechers of Ohio State University in Columbus has focused on the junction between nerve and muscle. When a peripheral nerve axon is severed, a nearby axon sprouts, much like a tree growing new roots. In this way, a single nerve cell that originally controlled 100 muscle fibers can, over months, spread its influence to 300 to 1,000 fibers, and compensate for nerve cells that have been killed. Wiechers has shown that in some post-polio patients, these "sprouts" begin to fail or drop off after several decades.

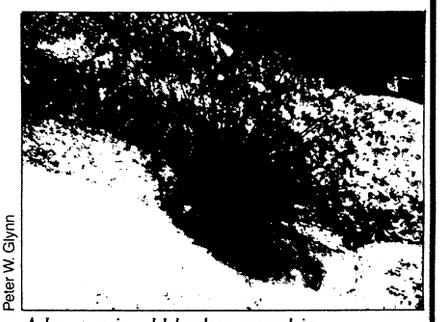
Marinos C. Dalakas and co-workers at the National Institutes of Health found slight immune system irregularities in 10 post-polio patients with new muscle weakness. In addition, in one patient, antibodies to polio virus were specifically elevated in the spinal fluid. The single finding has given rise to widespread, misplaced fear among polio survivors, Halstead says, that the virus can somehow lie dormant in the spinal cord, only to reemerge years later. In light of other evidence, he says, it is more likely that the patient was re-exposed to polio virus than that she carried a persistent infection. Dalakas agrees that while it is theoretically possible that persistent virus could cause new symptoms, no generalizations should be made from a single finding.

While there is presently no specific treatment or preventive for post-polio symptoms, Richard Bruno of Columbia-Presbyterian Medical Center in New York suggests that the "use it or lose it" exercise philosophy drilled into polio survivors 30 years ago may actually *contribute* to muscle weakening. Owen agrees that overexertion may be detrimental, and he and Bruno advocate tailored exercise programs that take advantage of modern, lightweight braces and wheelchairs to maintain fitness, while easing unnecessary stress on weakened muscles.

—D. Franklin

Sea urchins: Death in the Caribbean

The long-spined black sea urchin *(Diadema antillarum)*, known among divers and coral reef explorers for its sting, apparently has all but disappeared from large areas of the Caribbean Sea. During the last 18 months, a mysterious, fatal disease, its spread following the course of the Gulf Stream, has quickly swept through millions of these prickly creatures. Some scientists fear that the death of the sea urchins may also threaten living coral, while others see the epidemic as a chance to study how an ecosystem responds to the sudden removal of one important component.



Peter W. Glynn

A long-spined black sea urchin.

"This is the first reported occurrence of a mass die-off that I know of for this particular species," says Peter W. Glynn of the University of Miami's School of Marine and Atmospheric Science in Florida. Glynn is one of several researchers now studying the situation. The disease seems to have started somewhere along the coast of Venezuela or Colombia and had spread to Panama's coastal waters by early 1983. It then spread north and east, eventually reaching the Florida Keys, Puerto Rico and the Virgin Islands. "I think it may still be spreading to some of the eastern Caribbean areas," says Glynn, "but it seems to me that it has pretty well run its course in most other areas of the Caribbean."

So far, researchers are not sure specifically what is causing the death of the black sea urchins. Because only one species has been affected and because the disease seems to be waterborne, they suspect that the culprit is a pathogen rather than a physical factor like water temperature or a pollutant.

Black sea urchins, up to a foot in diameter and normally found in vast numbers, feed on algae. Their grazing keeps algal filaments from growing so tall that they change the environment by blocking sunlight and trapping sediment. In the absence of sea urchins, the competition between algae and corals intensifies and shifts to favor algae. This could endanger living corals, says paleontologist Porter M. Kier of the Smithsonian Institution in Washington, D.C.

Glynn is more optimistic. "We have found that although the black sea urchin died in great numbers in the Florida Keys, there are still large breeding populations of the urchins left on some of the reefs here," he says. "The population of urchins may bounce back to its previous normal level relatively quickly in Florida. But that is something we don't know. We're starting to monitor the situation here."

—I. Peterson