A brain spinal interface to alleviate gait deficits after neuromotor disorders

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Various neurological disorders disrupt the communication between supraspinal centers and the spinal circuits that control lower limb movements, which leads to a range of motor disabilities. Here, we introduce a brain spinal interface whereby cortical dynamics directly trigger electrical spinal cord stimulation protocols to adjust lower limb movements in freely behaving monkeys. Two healthy rhesus macaques were implanted with an epidural spinal electrode implant that was tailored to access flexor versus extensor motor pools of the left and right lower limbs. The spinal implant was connected to a Medtronic Activa RC stimulator with a modified firmware enabling real-time control over multiple sites of stimulation via Bluetooth communication. A 96-microelectrode implant was inserted into the leg area of the left motor cortex to monitor broadband neuronal modulation via wireless data transfer. We built a linear discriminant analysis (LDA) decoder that predicted bilateral foot off and foot strike events based on cortical dynamics with an accuracy reaching up to 99% over several minutes of continuous locomotion. We next interfaced these motor predictions with control algorithms that updated the location, timing, and frequency of electrical spinal cord stimulation based on the desired locomotor movements. This brain spinal interface allowed the monkeys to enhance the degree of flexion versus extension of their left and right lower limbs during continuous locomotion without disrupting the natural dynamics of gait movements. The decoder anticipated the initiation and end of locomotion, turning on and off the specific electrodes with the appropriate timing based on the detected intention to walk or rest. We integrated technologies that have been approved for use in humans to demonstrate the feasibility of interfacing leg-area cortical signals

with a highly selective spinal neuroprosthesis to alleviate gait disorders and enhance neurorehabilitation after neurological disorders.

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