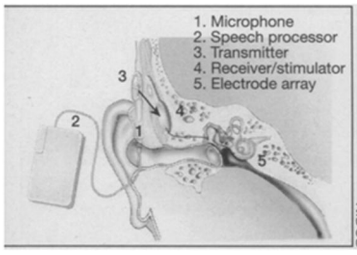
Panel expands use of cochlear implants

A panel of experts convened by the National Institutes of Health recommends expanding the role of the hearing devices known as cochlear implants. Currently, implants can be used only for adults and children who hear nothing with normal hearing aids. The panel now endorses implant use by adults with severe hearing loss who receive marginal benefit from hearing aids.

While cochlear implants were designed to aid the completely deaf, several studies indicate that "ironically, some adults with profound hearing loss using implants could perceive speech and sounds better than adults with less hearing loss using standard hearing aids," says panel chairman George A. Gates of the University of Washington in Seattle.

Standard hearing aids amplify sounds that go through the normal hearing channels from the outer ear through the middle ear to the fluid-filled cochlea, which turns them into electrical impulses to the brain. However, deafness ordinarily results from tissue damage that limits the amount of sound getting through, regardless of how loud it is. Cochlear implants bypass normal hearing channels by transforming useful sounds into electrical impulses and transmitting them directly to the brain. With training, people can perceive these impulses as sound, in some cases "hearing" so well that they can use the telephone.

Not all people achieve that level of proficiency, however. The panel notes that adults who lose their hearing after learning to speak and who receive implants soon after becoming deaf attain the clearest speech perception. Children who get implants at age 2 to 3 years also tend to improve their speech and language skills. Adults who were deaf before learning to speak usually gain the least from implants, but even they "can obtain some awareness of environmental sounds and danger sounds," says Gates.



The cochlear implant microphone hooks over the ear and transmits sound to an external speech processor. Signals travel to the transmitter on the scalp, which clings magnetically to the receiver embedded beneath the skin. Electrodes in the cochlea stimulate nerves to send messages to the brain.

New role for immune cells in diabetes

A study of mice susceptible to insulin-dependent diabetes mellitus (IDDM) establishes new roles for the immune cells that cause the disease and pulls the plug on a potential therapeutic approach.

A team of French researchers created a strain of mice that specifically generate the T lymphocytes—known as T1 and T2—associated with IDDM. They report in the May 26 SCIENCE that contrary to previous speculation, T2 cells don't protect against the disease.

IDDM occurs when T lymphocytes infiltrate the pancreas and destroy the insulin-producing cells. Many scientists had speculated that a battle between the destructive T1 and protective T2 cells then ensued. The Université Louis Pasteur researchers tested the response of newborn mice to each type of T lymphocyte and to both together. Although they found that T1 cells alone caused IDDM, T2 cells neither caused nor protected against the disease.

"Our experiments do not augur well for trying to use T2 cells to modify the disease once it has started," says author Diane Mathis. But it may help researchers combat IDDM by directing attacks against T1 cells.

Scientists identify new Ebola virus . . .

In November 1994, a 34-year-old zoologist studying a wild chimpanzee troop in West Africa's Taï National Park performed an autopsy on a chimp that had died of an unknown illness. Eight days later, the woman got sick herself.

She was flown home to a Swiss hospital and eventually recovered. Now, a team of scientists has discovered the cause of this woman's sickness—a new strain of Ebola virus.

It joins three other strains of Ebola, including the lethal Zaire version that is currently fueling an epidemic of human illness in the city of Kikwit (SN: 5/27/95, p.333).

The new Ivory Coast strain causes both fever and hemorrhage in chimp and human victims. Reports of those symptoms alerted Bernard Le Guenno of the Pasteur Institute in Paris and his colleagues to the possibility of infection with a type of Ebola virus.

The researchers obtained samples of the woman's blood and flushed out a new strain of Ebola virus.

"The new strain is lethal for chimpanzees and we may suppose for humans," the authors write in the May 20 LANCET.

Ebola viruses are known as filoviruses for their long, filamentlike appearance under a microscope. "This is the first time that a human infection has been connected to naturally infected monkeys in Africa," say the authors.

. . . and find the werewolf gene's lair

The legend of the werewolf has made its way into the annals of science. Researchers report that they have homed in on the location of a gene responsible for a werewolf syndrome.

Congenital generalized hypertrichosis is a rare genetic trait thought to be transmitted on the X chromosome. Individuals with this disorder have an upper body and face covered with hair and have often ended up in sideshows as human werewolves, say the authors.

Pragna I. Patel of the Baylor College of Medicine in Houston and her colleagues studied 19 members of a single, multigenerational family with this disorder. Her group extracted DNA from blood drawn from family members. Using a technique known as linkage analysis, they traced the gene to the long arm of the X chromosome.

Although Patel's group has narrowed the location of the gene to a neighborhood of DNA, they have yet to identify the actual gene itself.

Biologist Brian K. Hall of Dalhousie University in Halifax, Nova Scotia, proposes this gene as an example of one that was important in our ancestral past but that has been turned off in modern humans. Patel points out that almost all mammals retain a protective furry coat. Perhaps this gene provided early humans with the same benefits, she speculates.

Patel's report and Hall's commentary appear in the June NATURE GENETICS.

The new report may spur an interest in other genes that control human hairiness. "It gives us another tool for understanding the genetics of hair growth," Patel says.

Gallo to launch new research venture

AIDS researcher Robert Gallo will leave the National Institutes of Health this summer to start a new research institute that will be associated with the University of Maryland at Baltimore.

Gallo had in recent years been under attack for his role in a dispute over HIV, the virus that causes AIDS. That quarrel centered on whether Gallo had misappropriated a virus that had been discovered by French scientists (SN: 7/16/94, p.37).

Gallo's new venture will be called the Institute of Human Virology. It will focus on AIDS research, some cancers, and autoimmune diseases.