Higher Nervous Control of Quadrupedal vs. Bipedal Locomotion in Non-Human Primates; Common and Specific Properties

S. Mori

National Institute for Physiological Sciences, Okazaki 444-8585, mrsgm@nips.ac.jp

We acquire a novel capability of walking bipedally according to a genetically designed program. Based on such a program, we postnatally develop our musculoskeletal system and its control system so as to elaborate bipedal (Bp) standing and Bp walking. The musculoskeletal system comprises of multiple motor or movement segments such as head, neck, trunk, fore- and hind-limbs, each segment having a number of degree of freedom [1]. The locomotion control system is the central nervous system (CNS) comprised of the cerebrum, basal ganglia, cerebellum, brainstem and spinal cord. Neural circuitries functionally uniting each of them also develop postnatally with maturation of individual CNS component. Spinal motoneurons (MNs) integrate command motor signals descending from the supraspinal structures and ascending signals arising from the motor segments, and in turn send out final motor outputs to the skeletal muscles of the motor segments [2].

During Bp standing and Bp walking, changes in body configuration are first registered by both the labyrinthine and proprioceptive receptors embedded in the motor segments. Changes in the external world are received by distant receptors, such as eyes and ears [2]. By continuous reception and processing of multi-modal interoceptive and exteroceptive afferent signals by daily practice and experience, we can build up 'locomotor memory' and/or 'internal reference'. With the use of this internal reference, we compare the body's moment-to-moment configuration relative to the immediate and distant environment. Although the movements of Bp standing and Bp walking demonstrate that there had been a transformation in the operation of the neural machinery of the CNS and reconfiguration of the musculoskeletal system [3], far fewer studies have been undertaken from a movement neuroscience perspective, and our knowledge of the neuronal machinery involved in the control of Bp standing and Bp walking is still inadequate.

To better understand the critical CNS mechanisms involved in the control of Bp standing and Bp walking, we need a non-human primate model, wherein not only kinematic and neurophysiological studies but also non-invasive imaging studies are feasible. With such multidisciplinary approach, causal relationships between CNS mechanisms and control mode of multiple motor segments could be elucidated. For this purpose, we have recently developed a new monkey model, Japanese monkey (*M. fuscata*), which walks either quadrupedally (Q) or bipedally on the surface of a moving treadmill belt. Our preliminary study using PET (Positron Emission Tomography) has already revealed that the neuronal activity of the primary motor cortex (M1), supplementary motor area (SMS), visual cortex, and cerebellum increased in parallel, with some intriguing differences between Bp walking and Qp walking in a single monkey [4]. Chemical inactivation of the M1 and SMA has resulted in focal and general impairments in the movements of motor segments specific to the inactivated cortical sites [4].

Based on the results we have obtained from this model animal, I will introduce and discuss four major aspects relating to the elaboration of Bp standing and Bp walking: (a) our concept of locomotor control CNS mechanisms including anticipatory and reactive control mechanisms, (b) emergence, acquisition and refinement of Bp walking in juvenile Japanese monkeys, (c) common and different control properties of Bp and Qp walking, and (d) similarity and differences in the kinematics of lower limbs in Bp walking monkey and the human.

Reference

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