Disorder: Stroke
Title: Harnessing neuroplasticity after stroke
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Ischemic stroke is a devastating neurological disease; worldwide, it is the 2nd most common cause of death, second only to coronary heart disease, and the leading cause of disability amongst adults (Kolominsky-Rabas et al. 2001; Feigin et al. 2009). The disease is accompanied by an enormous economic burden, as approximately one third of patients who survive 6 months are dependent upon others to perform activities of daily living. Further, stroke is associated with a high degree of clinical variability, in terms of the cause, duration, localisation, severity and co-morbid conditions (Stinear 2010; Dimyan and Cohen 2011).

Ischemic stroke is the result of thrombotic or embolic occlusion of cerebral arteries, the middle cerebral artery being the most commonly affected site. Within minutes of occlusion and disruption in cerebral blood flow (CBF), a cascade of pathological events is initiated, ultimately resulting in cell dysfunction and death.

Despite the complex and extensive pathology of ischemic injury, patients who survive a stroke event undergo a degree of functional recovery. The brain has a unique ability to actually change itself in response to changes in behaviour, environment, stimulation and even brain injury. This process, known as neural plasticity underlies the processes of learning and memory and is thought to underpin recovery after stroke. Understanding neural plasticity and brain adaptation will lead to improvements in rehabilitation therapies aimed at improving functional recovery.

After a stroke, changes in cortical maps involving the voluntary control of movement pathway have been observed (Dimyan and Cohen 2011). During and immediately after a stroke, the brain is hyperactive and this may facilitate recruitment of adjacent, intact areas of motor cortex to compensate for loss of brain tissue. These changes reflect the scope of neuroplastic alterations in the brain, which ranges from adaptations at a cellular level, through to changes in neural networks and cortical maps.

Current treatments for stroke survivors are primarily aimed at improving and maintaining remaining function and therefore quality of life. Rehabilitation is started as soon as possible and usually involves physiotherapy and or speech therapy, depending on the functional deficits of the patient. The neurological basis of these therapies is basically that of neural plasticity. Current research is also directed at methods that can enhance neural plasticity.

Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive means of stimulating the brain. As many neurological disorders are thought to involve abnormal or dysfunctional neuronal activity, it is hypothesised that the therapeutic action of rTMS may occur through modulating and reversing abnormal activity and facilitating neuroplasticity.

Numerous clinical studies have investigated the safety and efficacy of rTMS treatment for a wide variety of conditions including depression, anxiety disorders including obsessive compulsive disorder, Parkinson's disease, stroke, tinnitus, affective disorders, schizophrenia and chronic pain.

In terms of stroke therapy, TMS has prognostic value in that the remaining function of damaged areas can be easily assessed. From a treatment standpoint, a number of research groups are investigating rTMS as a means of promoting neural plasticity. This could occur via multiple pathways, by improving synaptic connections (Rodger et al. 2012), altering neurotransmitters (Strafella et al. 2003) and changing cerebral blood flow. (Speer et al. 2000) The effects of rTMS on inflammation are also being investigated (Bates et al. 2012).

Harnessing the neuro-plastic potential of the brain is an exciting avenue for therapeutic development for a number of neurological conditions, particularly stroke. These therapies are aimed at improving the brain's healing potential by altering brain structure from a cellular through to neural network level.

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