

Brain Spine Interface to Restore Walking After Spinal Cord Injury

Henri Lorach^{1,2,3,10}, Andrea Galvez^{1,2,3,10}, Valeria Spagnolo^{1,2,3}, Felix Martel⁴, Serpil Karakas⁴, Nadine Interling^{1,2,3}, Molywan Vat^{1,2,3}, Olivier Faivre⁴, Cathal Harte^{1,2,3}, Salif Komi^{1,2,3}, Jimmy Ravier^{1,2,3}, Thibault Collin^{1,2,3}, Laure Coquoz^{1,2,3}, Icare Sakr^{1,2,3}, Edeny Baaklini^{1,2,3}, Sergio Daniel Hernandez-Charpak^{1,2,3}, Gregory Dumont^{1,2,3}, Rik Buschman⁶, Nicholas Buse⁶, Tim Denison^{6,7}, Ilse van Nes⁸, Leonie Asboth^{1,2,3}, Anne Watrin⁹, Lucas Struber⁴, Fabien Sauter-Starace⁴, Lilia Langa⁵, Vincent Auboiron⁴, Stefano Carda², Stephan Chabardes^{1,5}, Tetiana Akseno⁴, Robin Demesmaeker^{1,2,3}, Guillaume Charvet^{1,11}, Jocelyne Bloch^{1,2,3,11} & Grégoire Courtine^{1,2,3,11}

¹ NeuroX Institute, School of Life Sciences, Ecole Polytechnique Fédérale de Lausanne (EPFL), Geneva, Switzerland. ² Department of Clinical Neuroscience, Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), Lausanne, Switzerland. ³ NeuroRestore, Defitech Center for Interventional Neurotherapies, EPFL/CHUV/UNIL, Lausanne, Switzerland. ⁴ University Grenoble Alpes, CEA, LETI, Clinatec, Grenoble, France. ⁵ University Hospital, Grenoble, France. ⁶ Medtronic, Minneapolis, MN, USA. ⁷ Department of Engineering Science, University of Oxford, Oxford, United Kingdom. ⁸ Department of Rehabilitation, Sint Maartenskliniek, Nijmegen, Netherlands. ⁹ ONWARD Medical, Lausanne, Switzerland. ¹⁰ These authors contributed equally. ¹¹ These authors jointly supervised this work. ✉ e-mail: guillaume.charvet@cea.fr, jocelyne.bloch@chuv.ch, gregoire.courtine@epfl.ch

Introduction: To walk, the brain delivers executive commands to the neurons located in the lumbosacral spinal cord. While the majority of spinal cord injuries does not directly damage these neurons, the disruption of descending pathways interrupts the brain-derived commands that are necessary for these neurons to produce walking. The consequence is permanent paralysis. Here, we hypothesized that a digital bridge between the brain and spinal cord enables volitional control over the timing and amplitude of muscle activity, restoring a more natural and adaptive control of walking in one participant with chronic spinal cord injury.

Material Methods and Results: To establish this Brain-Spine Interface (BSI), we integrated two fully-implanted systems that acquire wirelessly the electrocortical activity (ECoG), decode motor intentions in real time and stimulate the lumbosacral spinal cord to elicit the corresponding movements. ECoG implants consist of an 8-by-8 grid of 64 electrodes^{1,2}. ECoG signals are sampled at 586Hz per channel. A decoding pipeline extracts temporal, spectral and spatial features embedded in the ECoG signals related to the intention to move. These features are then fed into the decoding algorithm that predicts the attempts to move the lower limbs based on a recursive exponentially weighted Markov-switching multi-linear model algorithm³. To support the control of lower limb movements, the outputs of the model are encoded into updates of joint-specific stimulation programs that are constrained within pre-established functional ranges of amplitudes. These commands are delivered to the spinal cord through the ACTIVA RC[®] implantable pulse generator⁴.

We first tested this BSI during voluntary elevations of the foot while standing. After only 5 minutes of calibration, the BSI supported continuous and intuitive control over the activity of hip flexor muscles, which allowed the participant to achieve a fivefold increase in muscle activity compared to attempts without the BSI. Similarly, up to 6 independent joint movements could be independently controlled with a single model. We provided the same configuration to support walking with crutches. The BSI enabled continuous, intuitive and robust control of walking. When the BSI was turned off, the participant instantly lost the ability to perform any step despite detected attempts to walk from the modulation of cortical activity. Walking resumed as soon as the BSI was turned back on. The participant completed 40 sessions of neurorehabilitation that involved walking with BSI, single-joint movements with BSI, balance with BSI, and standard physiotherapy. The participant exhibited improvements in all the conventional clinical assessments such as the 6-minute walk test, weight-bearing capacities, timed up and go, Berg Balance Scale, and walking quality assessed. Finally, we designed a system that could be operated by the participant without any assistance. This system includes a walker equipped with an integrated case that embeds all the components of the BSI.

Discussion: These results demonstrate that a fully implanted BSI can restore voluntary motor control over previously paralysed leg muscles. Additionally, they suggest that establishing a continuous link between the brain and spinal cord promotes the reorganization of residual neuronal pathways that link these two regions under normal physiological conditions.

Significance: This proof of concept in one human participant augurs a new era in the treatment of motor deficits due to neurological disorders. We anticipate that the approach is generalizable to a broad population of patients and could even be applied to restore upper limb function after cervical spinal cord injury or stroke.

References:

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