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Neural population dynamics in premotor, motor and somatosensory cortices during locomotion in primates

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While movement may seem effortless, the underlying processes orchestrated by the nervous system are remarkably complex. From the central nervous system to the muscles, motor information must be well coordinated to elicit an appropriate movement. Although our knowledge of motor control has drastically increased, the precise role of cortical areas is still debated. For instance, locomotor gait (i.e. locomotion) is thought to be governed by automated neural circuitry residing within the spinal cord, or within the brain stem, while cortical areas may only adapt the adjustment of steps during walking on rugged terrain. In this context, it is important to better understand the role of different cortical area structures underlying the production of locomotor behavior. New findings could result in improving available treatments or in developing new therapeutic strategies to help patients with motor disabilities, such as spinal cord injury, stroke, or Parkinson's disease.

Nonhuman primates represent an adequate model to study the role of cortical areas during locomotion due to their advanced sensorimotor attributes which enable highly skilled capabilities. We thus trained animals to walk on a treadmill, on a flat surface, on an uneven horizontal ladder, and over stairs and obstacles. To study cortical dynamics during locomotion, monkeys were implanted with intra-muscular electrodes, as well as multi-electrode arrays in the premotor, primary motor, and somatosensory cortices. The wide range of locomotor behaviors revealed specific neural dynamic patterns varying along the rostro-caudal axis: from premotor, to primary motor, to somatosensory cortices. These neural dynamics were confined within a low-dimensional neural manifold: the locomotion subspace. The locomotion subspace can reliably predict locomotor-related events that could be used to trigger spinal cord stimulation at the lumbar segments in order to alleviate gait deficits after tetraplegia.

Jury:

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