

Implanting an Electronic Earful

An ambitious research project aimed at creating an "artificial ear" may someday break the silence surrounding people who are deaf because their ears cannot transform a sound's mechanical vibrations into electrical signals sent to the brain. Electrical engineer Robert L. White and his colleagues at Stanford University in Palo Alto, Calif., are developing tiny electrodes, implanted in the ear's cochlea, that directly stimulate auditory nerve fibers.

Normally, sound waves enter the human ear and reach the eardrum, which vibrates in tune with the sound waves striking it. A set of three bones amplifies the vibrations and passes the sound on to the inner ear. There, inside the snail-shaped, fluid-filled cochlea, cells fringed with fine hairs transform the vibrations into electrical signals that excite nearby auditory nerve cells. If the hair cells are damaged, say through continual exposure to loud noise or because of a hereditary disorder, profound deafness may result, and conventional hearing aids are of no value.

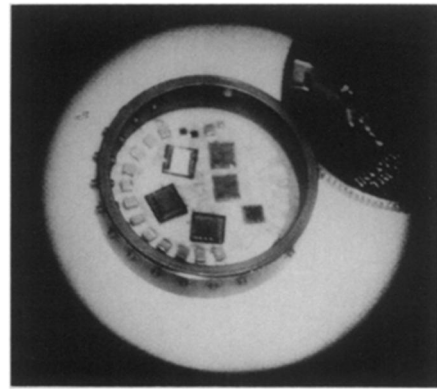
The National Institute of Neurological and Communicative Disorders and Stroke supports several projects devoted to developing cochlear implants to overcome this problem. Researchers at Stanford, the University of California at San Francisco and the University of Washington in Seattle are all designing various forms of these devices. Worldwide, more than 200 systems, which allow a profoundly deaf person to sense that, for example, someone is talking or the telephone is ringing, have been implanted. However, White's ultimate goal is to perfect a more complex system that allows a person to recognize individual words as they are spoken. This requires sensitivity to a range of frequencies. White says that the advanced device his team is developing has been tested on two patients so far. Soon, Biostim, Inc., in Princeton, N.J., plans to start more extensive clinical trials that involve implanting and testing a simpler, single-channel version of the Stanford invention.

In the Stanford "artificial ear," a microphone outside the body picks up the sound, processes the signal and converts it to radio waves, which are transmitted to a miniature receiver implanted in the ear. The receiver, smaller than a quarter, converts the radio signals to electrical impulses. Eight wires carry these impulses to electrodes inserted in the bundle of auditory nerve fibers leaving the inner ear. The trick is to stimulate fibers associated with a selection of different frequencies so that the brain can distinguish different tones.

One of the difficulties was finding electrode materials that would survive for years in a biological environment. Last week, at a meeting of the American Vac-

uum Society in Baltimore, Md., White presented details of the unique, thin-film electrodes his team has developed. These electrodes consist of the metallic conductor tantalum on a sapphire backing and insulated with a layer of tantalum pentoxide. "You need to have insulation that remains insulating for decades over distances which are extremely short, a tenth of the diameter of a human hair," White says. "You can't wrap them in plastic."

White admits that even with eight microelectrodes word recognition is still difficult. This small number of electrodes cannot be expected to replace the 30,000 nerve fibers human beings are born with in each ear. "Our target is primarily unaided speech comprehension," says White. "We're not there by quite a bit. We do have substantial speech discrimination in which an individual can identify words from a short list."



R. L. White/Stanford Univ.

This 8-channel radio receiver-stimulator, when implanted in the ear and attached to a microelectrode array, excites auditory nerve fibers.

White has been working on the project for eight years. "The most difficult aspect has been shrinking the electrodes. We're on our third generation of those electrodes," White says. "We are making good progress in hardware, but cracking the code, finding just where to implant, is not clearly convergent." —I. Peterson

Childhood autism linked to brain allergy

Autism, a severe emotional disorder that begins to appear in infancy and usually lasts a lifetime, may be caused by an allergic reaction to one's own brain tissue, according to a new study by a team of Israeli scientists. Preliminary reaction to the findings ranges from extreme caution to plain skepticism.

According to psychiatrist Abraham Weizman and his colleagues at the Geha Psychiatric Hospital in Petah Tiqva, a recent experiment has yielded the first evidence that the immune systems of autistic children may be misperceiving a basic brain protein as a foreign body and, as a result, systematically devouring it. The resulting brain damage, although undetectable, may be responsible for the constellation of emotional, intellectual and social handicaps that characterize the disorder, the scientists suggest.

As reported in the November *AMERICAN JOURNAL OF PSYCHIATRY*, the researchers conducted a test tube experiment on human lymph cells of autistic children and controls to compare their reaction to myelin, the protein that makes up the protective sheathing of nerve cells. Where under normal conditions certain scavenger cells (called macrophages) tend to move about in the body, in the autistic subjects the lymph cells tended to keep the macrophages in the vicinity of the myelin, presumably to destroy it; this allergic reaction appeared in 76 percent of the autistic children and in none of the non-autistic controls.

The salient symptom of childhood autism is a failure to communicate and to establish normal social relationships, a

characteristic that was for a long time blamed on cold and over-intellectualized mothering. That psychological theory has been almost completely discredited by the last decade of neurological research, which points convincingly to some kind of brain disorder. But scientists have been unable to identify a specific cause of brain damage — a virus or metabolic problem, for example — that is consistently related to autistic behavior. Although it is conceivable that auto-immunity causes some autism, researchers say, these new data should be viewed with skepticism.

"I would describe the study as extremely preliminary, exploratory in nature, a small pilot study, and very tentative," says psychiatrist Edward Ritvo of the University of California at Los Angeles. Not only must the findings be replicated, he emphasizes, but even if they are, it would not be clear that the immunological reaction is related to autism.

Others share Ritvo's caution. Although a myelin allergy has been similarly implicated in nervous system disorders such as multiple sclerosis for years, scientists have been unable to figure out what is going on in the immune response. The theory, according to Wayne State University immunologist Robert H. Swanborg, is that an antigen (a real or perceived foreign substance) activates the body's lymph cells, which in turn act to prevent the normal migration of macrophages; lack of macrophage movement is therefore an indicator of an allergic reaction. But this particular laboratory method, Swanborg says, is "extremely tricky" and its results difficult to interpret. —W. Herbert