Medical Hypotheses 88 (2016) 34

Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

Correspondence

The low-frequency (delta and theta) oscillations model of hallucinations integrating neuronal mechanism of object representation, emotions, plasticity, memory and noise signal☆

The low-frequency oscillations model of hallucinations, proposed by Juszczak [1], is based on the observation that the magnitude of EEG slowing (1–8 Hz) is correlated with the frequency of occurrence of hallucinations in various neurodegenerative disorders and delirium. Furthermore, both hallucinations and increased delta and theta oscillations are present in healthy subjects intoxicated with the anticholinergic drugs. According to the model, pathological low-frequency oscillations may interfere with information processing in two different ways leading to hallucinations. First, pathological delta and theta oscillations may interfere with the mechanisms of short-term memory leading to the excessive activation of memory traces and to the intrusion of memories into consciousness. Second, cyclic fluctuations in neuronal excitability associated with the low-frequency oscillations may produce synchronized noise in discharges of neurons. Next, the noise signals propagate in neural networks storing representations of external objects. It is assumed that representations of emotionally relevant objects (e.g., spider, snake, man) are stored in networks with stronger synaptic connections than representations of emotionally neutral objects (e.g., tree, table). It is also expected that stronger synaptic connections increase the probability that the noise signal will activate the entire network. Therefore, the noise signal preferentially activates representations of emotionally relevant objects. Such noise-induced activation can be next reinforced by attention leading to hallucinations.

This model has been considered too speculative and therefore it has been published out of the mainstream of biomedical literature [1]. However, there are a growing number of data supporting the hypothesis. First, it has been confirmed that the oscillatory activity is not merely a byproduct of neuronal activity but it has a causal effect on perception in healthy subjects [2,3]. Second, it has been found that noise signals are correlated only within highly interconnected and feature-selective subnetworks [4] and that strongly coupled neurons are easily activated by nonspecific stimulation in contrast to weakly coupled cells [5]. These findings confirm that the feature-selective networks constitute channels facilitating propagation of noise and that their excitability depends on the strength of synaptic connections. Finally, fear enhances coding of sensory information [6] and nonspecific stimulation brings the content of short-term memory into consciousness [7]. Therefore, these data show that each aspect of the hypothesis is viable.

Although there are a growing number of studies investigating the functional significance of the low-frequency oscillations in healthy subjects, there is still little interest in understanding contribution of aberrant oscillations to symptoms found in neurodegenerative disorders, delirium and drug poisoning. Therefore, it is worth drawing attention to this problem.

Conflict of interest

None.

References

[1] Juszczak GR. The low-frequency oscillation model of hallucinations in neurodegenerative disorders and in delirium. Iran J Med Hypotheses Ideas 2011;5:11.
[2] Busch NA, Dubois J, VanRullen R. The phase of ongoing EEG oscillations predicts visual perception. J Neurosci 2009;29:7869–76.
[3] Inyutina M, Sun HM, Wu CT, VanRullen R. Who wins the race for consciousness? Ask the phase of ongoing ~7 Hz oscillations. J Vis 2015;15:569.
[4] Ko H, Hofer SB, Pichler B, Buchanan KA, Sjostrom PJ, Mrsic-Flogel TD. Functional specificity of local synaptic connections in neocortical networks. Nature 2011;473:87–91.
[5] Okun M, Steinmetz NA, Cossell L, et al. Diverse coupling of neurons to populations in sensory cortex. Nature 2015;521:511–5.
[6] Gdalyahu A, Tring E, Polack PO, et al. Associative fear learning enhances sparse network coding in primary sensory cortex. Neuron 2012;75:121–32.
[7] Liao HI, Wu DA, Halelamien N, Shimojo S. Cortical stimulation consolidates and reactivates visual experience: neural plasticity from magnetic entrainment of visual activity. Sci Rep 2013;3:2228.

Grzegorz R. Juszczak *
Department of Animal Behavior, Institute of Genetics and Animal Breeding, Jastrzebiec, ul. Postepu 36A, 05-552 Magdalenka, Poland
* Tel.: +48 (22) 736 70 83; fax: +48 (22) 756 14 17.
E-mail addresses: g.juszczak@ighz.pl, gjuszczak@yahoo.com

☆ The study was supported by S.VI.2 intramural Grant.

http://dx.doi.org/10.1016/j.mehy.2016.01.010