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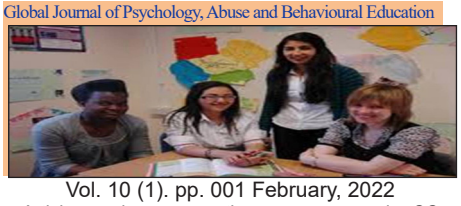

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Activity of brain during neurogenic pain

X She*

Department of Psychiatry, Peking University, Beijing, China
*Corresponding author. E-mail: sheshu@pku.edu.cn

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DESCRIPTION

The actual or potential tissue damage results in an unpleasant sensory and emotional experience referred to as Pain. Pain may be a diverse phenomenon that results in the excitation and activation of several regions of brain. An outsized network of neurons within the thalamic and cortical areas of the brain functions because of central processing unit of pain. Several neuroimaging studies indicate that additionally to the associative cortical areas, several para limbic structures also are playing a big role within the overall experience of pain. Various parts of the brain fluctuating during the pain perception and thus called because the “pain matrix” include thalamus, somatosensory cortex (primary and secondary), medial and lateral prefrontal cortex, posterior and anterior insular cortex, basal ganglia, cerebellum, orbitofrontal cortex, premotor cortex, and posterior parietal cortex.

The dysfunction of peripheral and central nervous system gives rise to neurogenic pain. Neurogenic pain is perceived as discomfort without nociceptive stimulation from the periphery. The terms like neuropathic pain, central pain and differentiation pain all are summed up into a broader term referred to as “neurogenic pain”. Up to 25% of the patients visiting pain clinics suffer from clinical syndromes representing this sort of pain. Several concepts about the pathophysiology of neurogenic pain are discussed within the literature. There’s a dense and reciprocal linkage of certain neuronal structures or interconnection between thalamic and cortical areas of human brain. Furthermore, various studies have reported that the interruption within the normal functioning of Thalamo-cortical interaction is that the source of neurogenic pain. The alteration or slight change within the signal transmission of Thalamo-cortical pathways gives rise to “Thalamocortical dysrhythmia”, observed in patients exhibiting the symptoms of neurogenic pain. Moreover, it’s also suggested that the reticular nucleus of Thalamus inhibits the central lateral and ventro-posterior nuclei, which results in the imbalance between both the nuclei thus leading to neurogenic pain.

Multiple levels of system nervous are involved within the extensive and sophisticated process of pain transduction and perception. The cortical and subcortical distribution of neurogenic pain is examined using electrophysiological and neuroimaging studies. Brain’s responses to pain are identified within the literature through Functional Resonance Imaging (FMRI), Positron Emission Tomography (PET) and Magnet Encephalography (MEG). Magneto encephalography detects the physiological rhythm of the temporal region and is a clinical tool of neurophysiology. To work out the electrical activity of brain during neurogenic pain, various studies are conducted using Electroencephalography (EEG) and therefore the waveband power of alpha beta and theta waves during the method are discussed. EEG contains continuous and wide frequency spectrum with lower and upper limit range. Furthermore, differences between both the groups were observed within the range of delta band also but insignificant differences within the other frequency bands. The height values and significant differences within the theta band showed neurogenic origin of the pain within the patients suffering chronic pancreatitis.

This review has suggested that the neurogenic pain leads to the alterations within the electrical activity of brain as detected by EEG. Furthermore, the literature clearly shows that over activation of brain’s theta and beta waves, coupling between theta and beta frequency range, Thalamocortical interplay and significant differences within the ta frequency range among patients and controls are a number of the common features in the electrical activity of brain of the patients exhibiting neurogenic pain.