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NEWS

Engineers to Implant Antivertigo Device

Vestibular prosthesis is a pacemaker for the inner ear

By JOSEPH CALAMIA / OCTOBER 2010

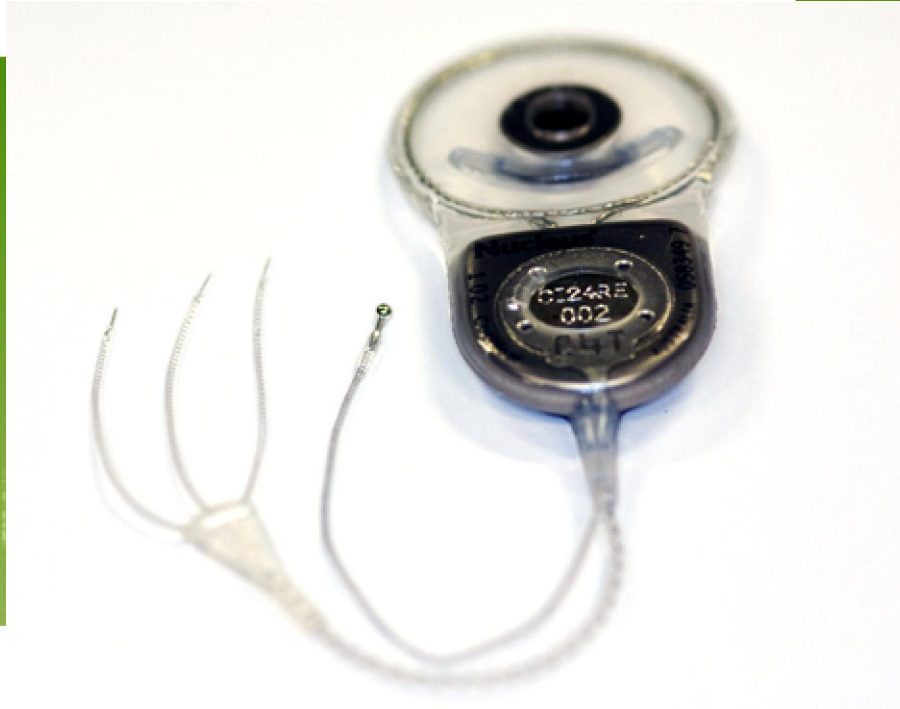


Photo: Cochlear

once a week." For most, diet changes and diuretics can stop the attacks by lowering inner ear pressure, but about 15 percent of patients require surgery to decrease the sensitivity of the inner ear—and the most severe cases require disconnecting nerves to the inner ear altogether.

"If the alternative is to go in and kill your ear," says James Phillips, a vestibular neurophysiologist at the University of Washington who developed the device with Rubinstein, "then maybe it makes sense to provide a prosthesis."

For such severe cases, the FDA has approved a 10-person trial of the device. The prosthesis resembles a cochlear implant, which is designed to help deaf people hear. But while a cochlear implant uses one electrode array to stimulate the auditory nerve, this prosthesis will rely on three shorter arrays to stimulate the vestibular nerve, which is crucial for balance.

Two and a half millimeters long and 150 micrometers in diameter, each array will go into one of the inner ear's three perpendicular semicircular canals. When functioning normally, these canals work as biological gyroscopes, allowing the brain to determine head rotations in any direction by monitoring the deflections of tiny hair cells.

"The hair cells are imbedded in a sail, essentially," which projects out into the fluid-filled semicircular canal, Phillips says. "As you turn your head, the fluid lags behind, and the sail billows." This billowing bends the hair cells; the hair cells in turn trigger an electrical signal to the vestibular nerve. It's believed that these electrical signals tell the brain that you are turning.

The exact cause of any one Ménière's attack is unknown and, depending on the severity of the attack, may stem from a variety of sources, Rubinstein says. His team's device is meant to halt attacks caused by a potassium leak from ruptured

20 October 2010—Tomorrow, 21 October, a surgeon will attempt to fight debilitating vertigo by rewiring the body's balance center. Jay Rubinstein, a surgeon and biomedical engineer at the University of Washington, in Seattle, will insert a "vestibular prosthesis" inside his patient's head, weaving electrode arrays into the depths of the inner ear. He hopes that pulses from these electrodes will stop vomit-inducing dizziness caused by Ménière's disease.

"When a Ménière's attack occurs, you basically have to lie down and curl up into a ball," Rubinstein says. "It's not very conducive to a productive existence if these are happening

inner-ear membranes. The leak disrupts the delicate sodium-potassium balance in the canal membrane, which acts as an inner-ear "battery." The extra potassium disables this battery, "short-circuiting" the canal and shutting off power to the hair cells. This tricks the brain—which is always monitoring the difference between signals coming from the two inner ears—into perceiving a sudden spin.

Enter the vestibular prosthesis. Rubinstein's theory is that by supplying bursts of electricity to the vestibular nerve to make up for the temporarily disabled hair cells, the device should bring the perceived spinning to a stop.

"The idea as they've described it is to use it like an as-needed pacemaker," says Charley Della Santina, a surgeon and biomedical engineer at Johns Hopkins University, in Baltimore, who is not involved with the study. He explains that the user will activate the device during a Ménière's attack. "It's not clear yet whether or not that will work to override abnormal inner ear signals that cause vertigo, but it will be clear soon," he says, noting tomorrow's surgery.

A team led by Della Santina is creating a [different kind of vestibular prosthesis](#), which constantly sends signals to the brain, to treat a different disorder. Though not yet at the clinical trial stage, the Johns Hopkins device will use signals from implanted gyroscopes in an attempt to restore balance for patients with bilateral vestibular loss.

Typically, our eyes respond to signals from the vestibular nerve, moving in the opposite direction of our heads to keep our view steady. Patients with bilateral vestibular loss lack that ability, and their world appears to shake during any head movement. That makes it difficult for them to see while driving, for example. They also tend to walk as if they are drunk and have a greater risk of falling.

The Johns Hopkins device takes 3-D head movements, picked up by the gyroscopes, and converts them into vestibular nerve signals, allowing the eyes to stay on target. Della Santina will present an update on this prosthesis as a keynote speaker at the [IEEE Biomedical Circuits and Systems Conference](#) next month.

Although the Johns Hopkins project aims to treat a different balance disorder, Rubinstein hopes that by providing more human data on vestibular implants in general, the device he will implant tomorrow will mean progress for both teams.