Could a Pill Fix the Brain?

Neurologists are exploring medications that would help the brain heal itself after a stroke or traumatic injury.

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By Rachel E. Gross

Sept. 4, 2025

The first thing Debra McVean did when she woke up at the hospital in March 2024 was try to get to the bathroom. But her left arm wouldn't move; neither would her left leg. She was paralyzed all along her left side.

She had suffered a stroke, her doctor soon explained. A few nights before, a blood clot had lodged in an artery in her neck, choking off oxygen to her brain cells. Now an M.R.I. showed a dark spot in her brain, an eerie absence directly behind her right eye. What that meant for her prognosis, however, the doctor couldn't say.

"Something's missing there, but you don't know what," Ms. McVean's husband, Ian, recalled recently. "And you don't know how that will affect her recovery. It's that uncertainty, it eats away at you."

With a brain injury, unlike a broken bone, there is no clear road to recovery. Nor are there medical tools or therapies to help guide the brain toward healing. All doctors can do is encourage patients to work hard in rehab, and hope.

That is why, for decades, the medical attitude toward survivors of brain injury has been largely one of neurological “nihilism,” said Dr. Fernando Testai, a neurologist at the University of Illinois, Chicago, and the editor in chief of the Journal of Stroke and Cerebrovascular Diseases. Stroke, he said, “was often seen as a disease of ‘diagnose and adios.’”

That may be about to change. A few days after Ms. McVean woke up in the Foothills Medical Center in Calgary, she was told about a clinical trial for a pill that could help the brain recover from a stroke or traumatic injury, called Maraviroc. Given her level of physical disability, she was a good candidate for the study.

She hesitated. The pills were large — horse pills, she called them. But she knew the study could help others, and there was a 50 percent chance that she would get a drug that could help her, too.

Eventually, she agreed. “I was game,” she said. “I didn’t want to be in a wheelchair all my life.”

Debra McVean of Calgary, Alberta, suffered a stroke in 2024 that left her paralyzed along her left side. Amber Bracken for The New York Times

A ‘Harsh Decree’

Dr. S. Thomas Carmichael, the head of neurology at the Geffen School of Medicine at the University of California, Los Angeles, was taught the same thing again and again in his medical training: The brain doesn’t grow back. “Unlike, say, the liver, there is no regenerative capacity,” he recalled being told in the 1990s. “You work with what you’re given.”

In many ways, neuroscience was stuck in the age of Santiago Ramón y Cajal, one of neurology’s greatest minds. Dr. Carmichael’s teachers often quoted Dr. Cajal’s 1928 declaration that, in the adult brain, “the nerve paths are something fixed, ended and immutable. Everything may die, nothing may be regenerated.”

But in his rotation at a rehabilitation center for brain injury survivors, Dr. Carmichael saw evidence to the contrary. His patients relearned how to walk, to grasp, to string words into sentences. Somehow, their brains were healing and adapting.

“There is something happening,” he said. “It just doesn’t get very far.” That something, he learned, was the brain reorganizing.

Against the advice of his thesis advisers, he set out to discover whether the brain could repair itself. What he learned would astonish the field: After injury, healthy neurons far from the site of damage sprouted new axons, the rootlike tentacles that conduct electrical signals.

A stroke does not just kill off part of the brain. It also disrupts a vast network of neurons that exchange messages with far-off regions. The death of one neuron can take thousands of these connections down with it, like downed power lines.

And yet, Dr. Carmichael found, the injury initiates a wave of plasticity and growth throughout the brain, an event previously thought to occur only in development. Neurons come alive again, sprouting new rootlets that poke their way into gray matter and try to re-establish lost connections.

Not many succeed. But it may take only a few to rewire distant parts of the brain. That is most likely how Ms. McVean woke up one morning in a rehab center, a month after her stroke, and found that she could rotate her left thumb. A few days later, she waggled a finger. “That was a big, big deal,” she said.

Therapist Azul Gordiano helped Ms. McVean stretch and move her fingers during an in-home rehabilitation program. Amber Bracken for The New York Times

While the brain can regenerate, that process is limited. Very few stroke survivors ever achieve close to a full recovery, according to the American Stroke Association. It's as if, at some point, the brain decides it is done healing and returns to its default state.

Dr. Carmichael wanted to go further, to keep the window of plasticity open longer and allow the brain to heal beyond its natural limits. There was, he recalled, a second half to Dr. Cajal's statement: "It is for the science of the future to change, if possible, this harsh decree."

Maybe the science of the future was finally here.

Opening the Floodgates

In 2015, Dr. Alcino Silva, a leading memory researcher and colleague at U.C.L.A., was studying “smart” mice — mice with mutations that enhanced their ability to learn and remember. One day, he called Dr. Carmichael over to see a mouse that was smart for an unexpected reason: It was missing an immune gene.

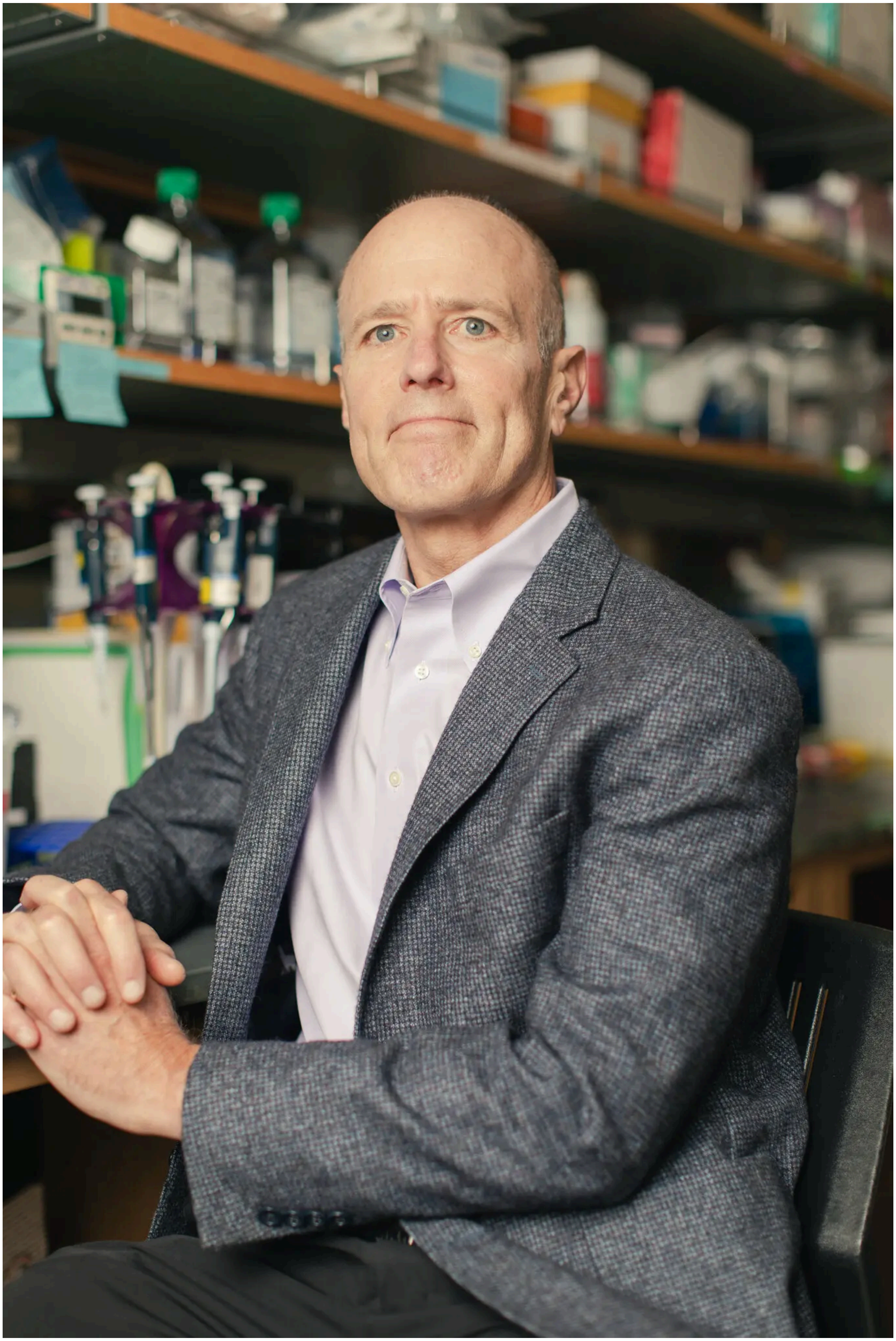
The gene coded for a receptor called CCR5, which, Dr. Silva’s lab had found, seemed to suppress plasticity, memory and learning. He wondered if it might play a role in recovery from stroke, which triggers the immune system to flood the brain with inflammatory cells.

Dr. Carmichael was intrigued. In a healthy human brain, CCR5 was not present in neurons. But after a stroke or other brain injury, the receptor suddenly appeared everywhere in the brain.

The period of initial plasticity following a stroke, he realized, was being cut short by CCR5. Like a dam closing, the receptor seemed to tell the brain: Enough. Let’s lock in what we’ve learned, and call it a day. Maybe this was why stroke survivors rarely fully recovered: The brain was holding itself back.

The mutant mice did not have that safety valve, however. Their window of brain plasticity stayed open longer. After a stroke or traumatic injury, Dr. Carmichael and Dr. Silva found, they recovered faster and more completely.

The next step was to see whether the same was true for humans with the mutation, a group that included Ashkenazi Jews. By this point, the researchers were leading an effort funded by the Adelson Medical Research Foundation to find new approaches to recovery from brain injury.



Dr. S. Thomas Carmichael, the head of neurology at the Geffen School of Medicine at the University of California, Los Angeles, was taught the same thing over and over in his medical training: The brain doesn't grow back. Alex Welsh for The New York Times

The foundation connected them to Dr. Einor Ben Assayag, a neurologist at Tel Aviv University in Israel who was tracking a cohort of 600 stroke patients to see which ones developed dementia.

Amazingly, she had kept blood samples of every patient, in addition to cognitive evaluations over time. When she analyzed her data, she found that patients with some form of the CCR5 mutation had better language, memory and attention scores. This was groundbreaking: They had identified the first gene associated with stroke recovery.

But the researchers had more than just a target; they also had a drug that mimicked the mutation. Tawnie Silva, Dr. Silva's wife and a researcher in his lab, had found it while researching the mutant mouse strain: a little-known H.I.V. treatment that had been approved by the Food and Drug Administration in 2007. It was called Maraviroc.

"I mean, that's a unicorn kind of thing," Dr. Silva said. "That's incredibly rare."

As it turned out, the CCR5 receptor was also known as the portal that H.I.V. binds to in order to enter cells. In the 2000s, as the deadly virus gained resistance to older medications, Pfizer developed a drug that blocked this portal and protected cells from infection.

But no one had looked at what Maraviroc might be doing in the brain. In 2019, Dr. Carmichael laid out three lines of evidence showing that Maraviroc boosted neuroplasticity after brain injury, and published his findings in a landmark paper in the journal Cell.

As he was sharing his results at a conference later that year, Dr. Sean Dukelow, a Canadian stroke neuroscientist sitting in the back row, grew excited. Dr. Dukelow would become the main investigator conducting the Maraviroc trial at the Foothills Medical Center and across Canada.

When Dr. Dukelow was a teenager, at around the same time that Dr. Carmichael was being taught that the brain was static, he watched his grandfather suffer a mini-stroke at home. Since there were no therapies for brain recovery, all his family doctor could offer was bed rest and an aspirin. Within a year, his grandfather died of a full-blown stroke.

For 70 years, the field had believed the brain could not rewire. Now, "we're actually on the verge of guiding that rewiring," Dr. Dukelow said. "Do I wish it would have moved faster? Yes. But it's actually pretty incredible to have come through and watched it happen, to go from absolutely nothing to now there's hope."

Ms. McVean performed arm exercises at home. Amber Bracken for The New York Times

Changing Minds

Maraviroc is not a perfect drug, Dr. Carmichael said. It does cross the blood-brain barrier, but only in limited amounts. That's why his allegiance is not to one drug, but to laying the groundwork for future therapies by deepening the understanding of the brain's recovery systems.

In May, in his office at U.C.L.A., he projected onto the wall an image of what looked like a glowing green centipede covered in knobby legs. This was a dendrite, a branch of a mouse neuron that receives signals from other neurons. The knobs were dendritic spines.

After a stroke, his next image showed, many of these spines disappeared — the centipede lost some legs. But if the mouse was made to perform precise motor tasks for a month, it could actually sprout new ones. “Rehab boosts these,” Dr. Carmichael said. “There are more little green things.”

He recently identified a drug that produced a similar effect in the brain, leading to better motor recovery in mice. While promising, it would take years and “a lot of non-sexy science” to bring this “neurorehabilitation pill” to market, he said.

If any of these therapies make it to F.D.A. approval, it could change not only the way doctors treat brain injury patients, but also the way those patients imagine their own futures.

Ms. McVean still doesn’t know whether she received Maraviroc; the trial won’t be complete for another two years. But she knows her brain is still rewiring, reorganizing and adapting to its new reality, more than a year after her stroke.

Sitting in her folding wheelchair in her kitchen last May, she lifted a one-pound weight with her left hand, a feat that would have been impossible six months ago. She can now wheel from her bed across the kitchen to make herself coffee. She can walk upstairs, tentatively, with a brace. “I count the stairs,” she said. “I know there’s 15.”

Recently, she noticed her fingers on her left hand becoming more mobile. “They don’t feel like they don’t belong to me anymore,” she said.

Whether or not she received the drug, she knows some innate capacity for recovery is there. In their own ways, she and Dr. Carmichael are continuing to challenge Dr. Cajal’s harsh decree.

Ms. McVean receiving a massage as part of a strengthening and stretching program at home. Amber Bracken for The New York Times