

New Hope For Stroke Patients: Reversing Stroke Damage By Jumpstarting Growth Of Nerve Fibers

But now researchers report a technique that potentially could restore functions to patients weeks or even months after a stroke. The technique involves jumpstarting the growth of nerve fibers to compensate for brain cells destroyed by the stroke.

"In the best-case scenario, this would open up the window of time that people could recover and go back to normal functional status," said Gwendolyn Kartje, MD, Ph.D., a professor in the department of cell biology, neurobiology and anatomy and department of neurology at Loyola University Chicago Stritch School of Medicine in Maywood, Ill. and chief of neuroscience research at Edward Hines Jr. VA Hospital in Hines, Ill.

Kartje and colleagues described the experimental approach, called anti-nogo-A immunotherapy, in a recent review article in the journal Topics in Stroke Rehabilitation.

Anti-nogo has dramatically improved functions in lab animals that have experienced strokes. And an ongoing clinical trial in Europe and Canada is testing anti-nogo in humans who have suffered spinal cord injuries.

Most strokes are caused by clots that block blood flow to one part of the brain, killing brain cells within hours. The drug TPA can minimize damage by dissolving the clot. But TPA is safe and effective only when given within about three hours of the onset of symptoms. Most patients don't receive treatment within that brief window. Patients typically arrive at the hospital too late, or hospitals do not begin administering TPA soon enough.

Anti-nogo is among several new approaches under study that potentially could reverse stroke damage, researchers wrote. Nogo-A is a protein that inhibits the growth of nerve fibers called axons. It serves as a check on runaway nerve growth that could cause a patient to be overly sensitive to pain, or experience involuntary movements. (The protein is called nogo because it in effect says to axons: "No go.") In anti nogo immunotherapy, an antibody disables the nogo protein.

The left side of the brain controls movements on the right side of the body, and vice versa. Thus, a stroke on the left side of the brain can cause paralysis on the right side of the body. In such a patient, anti-nogo would, it's hoped, spur the growth of axons from the healthy right side of the brain. These axons would then grow into the right side of the body and restore functions lost by the stroke.

Anti nogo has been tried on rats that have experienced strokes in old age. As in people, strokes in rats affect one side of the body. Following strokes, the rats were unable to pick up pellets of food with the front paw on the affected side. After anti-nogo, function in this paw was almost completely restored in some rats.

The Swiss pharmaceutical company Novartis is sponsoring a phase 1 clinical trial of anti-nogo for patients paralyzed by spinal cord injuries. Kartje believes anti-nogo also has great potential for stroke patients. A clinical trial for stroke patients could begin as early as 2012, she said. Loyola is among the potential sites for such a trial.

Anti nogo "offers the potential for stroke patients to recover, return to nearly normal functional status, and stay out of nursing homes," Kartje said. "Theoretically, there's no reason why this should not happen."

Kartje began studying the nogo protein in 1992, and has published numerous papers on the topic. Her lab at Hines is funded by the Veterans Administration, with additional funding from the National Institutes of Health, Neuroscience Institute at Loyola University Chicago Stritch School of Medicine, Falk Foundation and Illinois Regenerative Medicine Institute.

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