Susan J. Harkema, Ph.D., is director of the Reeve Foundation NeuroRecovery Network®, professor of neurosurgery and rehabilitation at the University of Louisville, and research director of the University of Kentucky’s Spinal Cord Research Center and Frazier Rehab Institute. Her career has been built around a basic concept in human biology: there are nerve bundles in the spinal cord that control major functions, such as stepping. These circuits are smart on their own; they don’t require connection to the brain. Harkema’s work with people with spinal cord injuries has shown that recovery of function is possible – even in people thought to be completely paralyzed – by activating spinal circuits. In 2011, along with her mentor Reggie Edgerton, Ph.D., from UCLA, Harkema’s lab implanted an epidural stimulator next to the lumbar spinal cord of paraplegic Rob Summers. Surprisingly, he regained voluntary leg function when the stim was on. Two more subjects have since received an epidural stimulator and a fourth is set for January; Harkema said the results, to be published soon, are just as exciting as Summers’ were. Harkema spoke with Reeve staffer Sam Maddox in her office at Frazier Rehab, overlooking downtown Louisville.

Q. Some scientists are inspired early on to their vocation. How were you drawn toward this career?
A. I tried a lot of other things. I was a computer science major, an electrical engineer major and then became an athletic trainer because, well, just because my dad was a college football coach, and he’s like, ‘I’m tired of you changing your major, so why don’t you be an athletic trainer?’ Being a trainer wasn’t going to sustain me; medical school was a brief consideration. I took a gig as a research tech, this while I was at Michigan State. I loved it. I did multiple surgeries for many different projects for the physicians. I was very good technically. That was my strength. I mean, I transplanted rat hearts. I was really driven and loved the idea of doing something that hadn’t been done before, figuring out how to do it. And one day, one of the scientists on one of the projects came in and said, ‘You should get your Ph.D.’

Q. What did you get the Ph.D. in?
A. Physiology – based on a really exciting question in the small-muscle energetics field, whether ATP was actually the substrate for oxidative phosphorylation in skeletal muscle. (It is not.) It was a great research experience, but it’s not a question with any direct link to any disease state.

Q. You wound up on the West Coast for your post-doctorate…. A. I found Reggie Edgerton at UCLA. His lab fit the criteria I was looking for, including warm weather! His work was about muscle, so I proposed to him that I could look at metabolism in paralyzed muscle; I had done all my work in cats, and they had a cat model at that time. I
did end up there but soon moved to a human project, and there started my career in spinal cord injury. Of course once I started doing it and learning about the lives of people with spinal cord injury my motivation and passion really changed. I love being a scientist. I love designing a question and figuring out how best to answer it and then actually getting the answer. It’s still incredibly exciting to me. But now there’s a true humanitarian mission; you can’t get to know people living with spinal cord injury without them inspiring you. I always come back to the ‘ASIA As,’ those people with clinically complete injuries; my real core passion is to figure out how we can incrementally change the lives of these individuals.

Q. What were some of your early projects with the Edgerton group?

A. These were the beginning of the experiments we talk about now: exploring how the circuitry of the spinal cord is able to generate a stepping pattern, apart from any input from the brain. We looked at activity-based therapies, including locomotor training. Could people with clinically complete injury demonstrate that sensory processing occurs in the spinal cord? The first thing we did was attempt to generate a locomotor pattern; if central pattern generation doesn’t exist in the spinal cord, then no matter what we might try, there should be no output whatsoever in a patient with a complete injury. Amazingly, we got a motor pattern. Then the next step is, can you modulate it? We said ‘Okay, if we change load, does the pattern change?’ And the answer was yes: change load, you get more EMG activity and get a better pattern. We kept showing that there was some sophistication in the neural networks of the spinal cord.

Q. When did you realize this work might lead to a therapy for people?

A. This is going to sound harsh, but it isn’t meant to be. We weren’t trying to get people better. We were trying to understand how this circuitry worked, with no expectation that people would regain function. But when we did these sensory experiments, we asked, ‘can we train humans to be able to independently step?’ And what happened to those with incomplete injury is that a significant number of them came into the clinic in wheelchairs and they walked out – they didn’t walk out like before their injury, but they could take steps.

We started noticing other things. People began to report to us that this really changed their daily lives. They felt better, and their circulation and healing were better. People who were C4-complete regained a lot of trunk and arm strength; some were able to move from powered wheelchairs into manual ones. And this was just from standing. People reported that their bowel movements were improving, as was their bladder function. We also started seeing changes in muscle and bone and all these other aspects – respiration, cardiovascular function.

Q. It seems the notion of recovery, or cure, is defined on a sliding scale …

A. I learned that we can’t judge whether what we discover and are able to disseminate to the population is important or not – that is up to the individual living with SCI and where he or she is in life, what that person’s expectations are. I remember one individual who could stand whenever he wanted as long as he wanted, and there was another who could only stand on one leg, for a minute or two. And the person who could stand anytime he wanted was very disappointed; he wanted to
walk. And the person who could stand for a minute or two was ecstatic. Obviously, we can’t ever stop until we’ve completely solved the paralysis riddle. But maybe we need to translate our promising research earlier, and not wait to translate until we have the whole puzzle solved.

Q. That’s how the NeuroRecovery Network (NRN) came about, right? Translating what we know, now.

A. Right. The only place people were able to get locomotor training was in the laboratory, and of course we couldn’t keep training them. So Christopher and Dana Reeve saw this was happening and the Foundation got involved — to develop research and a clinical component.

Q. And the NRN is of course a major Reeve Foundation program.

A. The NeuroRecovery Network has turned out to be something beyond our imagination. We’ve got all these amazing clinicians and scientists and administrators working together. We continue to study locomotor training. We’re developing new outcome measures. We’re also in a position now in a very cost-effective way to support clinical trials, because the infrastructure and capacity to standardize are in place. We have an amazing electronic Web-based system that can very inexpensively gather data from multiple sites. We’re submitting a lot of different grants to advance evidence at different levels, branching out into the cardiovascular arena, the pain arena and bladder function as well as other health-related aspects of SCI.

“I learned that we can’t judge whether what we discover is important or not — that is up to the individual living with SCI and where he or she is in life, what that person’s expectations are.”

There’s also a significant pediatric component of the NRN; recent evidence tells us that the plasticity in the pediatric population may be even more accessible than in adults. Here at Frazier we’re building a...
research-clinical pediatric program, led by Andrea Behrman, Ph.D. She was at the University of Florida for many years but she just moved her clinical studies here to Louisville.

Q. You spent 10 years with Edgerton at UCLA, got recruited to Louisville in 2005. You have a lot of duties here, between teaching, running a lab, writing grants, traveling to meetings. Plus, you are married, with kids. How are you balancing the Mom job?

A. You don’t balance it. You just do the best you can every minute of the day. My husband is a comedian and an actor, and when we moved to Louisville he decided to stay home with the kids, so that helps a lot in having a parent available all the time. But it is a struggle to make sure that you’re spending enough time with them. Graduate students will ask me ‘When is the best time to have your family? When you’re in graduate school, or is it after you get your first RO1 [major federal funding]?’ I always say, ‘When you have them, that’s the best time.’ Somehow, you figure it out.

Q. What’s the take-home message for the series of NRN articles published in September in the Archives of Physical Medicine & Rehabilitation?

A. Those articles lay out what’s possible with activity-dependent plasticity. It’s not the end-all. It’s the start. First of all, in relationship to spinal cord injury, or any neurologic disorder, recovery doesn’t stop one year after injury. For me that’s almost the most compelling message: We had people in the NRN who had been a decade, two decades in a wheelchair who recovered their ability to walk. Now, they aren’t running a marathon, but they are walking around in their homes and in their communities. We were able to generate some predictive models for which person will recover what function based on our data of 400 people.

The other main point is that the outcome measures we have available are very limited — they only are sensitive for a slice of the population that we’re studying. This is really critical as interventions come forward; we may be missing potential options because we’re measuring a change in one type of score, such as ‘we’re looking for a 10-point change in the ASIA’ [a measure of motor and sensory function]. But you may have recovery that’s unrelated to that change. So the NRN developed recovery curves that are applicable to any number of interventions. Now we can demonstrate that with this intervention (say, locomotor training), for this type of patient, this is the recovery curve that you can expect for this number of training sessions. We introduced the Neuromuscular Recovery Scale in the Archives; it has an advantage over current measures in that it is sensitive for the whole continuum of recovery; it can pick up changes at the earliest stage, things for people who can’t stand or walk, but also through standing and walking.

Q. Would you say this is a hopeful time for the SCI world?

A. It’s a very hopeful time because there’s so much research going on, and there is a widespread commitment to translation. It’s clear that activity-dependent plasticity can play a huge role in recovery. We have an obligation to try to make therapies accessible to the community. The NeuroRecovery Network is one way to do that, not only to introduce new activity-based therapies into clinical settings and figure out how to most cost-effectively provide them but then also to disseminate information about the benefits so individuals and rehabilitative centers can decide. ‘Okay, look, I know that if I train this individual with this intervention for 80 sessions they’ll be able to sit independently.’ Well, then everybody can decide if that’s something that’s worth their investment, from the patient to the center to everybody else.
Q. What about for people with so-called complete injury?
A. I don’t know of anyone who’s walked who has been classified as a clinically complete injury but there are important effects in regaining trunk or arm control. But it’s all the other kinds of side effects of locomotor training that are really critical for the clinically complete population: their cardiovascular function gets better; they report fewer autonomic dysreflexia episodes; they have better circulation; they feel better. When you’re sitting all the time you’re not activating the neuromuscular system because you are unloaded. And as a physiologist it’s very clear to me that being unloaded throws off all the other aspects. The irony is that the patients most in need of locomotor training are probably those who aren’t going to walk as a result of it. They need it because that’s their only way to bear weight and get neuromuscular activation of their bodies, which in turn helps to improve and/or maintain critically important health outcomes.

Q. Epidural stimulation opens possibilities for complete injury, yes?
A. We selected motor-complete individuals but when the stimulator is turned on, they can move voluntarily; they intend to move a toe and they actually do move that toe. And so people say, ‘Well, those patients must not be complete.’ Well, okay, but that opens up a whole other set of possibilities. If we assume someone’s complete, the interventions available to incomplete individuals are sort of closed off to them – we know they are not going to respond because they’re complete. Clearly epidural stimulation is helping us understand not only how we classify individuals but also that it is something that can be useful for people with the most devastating injuries. It is encouraging that individuals who, by conventional measure, are considered clinically complete actually have capacities for recovery.

Q. You have tested three patients with epidural stimulators now?
A. Right. We have Rob Summers and two more implanted with epidural stimulators; all three have had, in general, the same result. All have regained significant muscle mass. They all can stand with the stimulator, independently of any assistance. Rob is an ASIA B [no motor, some sensory function] and we have another B and an ASIA A; the B has regained more sensory control without the stimulator but each of them can move voluntarily with the stimulator on. Our second B has now officially converted to a C [some motor and sensory recovery]; he can do some voluntary movement of his toes and ankles without the stimulator on. We’re three-for-three in the results. Clearly we have to expand the number of people we study to see how generalizable these results are.

Q. So complete may not be so complete.
A. Being complete may not be anatomically complete. Is there some tiny thread of a nerve tract in these individuals that just happened to get spared? Is that what’s allowing this voluntary activity once we excite the spinal cord? We don’t know. To me the most astonishing thing is that you turn the stimulator on and then you turn it off and there’s a difference. We’re assuming there must be some connection – these patients intend to move their legs and they do, so there has to be a connection. Yet we have
Jeff Petruska is a scientist at the Kentucky Spinal Cord Injury Research Center at the University of Louisville. He got the science bug early on: “I had a pet frog that died. Instead of burying it, my mom said, ‘Well, let’s dissect it. I’ll show you the things on the inside.’ She took it apart and showed me where the heart was and how the muscles attached to things and all this stuff that you don’t see from the outside. From that point on, third grade, I loved biology.”

He’s still looking inside. After grad school at the University of Florida, Petruska became a post-doc Associate in Lorne Mendell’s laboratory at SUNY-Stony Brook, one of six labs in the Reeve Foundation International Research Consortium on Spinal Cord Injury. “I was fascinated by the electrophysiology they were doing there, how neurotrophins affected the physiology and anatomy of neurons. I loved it.” He didn’t anticipate the impact this would have on his career.

“The Consortium is an amazing entity; it trains its Associates in how science is really being done now. You have to be multidisciplinary. You have to collaborate. The Consortium is about working across disciplines, across many labs.”

The Associates from the Consortium labs meet formally twice a year. “We spent a lot of time together. This was absolutely invaluable to me. I formed a cadre of people I trust. I know their science. I know their approach. I’ve gotten calls to help them design experiments, and I do the same thing, reaching out to them when I need help.”

As an Associate, Petruska spent a lot of time in the Consortium labs of Reggie Edgerton (UCLA), Mary Bunge (Miami), and Fred Gage (Salk). “I learned to speak the language of their experiments, and they learned mine,” he said. “I learned more from the Consortium than I would have by going on to a second post-doc. I set up a number of collaborations that have been invaluable as I moved on and started my own lab.”

Petruska was attracted to the Kentucky SCI program because he says it reminded him of the Consortium model. “I interviewed with a bunch of places that all had really great stuff going on. But this place felt the closest to that collaborative model.”

Petruska’s current work evolved from a Consortium assignment he began at the Mendell lab. “The question I asked is: How do adult neurons that are not injured extend new branches of their axons? Of those that are injured, some extend axon branches using regeneration. But some axons that are not injured grow and change their connections too. It’s called collateral sprouting. So I put this idea in front of the Consortium, all of the principal investigators, the advisory panel – they said ‘do it.’ That was a powerful expression of faith and confidence and very meaningful to me as a postdoctoral fellow.”

Once set up in Louisville, Petruska put a team together. “I’m not a molecular biologist. So that’s where I went to Laurence Moon, who at the time was the Associate in Mary Bunge’s Consortium lab at the Miami Project. He had just completed his own micro-array [screening for genes] experiment, which was outstanding. He compared
the genes in the central nervous system neurons that could regenerate after injury with those that could not regenerate.”

Petruska recruited his own post-doc, molecular and cellular biologist Ben Harrison. The university already had a computational/bioinformatics expert, Eric Rouchka, to develop tools to analyze huge datasets. "Together we've gone from a broad data set to one that identifies, for the first time, the specific genes necessary for collateral sprouting."

The genes needed to turn on regeneration are not the same as those that control sprouting. Is there clinical relevance? “Possibly. We think that a lot of the plasticity observed after TBI and SCI is not actually regeneration. It might be collateral sprouting. This can be good or it can be bad; recovery after stroke involves collateral sprouting of non-injured neurons. But so does autonom-ic dysreflexia [blood pressure disregulation]. In AD you want to stop collateral sprouting. But you don’t want to stop it in TBI if it’s helping to recover certain functions.”

Using genetically modified animal models to explore the difference between the two processes, Petruska is hoping eventually to manipulate the genes for a therapeutic advantage.

— Sam Maddox

NACTN: Reeve Network Focus of Journal Series

Clinical trials are being held, or planned, for several promising therapies for spinal cord injury. The translation of research to application requires a coordinated approach, from preclinical data assessment, trial protocols to actual treatment and outcome measures.

The Journal of Neurosurgery: Spine recently dedicated an entire issue to clinical trial developments in SCI. The single-focus supplement centers on the activities of the Reeve Foundation’s North American Clinical Trials Network (NACTN). Proving a therapy effective requires a framework for studying large numbers of patients. That’s where NACTN comes in, to provide expertise, infrastructure, and efficiency to run trials and evaluate their results. Funding is from the Reeve Foundation and the Telemedicine & Advanced Technology Research Center, US Army Medical Research and Materiel Command.

NACTN is a consortium of university hospital neurosurgical and neurorehabilitation teams. Said NACTN’s lead investigator, neurosurgeon Robert G. Grossman, (Methodist Hospital, Houston), “There can be no progress without partnerships, without collaborations, without alliance-building. Spinal cord injury is too difficult and too expensive to go-it-alone.” There are many challenges – organizational, regulatory, and financial – that can slow the process of conducting clinical trials. “The multicenter NACTN approach,” said Grossman, “will minimize delay.”

The journal supplement comprises 17 articles and several editorials. It is an academic collaboration between the journal and AOSpine North America (an international community of spine and orthopedic surgeons, neurosurgeons, academics, and other spine care professionals). The supplement was directed by Michael Fehlings, Professor of Neurosurgery at the University of Toronto and Medical Director of the Krembil Neuroscience Centre at the Toronto Western Hospital.

The 246-page peer-reviewed supplement includes papers related to identifying and evaluating different types of spinal cord trauma, including a characterization of the incidence and severity of acute complications after SCI, based on a large patient database maintained by NACTN. Also, the NACTN decision making process for choosing which therapies to study is evaluated.

A number of reviews summarize evidence for predicting neurological outcome in patients with both cervical and thoracic SCI, identifying gaps in research and making recommendations for future research. The discussion includes the development of newer graded assessments to better define the scope and extent of injury, and of recovery.

The supplement also features original clinical studies and review articles on current and potential drug-based therapies, including the drug Riluzole, which was tested in the first NACTN clinical trial, a Phase I safety study completed last year. The process has begun at NACTN (and other yet-to-be-named) centers to stage a Phase II Riluzole trial in 2013; the trial is planned as a collaboration with AOSpine North America.
Progress IN Research

By Martin E. Schwab and Anita D. Buchli

Spinal cord injuries affect hundreds of thousands of people in Europe and the United States, and around the world. Traumatic brain injuries are about ten times more common. Parkinson’s disease, Alzheimer’s and ALS add to the great social and economic burden caused by neurological disease or trauma.

Treatments that could restore lost functions to people with such injuries would radically change their lives and decrease the burden to their families and social environment. The economic interest to drug companies and health insurers seems obvious. Yet drug companies have withdrawn from neuroscience, more so than from any other disease area. Last year, Novartis closed its preclinical neuroscience research facility in Switzerland. Pfizer, GlaxoSmithKline and AstraZeneca had made similar moves. Merck and Sanofi are also cutting research on brain diseases.

Until recently, industry funded nearly half the budget for research and drug development for brain disorders. Its retreat has left an abyssal hole.

The reason for companies’ reluctance to pursue drugs for neurological disorders is fairly straightforward: their investments haven’t paid off. In the past 10-15 years, dozens of clinical trials for stroke neuroprotection – involving thousands of patients – have failed.

To get drug development going again, we must tackle the problems that have stalled it in the past by building a culture of interdisciplinary exchange to generate promising compounds, and by setting aside public funds to conduct small, well-designed clinical studies of those compounds. We realize that in such a tight funding situation, every field is asking for more. But given the extraordinary burdens neurological diseases cause, they must become more of a priority.

Drug companies have pulled out of neuroscience just as our understanding of brain plasticity has exploded. The antiquated view of the central nervous system as a hard-wired supercomputer has been overturned; the brain and spinal cord now appear as dynamic and adaptable biological systems.

Large injuries to the brain and spinal cord are not repaired spontaneously, causing lifelong impairment. But scientists have recently developed experimental interventions that enhance nerve-fiber growth and regeneration in animals with massive brain injury. In experiments with rats, mice and monkeys, researchers (in our laboratory and others) have induced regrowth of injured nerve fibers in the brain and spinal cord by suppressing growth inhibitors – enough for the treated animals to regain lost functions.

We and our colleagues at Novartis recently conducted a clinical trial in which people with spinal cord injuries received an antibody that counteracts the neural growth inhibitor Nogo-A. Other clinical trials to enhance repair of the spinal cord and brain are or will soon be under way. But progress is slow – the biotechnology company Geron, for example, abandoned a promising phase I trial of stem cells in spinal cord injury to concentrate instead on cancer.

A drug may be effective and still fail in a trial. One reason is that companies often look for the most broadly applicable drug – for example, ‘for all stroke patients’ – but disease conditions often differ among patients, resulting in huge variations in treatment responses. Another problem: the often crude clinical endpoints have missed small but meaningful treatment effects, such as improvements in hand, leg or bladder function. With novel approaches, we can do better.

More exchange should be fostered between basic and clinical scientists. When spinal cord researchers began organizing retreats and workshops to bring together basic researchers and clinicians, they saw firsthand how little each side knows about how the other works. The mutual lack of knowledge was huge; each side had a completely different language to...
describe the same scenario.

If researchers collaborate from the outset, they are more likely to produce a drug that works. For instance, they could establish a set of criteria to evaluate a particular therapy in both animals and humans, so that what seems to work in one is more likely to seem to work in the other. Clinicians are now standardizing observations of functional improvement so that they can spot subtle changes that would have gone unnoticed in the past.

Neuroscience faculties and medical centers must work together to establish research consortia and networks that unite basic and clinical scientists. Already, studies of spinal cord injury are more focused now that the two sides are communicating – some basic researchers have begun using clinical criteria for functional improvement.

We can’t just throw money and resources at the problem; we must use them wisely. Instead of investing billions in one drug, let’s spread funding among smaller, proof-of-concept trials for compounds with good preclinical evidence. By focusing on well-selected populations (with tens of patients, not hundreds) and concentrating on a few centers, such trials would cost a few million dollars rather than the $50 million or more needed for one large trial. If smaller trials can bring a promising compound to an advanced stage, industry may then be willing to take it to market.

And, because the pharmaceutical industry isn’t ready to invest in early-stage research in neurological diseases, we must turn to other sources. Insurance companies spend up to $2 million for each patient with a spinal cord injury – a drug that could lower a patient’s disability would save insurers huge amounts. In 2009, the top five U.S. health insurers earned more than $12 billion; investment of even a small percentage of these profits in research could result in a true win–win situation.

In 2011, a report commissioned by the European Brain Council found that, in terms of health-care costs and lost productivity, brain disorders are a greater socio-economic burden than cancer, cardiovascular diseases and diabetes combined. Yet in 2005, research funding for cancer and neurological diseases was roughly equal. More than half of that total comprised private funding; now that drug companies have shifted focus, cancer funding is likely to eclipse that of neuroscience. Funding agencies must revise their budgets to reflect the immediate and future needs of our society.


Martin E. Schwab, Ph.D., is a member of the Reeve Foundation International Research Consortium on Spinal Cord Injury. He and Anita D. Buchli, Ph.D., are at the Brain Research Institute of the University of Zurich. This article originally appeared in the comment section of the journal Nature.

**BLOCK THAT NOGO: SCHWAB LAB IDENTIFIES BARRIERS TO REGENERATION**

The Martin Schwab lab in Zurich pioneered the notion that repair of spinal cord nerves is blocked by a molecule found in the lining (myelin) of axons. His lab identified the inhibitory molecule (called Nogo) then went one better – they also found a way to neutralize it (with an antibody called anti-Nogo). This has resulted in several experimental treatment strategies aimed at enhancing the limited recovery from spinal cord or brain injuries. Several experimental anti-Nogo treatments have shown beneficial effects in animal models of spinal cord injury.

Schwab’s recent publications report that blocking Nogo results in significant functional recovery of locomotion or skilled forelimb reaching after spinal cord or stroke in rats and monkeys. Novartis completed a Phase 1 human safety trial for anti-Nogo last year; results have not been published. His group continues to study the optimal time window for successful anti-Nogo treatments; in rodents, spinal cord nerve fibers regenerated over several millimeters after acute or one-week-delayed treatments, but not as well when the antibody treatment was started with a delay of two weeks.
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Harkema....
(continued from page 5)

no way to measure that. So what is that tract that’s still remaining? If we knew that, maybe that’s where a regenerative therapy could focus.

Q. When does this become clinically relevant?
A. The question that needs to be posed to all the stakeholders is this: Look at the daily lives of people with spinal cord injury; if you can change one of the consequences, is that worth the translation; is that worth the investment? For me, having the privilege of interacting with people living with spinal cord injury every day, my answer is ‘Yes.’ If we could just maintain normal blood pressure in these individuals, that would represent an immense change in health and quality of life. Cardiovascular function in people with cervical injuries, it is a really life-limiting situation. Their pressures are dangerously low and they can become so hypotensive that they get dizzy, lose consciousness. Can we use an epidural stimulator just to maintain blood pressure at a normal level; is that incremental change worth the investment?

For our young men in the epidural stimulation program, one of the things that’s most important to them is that their legs look ‘normal’ again. Is that clinically relevant? Well, think about it. People with spinal cord injury have a higher incidence of cardiovascular problems, a higher incidence of diabetes, a higher incidence of metabolic deficiencies, and why is that? It’s related to muscle atrophy. If you can maintain muscle mass you lower chance of developing many other complications.

If we look at it from a cost-benefit ratio, what’s the cost of cardiovascular disease to the healthcare system, and to an individual? What’s the cost of a fracture? What’s the cost of a pressure sore to the healthcare system? I’m a little biased. I think the primary stakeholder is the person with the spinal injury. In my perfect world they get to make the decision; but that’s not reality. If you look at this from the point of view of caregivers, or the insurance companies – I think there is value-added to all the stakeholders in starting to translate these therapies now.
IT’S NOT WHEN YOU FINISH. IT’S WHY.
A spinal cord injury can happen in a split second—to anyone at any time. Nearly 5.6 million Americans are living with paralysis, of whom 1.3 million are living with spinal cord injury.

The Reeve Foundation is the source for people affected by paralysis, offering expert information and programs to advance spinal cord injury care and cures. We believe in empowering individuals and families affected by paralysis with the best knowledge, resources, support and community.

Use just one hour this season to make a positive and lasting difference for our community members.

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