Editorial

iMedPub Journals
http://www.imedpub.com

Acta Psychopathologica
ISSN 2469-6676

2016
Vol. 2 No. 3: 31

DOI: 10.4172/2469-6676.100057

Abnormal Neural Circuits Model in Central Post-stroke Pain

Yufen Zhang[1,2], Sehui Ma[1,2], Yao Yi[1,2] and Lei Pei[1,2]

1 Department of Neurobiology, Huahzong University of Science and Technology, Wuhan, 430030, China
2 The Institute for Brain Research (IBR), Huazhong University of Science and Technology, Wuhan, 430030, China

Corresponding author: Lei Pei

peilei@hust.edu.cn

Lecturer and Researcher, Department of Neurobiology, Tongji Medical College, The Institute for Brain Research (IBR), Collaborative Innovation Center for Brain Science, Huazhong University of Science and Technology, Wuhan, China.

Tel: +86-27-83692630
Fax: +86-27-83692625

Citation: Yufen Zhang, Sehui Ma, Yao Yi, et al. Abnormal Neural Circuits Model in Central Post-stroke Pain. Acta Psychopathol. 2016, 2:3

Received: June 25, 2016; **Accepted:** June 26, 2016; **Published:** June 30, 2016

Keywords: Central post-stroke pain; Neural circuits

Editorial

Central post-stroke pain (CPSP) was first described by Dejerine and Roussy as a consequence of stroke-related lesions in the thalamus [1]. CPSP can develops after both haemorrhagic and ischemic lesions occurring at any level of somato-sensory pathway of the brain, including medulla, thalamus, and cerebral cortex. CPSP is characterized by either spontaneous or evoked unpleasant sensory described as allodynia, hyperalgesia, and dysesthesia, which severely affect the quality of life. The prevalence of CPSP has been reported to be 8–55%, which is due to different locations of the lesion in the brain. Evidences suggest that the occurrence of CPSP is particularly high after lateral medullary infarction or lesions in the ventroposterior thalamus [2]. In addition, lesions in other brain areas in the somatosensory pathway also result in CPSP [3-5].

Even though the mechanisms of CPSP remain elusive, CPSP has been considered to be maladapted reorganization of neural network after stroke [6]. Based on basic and clinical studies, the following various neural circuit models have been proposed to illustrate the abnormal network of CPSP in the brain [4, 6].

First, Spino-Thalamo-Cortical (STC) circuit model. Studies show hyperactivities of neurons in STC circuit after cerebral ischemic stroke. Spontaneous pain has been found to be due to hyper excitability in STC circuit [7], and neurophysiological examinations on CPSP patients showed abnormally increasing bursting activities in the thalamus [8]. Medications suppressing neuronal activities or inhibiting neural projecting fibers between Spino-Thalamo area or Thalamo-Cortical area were effective for CPSP alleviation [9].

Second, Cortical-Thalamic (CT) circuit model. Like STC circuit, hyperactivity in the CT circuit is one of the mechanisms of CPSP. This abnormal neural activity in the CT circuit is due to the disinhibition from the cortex, which called the “disinhibition theory” [10]. The sensory in central nervous system is modulated by a tonic balance between excitation and inhibition [11]. Stroke induced lesion in the CT circuit could interrupt this balance, thus cause abnormal sensations like pain in CPSP.

Third, Limbic-Amygdala-Insular cortex (LAIc) circuit model. Recently, using functional magnetic resonance imaging (fMRI) technique, clinical scientists have shown changes of activities in the LAIc neural network in chronic pain states of CPSP patients [12]. LAIc circuit is composed by amygdale and insular cortex in the limbic structures [13]. Damages in the LAIc circuit after stroke could produce both structural and functional abnormalities of the sensory neural network, which causes generation and development of CPSP.

Last but not least, Inter-hemispheric circuit model. The malfunctioning in inter-hemispheric circuit after stroke may be another pathogenesis of CPSP. Recent studies have shown that electrical stimulation of the contralesional hemisphere could suppress the activities in the epilesional hemisphere, which indicates that inter-hemispheric inhibition might be contributed to the pain suppression mechanisms in CPSP patients treated with electric stimulation [14, 15]. Therefore, electric brain stimulation could be choosed as an alternative treatment for CPSP patients.

In this editorial, we briefly summarized a few proposed CPSP neural circuit models contributing to the mechanisms of CPSP.

We conclude that abnormal structure (damage in the inhibitory neurons or neural fibers) and dysfunctions (hyper excitability) in these neural circuits play important roles in the generation of hyperalgesia and allodynia in CPSP patients. Applying newly developed single synaptic tracing technique with optogenetics on CPSP animal models, new specific neural circuits will be revealed, interventions or treatments targeting these abnormal circuits may be more effective for CPSP patients than traditional antipsychotic medicine. In spite of this, further investigation of network abnormalities in CPSP may shed light on the mechanisms and treatment of CPSP.

Acknowledgement

This work was supported by National Natural Science Foundation of China (81200863, 81571078).

References

1 Dejerine F, Roussy G (1906) Thalamic syndrome. Paris: Asselin et Houzeau.

2 Klit H, Hansen AP, Marcussen NS, Finnerup NB, Jensen TS (2014) Early evoked pain or dysesthesia is a predictor of central poststroke pain. Pain 155: 2699-2706.

3 O'Donnell MJ, Diener HC, Sacco RL, Panju AA, Vinisko R, et al. (2013) Chronic pain syndromes after ischemic stroke: PRoFESS trial. Stroke 44: 1238-1243.

4 Klit H, Finnerup NB, Jensen TS (2009) Central post-stroke pain: clinical characteristics, pathophysiology, and management. Lancet Neurol 8: 857-868.

5 Sprenger T, Seifert CL, Valet M, Andreou AP, Foerschler A, et al. (2012) Assessing the risk of central post-stroke pain of thalamic origin by lesion mapping. Brain 135: 2536-2545.

6 Hosomi K, Seymour B, Saitoh Y (2015) Modulating the pain network–neurostimulation for central poststroke pain. Nat Rev Neurol 11: 290-299.

7 Vestergaard K, Nielsen J, Andersen G, Ingeman-Nielsen M, Arendt-Nielsen L, et al. (1995) Sensory abnormalities in consecutive, unselected patients with central post-stroke pain. Pain 61: 177-186.

8 Lenz FA, Weiss N, Ohara S, Lawson C, Greenspan JD (2004) The role of the thalamus in pain. Suppl Clin Neurophysiol 57: 50-61.

9 Vranken JH, Dijkgraaf MG, Kruis MR, van der Vegt MH, Hollmann MW, et al. (2008) Pregabalin in patients with central neuropathic pain: a randomized, double-blind, placebo-controlled trial of a flexible-dose regimen. Pain 136: 150-157.

10 Craig AD, Bushnell MC (1994) The thermal grill illusion: unmasking the burn of cold pain. Science 265: 252-255.

11 Costigan M, Scholz J, Woolf CJ (2009) Neuropathic pain: a maladaptive response of the nervous system to damage. Annu Rev Neurosci 32: 1-32.

12 Baliki MN, Mansour AR, Baria AT, Apkarian AV (2014) Functional reorganization of the default mode network across chronic pain conditions. PLoS One 9: e106133.

13 Price DD (2000) Psychological and neural mechanisms of the affective dimension of pain. Science 288: 1769-1772.

14 Liu J, Qin W, Zhang J, Zhang X, Yu C (2015) Enhanced interhemispheric functional connectivity compensates for anatomical connection damages in subcortical stroke. Stroke 46: 1045-1051.

15 Volz LJ, Sarfeld AS, Diekhoff S, Rehme AK, Pool EM, et al. (2015) Motor cortex excitability and connectivity in chronic stroke: a multimodal model of functional reorganization. Brain Struct Funct 220: 1093-1107.