Useless Hand Syndrome and Astereognosis in Multiple Sclerosis

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Useless hand syndrome (UHS) by Oppenheim, comprising clumsiness of complex finger movements and loss of manual dexterity, arises from cervical cord lesions in multiple sclerosis (MS) [1]. From Oppenheim's report, UHS has been attributed to impaired proprioception, resembling the nature of sensory ataxia [1,2]. In fact, T2 MRI showed high cervical lesions (C2-C4), consistently involving the posterior cord at the C3 to C4 level, in all of our patients with MS and UHS. However, I do not necessarily support the above-mentioned mechanism underlying UHS, because UHS was not associated with disturbance of deep sensations in some cases. As reported previously, UHS might be attributable to astereognosis rather than sensory ataxia, suggesting a disorder of sensorimotor integration [3]. While posterior cord lesions cause both of UHS and sensory ataxia, the underlying mechanism appears to differ, at least in part. In this regard, concurrent sensory disturbances may be helpful in elucidating the underlying mechanism of UHS. In all of our patients, stereognosis was most severely disturbed, whereas disturbance of other combined sensations such as two-point discrimination and graphesthesia ranged from severe to moderate. Combined sense was not necessarily impaired in parallel with deep sense.

Stereognosis is related to dynamic tactile process, called active touch, involving active sense of moving of fingers. The loss of moving sensations can result in impairment of manual dexterity and stereognosis [4]. Although fibers mediating position, vibration, and kinesthetic sensations enter the spinal cord together and ascend in the posterior column, the physiology of sensory modalities is different from each other [5]. Compared with static joint position sense, dynamic tactile process conveys more successive and integrative information [4,6,7]. Furthermore, dynamic moving sense may be less likely to be transmitted than static position sense in demyelinating lesions of MS. Activity-dependent conduction block, failure to transmit high-frequency impulses, can occur in demyelinating lesions in the central nervous system, which would explain why impulses mediating sensations of dynamic moving of fingers are more substantially blocked than those mediating static position sensations of fingers in MS [8]. Following posterior cord lesions, UHS and astereognosis may therefore appear with or without sensory ataxia. Because MS and related disorders are frequently associated with pyramidal tract signs or disturbance of deep sense, it is not easy in many instances to differentiate UHS from paresis or sensory ataxia [3,9,10]. Careful examination of combined sensations is helpful in the local diagnosis of patients with MS and impaired dexterity. Clumsy hand with astereognosis in patients with MS suggests high cervical posterior cord lesions. It is concluded that the high cervical posterior cord plays an important role in the sensorimotor integration needed to execute complex finger movements.

References


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