



The role of the contralesional hemisphere during the recovery after a stroke: a short review

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Background

Cerebral plasticity plays a critical role after damage to central nervous system (CNS), with functional reshaping that underlies the clinical recovery¹. Neural substrates of “post-lesional” brain plasticity are under intense study, since knowledge of this phenomenon can lead to an appropriate rehabilitation treatment and a successful functional recovery. After a stroke, among the mechanisms of cortical reorganization, there is the increase of activity in the contralesional hemisphere², as assessed by longitudinal studies using functional imaging and direct cortical stimulation.

The role of the unaffected hemisphere during recovery after a stroke is still debated. The aim of this study is to assess **how and when the contribution of contralesional hemisphere influences the functional recovery after a cerebrovascular event**.

Materials and methods

A search was performed through the database PubMed, considering the publications between 2004 and 2015. The following *key words* were used: “**stroke**”, “**recovery**”, “**contralesional**”, “**functional magnetic resonance imaging (fMRI)**”, “**transcranial magnetic stimulation (TMS)**”, with several search strings (Tab.1).

All studies concerning adults after their first ischemic or haemorrhagic stroke, within the first six months after the event, were collected.

Twenty-four studies were included and a qualitative analysis of the selected studies was performed (Tab. 2).

Search strings	Number of studies (597)
(contralesional* cortex) AND stroke[MeSH]	176
(contralesional* hemisphere) AND stroke[MeSH]	127
(contralesional* activity) AND stroke[MeSH] recovery	53
fMRI AND contralesional cortex AND stroke[MeSH]	103
fMRI AND contralesional hemisphere AND stroke[MeSH]	57
TMS AND contralesional cortex AND stroke[MeSH]	18
TMS AND contralesional hemisphere AND stroke[MeSH]	63

Tab. 1 – Pubmed search strings

24 studies 367 patients	• 1 study: exclusive activation of affected hemisphere (Park CH, 2011)
	• 1 study: change of the gray matter volume (Dang C, 2013)
	• 22 studies: activity in the contra-lesional hemisphere
	• 14 studies: bilateral activation (Golestani 2013, Huynh 2013, Jung 2013, Wei 2013, Rehme 20011b, Grefkes 2010, Dechaumont-palacin 2008, Grefkes 2008, Puh 2007, Loubinoux 2007, Butefish 2005, Carey 2004, Ward 2004, Tombari 2004)
	• 8 studies: only contra-lesional activation (Cazzoli 2012, Seniow 2012, Rehme 2011 a, Carey 2011, Carter 2010, Askim 2009, Nowak 2008, Koch 2008)

Tab. 2 – Studies subjected to qualitative analysis

14 studies → contralesional hemisphere activated during the sub-acute phase

(Golestani 2013, Huynh 2013, Wei 2013, Cazzoli 2012, Rehme 2011a, Carey 2011, Grefkes 2010, Carter 2010, Grefkes 2008, Nowak 2008, Koch 2008, Puh 2007, Loubinoux 2007 Ward 2004)

8 studies → contralesional hemisphere activated during both the sub-acute and chronic phases

((Jung 2013, Seniow 2012, Rehme 2011b, Askim 2009, Dachaumont-Palacin 2008, Butefish 2005, Carey 2004, Tombari 2004)

7 studies → ipsilesional hemisphere activated in chronic phase

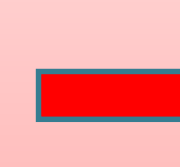
(Golestani 2013, Huynh 2013, Wei 2013, Askim 2009, Loubinoux 2007, Butefish 2007, Carey 2007)

In **7 studies**, contralesional hemisphere influences **positively** the functional recovery in acute and subacute phase.



→ Golestani 2013, Huynh 2013, Wei 2013, Rehme 2011a, Askim 2009, Dechaumont-Palacin 2008, Grefkes 2008

In **10 studies**, contralesional hemisphere influences **negatively** the functional recovery in subacute phase.



→ Jung 2013, Cazzoli 2012, Carey 2011, Grefkes 2010, Nowak 2008, Koch 2008, Puh 2007, Carey 2004, Ward 2004

In **5 studies**, the role of contralesional hemisphere is not specified during functional recovery.



→ Seniow 2012, Rehme 2011b, Carter 2010, Butefish 2005, Tombari 2004

Discussion

Most of the recent studies confirm that, in the acute phase after a stroke, the hyperactivity of the unaffected hemisphere appears to depend on the extension of the lesion, on the severity of the clinical injury and on the interval from the cerebrovascular event.

The underlying pathophysiological mechanism represents an adaptive compensation and may enhance functional recovery. **After the third month** after a stroke, the persistence of contralesional hyperactivity appears to be a maladaptive process, because it may slow down a functional recovery. Since the unaffected hemisphere is activated also when the ipsilesional limbs are moving, **the rehabilitation treatment should respect the different phases of cerebral plasticity and should avoid “overuse”**. After a cerebrovascular event, in fact, neurological deficits are caused not only by the lesion, but also by the same mechanisms that underlie functional recovery, such as diaschisis.

References

1. Daffau H. Brain plasticity: from pathophysiological mechanisms to therapeutic applications. Journal of Clinical Neuroscience 2006 Vol 13, 885–97.
2. Grafkes C. Ward N.S. Cortical reorganization after stroke: how much and how functional? The Neuroscientist 2014 Vol. 20 (1), 56 –70.

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