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Re-establishing the disrupted sensorimotor loop in deafferented and deefferented people: The case of spinal cord injuries

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ABSTRACT

Acting efficiently in the world depends on the activity of motor and somatosensory systems, the integration of which is necessary for the proper functioning of the sensorimotor loop (SL). Profound alterations of SL functioning follow spinal cord injury (SCI), a condition that brings about a disconnection of the body from the brain. Such disconnection creates a substantial deprivation of somatosensorial inputs and motor outputs. Consequent somatic deficits and motor paralysis affect the body below the lesion level. A complete restoration of normal functions of the SL cannot be expected until basic neuroscience has found a way to re-establish the interrupted neural connectivity. Meanwhile, studies should focus on the development of technical solutions for dealing with the disruption of the sensorimotor loop. This review discusses the structural and functional adaptive reorganization of the brain after SCI, and the maladaptive mechanisms that impact on the processing of body related information, which alter motor imagery strategies and EEG signals. Studies that show how residual functions (e.g. face tactile sensitivity) may help people to restore a normal body image are also reviewed. Finally, data on how brain and residual body signals may be used to improve brain computer interface systems is discussed in relation to the issue of how such systems may help SCI people to re-enter the world and interact with objects and other individuals.

1. Introduction

Normal sensorimotor circuits can be considered as loops integrating efferent information from the brain to the peripheral muscles with afferent signals (e.g. visual, auditory and proprioceptive) in order to compare the sensory feedback of an action with the desired motor command. Alterations of the sensorimotor loop induce a more or less severe inability to act efficiently in the world and a dramatic reduction of the patients' quality of life. Deficits of the sensorimotor loop are conspicuously present in people who sustain spinal cord injuries (SCI), as a consequence of a variety of causes such as trauma (e.g. road crashes, accidental falls), or infectious or inflammatory diseases. SCI typically prevents sensory and motor information from travelling to and from the brain below the injury level. Worldwide incidence of SCI is 40–80 new cases per million population per year (Bickenbach et al., 2013, see Lee et al. (2014) for an estimate of the global incidence of traumatic spinal cord injury). The recovery of locomotion in these patients is one of the main targets for current therapeutic approaches considering that the chances of regaining functional autonomy depend on the severity of the lesion (Scivoletto et al., 2014). The impact of sensory–motor loop alterations depends on the level and completeness of the lesion. Injury at cervical spinal cord levels C2–C7 leads to tetraplegia (or quadriplegia); a clinical condition with impaired sensory–motor functions in both upper and lower limbs. Injuries below the seventh cervical spinal cord segment lead to paraplegia, a condition where somatosensory and motor deficits affect only lower limbs. Moreover, SCI can induce a complete interruption of body–brain input–output communication, with little or no sensorimotor functions below the lesion level (see Kakulas and Kaelan (2015) for a description of the discomplete SCI syndrome' and Awad et al. (2015) for the report of a patient with functional somatosensory activation after a diagnosis of complete SCI), or an incomplete interruption, with some functions retained below the level of the injury (Kirshblum et al., 2011). The post-lesion sensorimotor loss is often accompanied by secondary complications involving the circulatory,

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neuromuscoloskeletal and respiratory systems (Bickenbach et al., 2013). These, in turn, dramatically affect quality of life for people with SCI (Williams and Murray, 2014; Rognoni et al., 2014; Rodrigues et al., 2013). Knowledge about how SCI affects brain signals, and how the brain reorganizes processing of sensory and motor information (Nardone et al., 2013), is fundamental for the development of applications based on non-invasive brain computer interfaces (BCIs). This system reads brain activity and real-time translates it into computer commands that enable the user to control a robotic or a neuro-prosthetic device (Wolpaw et al., 2002), a wheelchair (Leeb et al., 2007) or a virtual arm (Perez-Marcos et al., 2009). In the present review, existing evidence for structural and functional adaptive reorganization of the brain after SCI is examined, both at rest and during action-related tasks. Moreover there is a focus on studies showing that maladaptive plasticity may affect the processing of body related information. Studies that use BCI for motor and communication purposes in people with SCI are also of interest here; to examine how brain plasticity may represent an auxiliary tool to close the disrupted sensorimotor loop (e.g. providing facial sensory feedback to simulate a touch over the deafferented hand). Also, the challenge of creating BCI systems that use spared sensory afferences (e.g. proprioceptive and auditory feedback) to improve the control of an external device and to elicit the feeling that the remote effector is part of the body (Lebedev and Nicolelis, 2006).

2. Adaptive plasticity following SCI

2.1. Anatomical and neurophysiological changes of sensorimotor cortical circuits at rest

Recent functional and structural MRI studies show that in the early stages after SCI the motor and sensory cortices present significant anatomical changes; in particular the impairment of motor abilities, which is reflected in a decrease of gray matter volume in the primary motor cortex (Hou et al., 2014). Moreover, compensatory changes in the brain occur during the first year post-SCI, with a better recovery associated to an increase in cortical activation (Sabre et al., 2013) and greater corticospinal tract integrity in the acute post-lesion period (Freund et al., 2013). It is worth noting that short and long-term plasticity mechanisms (Tinazzi et al., 2000; Rosso et al., 2003, Freund et al., 2011, 2013; Corbetta et al., 2002) are responsible for plastic reorganization processes that occur at cortical and subcortical levels (Moore et al., 2000; see Kambi et al. (2014) for the relation between the brainstem and somatosensory plasticity in monkeys).

Crawley et al. (2004) measured morphometric changes in the human brain following complete and incomplete cervical SCI. Using both manual and automatic voxel-based morphometry (VBM) procedures, no differences were found between the SCI and healthy groups in gray and white matter volume within primary motor cortex (M1). This suggests that the reorganization process of the motor outputs following SCI is reflected more in functional rather than anatomical changes. Similarly, Jurkiewicz et al. (2006) observed no atrophy in M1 but a reduction of gray matter within the primary sensory (S1) and parietal cortices in a group of SCI patients relative to healthy controls. However, Wrigley et al. (2009) reported a significant reduction of gray matter in the left M1, medial prefrontal and the anterior cingulate cortices in people with complete thoracic SCI. A possible explanation for this seeming discrepancy may be attributed to the different characteristics of the SCI groups (see Supplementary Table S1). In line with Wrigley et al. (2009), two recent studies showed that gray matter atrophy affecting M1, S1, the supplementary motor area (SMA) and thalamus (Hou et al., 2014) occurs in the early stages after injury, and a progressive volume reduction of the gray matter within M1 (Freund et al., 2013) is observed within the first year after acute SCI. These results may indicate that the post-lesional reorganization implies anatomical changes in the human sensorimotor system, that occur from the early phase after SCI (see also Zhu et al. (2015) for reduced spontaneous brain activity in SCI patients). Although cortical plasticity may, in the long term result in maladaptive changes (e.g. neuropathic pain; Finnerup, 2013) and modify the patients' body representation (Fuentes et al., 2013, Scandola et al., 2014, Tidoni et al., 2014a, 2014b, 2014c, 2014d, Lenggenhager et al., 2012), such reorganization processes may be crucial to preserve sensorimotor functions and improve rehabilitation outcomes. The notion of a widespread reorganization of cortical and subcortical areas is also supported by a study with single photon emission computed tomography (SPECT), that revealed a reduction of cerebral blood flow in cortical and subcortical movement-related areas (Çermik et al., 2006). Moreover, Electroencephalography (EEG) studies show a reduction of brain wave activity in the range of 8–13 Hz (Tran et al., 2004; Herbert et al., 2007) and an increase in the beta range (Herbert et al., 2007), indicating that the effects of deafferentation can be captured by dynamic indices of brain activity. Research based on Transcranial Magnetic Stimulation (TMS): a non-invasive tool to assess cortical shifts of limb representation (Freund et al., 2011; see also Nardone et al. (2014) for a review on TMS studies in SCI patients), allowed researchers to show that people with SCI are unable to actively inhibit muscle activation (Roy et al., 2011). In a similar vein, using Positron Emission Tomography (PET) Roelcke et al. (1997) found that SCI patients exhibited a global reduction of glucose metabolism in the presence of increased activity in sensorimotor areas. This result suggests that the substantial deafferentation induced by SCI may alter the control of the inhibitory processes that regulate cortical activity. Overall, these studies indicate that following SCI, the reorganization process is reflected at both early and later stages within cortical and spinal areas. While the post-SCI lesion anatomo-functional changes at rest index that the deafferented brain is still active, very little information on how this activity may influence the efficacy of external applications based on BCI is currently available.

2.2. Functional brain reorganization in SCI contingent upon action related tasks

Studies report that people with SCI may show differences in motor imagery compared to healthy controls (Fiori et al., 2014). For example, people with SCI and concomitant neuropathic pain do report increased painful sensations below the lesion level during motor imagery tasks (Gustin et al., 2008). The example of motor imagery is particularly relevant because it represents one of the most used methods for communicating and interacting with the world by mere cerebral control and is based on the conversion of motor imagery brain activity into computer commands for controlling artificial devices (Wang et al., 2012). It is worth noting here that understanding functional brain changes during action-related tasks in SCI is of great importance for optimizing BCI in people with sensorimotor disorders (see Section 4).

Information concerning functional changes of action-related cortical activity comes from EEG studies, showing that while readiness potentials (which reflects motor cortical activity during motor programming, Touzalin-Chretien et al., 2010) are similar in spinal cord injured and healthy people, movement-related brain potentials (which reflect brain activity associated with processes of movement preparation and movement execution) in SCI patients are similar to the cortical potentials of healthy people at rest (Castro et al., 2007).

In addition, the results of a high-resolution EEG study indicate

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