TINNITUS NEUROFUNCTIONAL MODEL:

TINNITUS CLINICAL SYMPTOMS GENERATION AND MAINTENANCE

I.G, Toostani 1,4, H, Ekhtiari 3, N, Jangholi 2,4, A. C. B, Delbem 1

1 USP - Universidade De Sao Paulo, Sao Carlos, SP, Brazil; 2 UFABC - Universidade d0 ABC, Santo Andre, SP, Brazil; 3 ICSS - Iranian Institute for Cognitive Sciences Studies, Tehran, Iran; 4 FGS - Fanavaran Gostaresh Salamat, Tehran, Iran.

Introduction:

Our knowledge about Tinnitus has vigorously improved in current decay, but main mechanisms of changing perceiving neutral phantom sound into tinnitus distress have not yet been understood.

Non-causal association of Tinnitus clinical symptoms with neutral phantom sound specifications can be elucidated by learning operation.

Objectives:

Our suggesting for explaining negative symptoms related to tinnitus is based on top-down cognitive processes together with classical conditioning learning procedure when emotionally neutral conditioned stimulus (CS) is presented in association with an aversive unconditioned stimulus (US), and the conditioned response is the learned response to the previously neutral stimulus. Current models condition, or at least emphasize, the role of external events congruently paired with the causal physical event that originated the phantom perception(1).

To better understanding this phenomenon in tinnitus initiation and reinforcement, we proposed a new Neurofunctional model for tinnitus in early stages and its development.

Methodology:

We present empirical evidence from studies using neuroimaging, electrophysiology, brain lesion and behavioral techniques to support the model. This model represents an advance in our understanding of clinically-significant tinnitus symptoms and might eventually help to improve current treatments.

Conclusion:

Our detailed model included ascending auditory pathways, the thalamus (reticular, medial geniculate and dorsal nuclei), the limbic system, brain stem, basal ganglia, striatum, and the auditory and prefrontal cortices. We projected the mediating pathways of tinnitus distress incorporate with the convergence of the CS and US pathways in the medial geniculate
nucleus (MGN) and lateral nucleus of the amygdala (LA) from thalamic and cortical processing regions in the sensory systems that process the CS (auditory system) and US (emotional mechanism). The MGN projects into auditory cortices and LA. It also receives inhibitory input from thalamic reticular nucleus (TRN). The LA then connects with the central nucleus of amygdala (CE) directly and by the way of other amygdala regions. Outputs of the CE then control the expression of fear responses and related autonomic nervous system (e.g., blood pressure and heart rate) and endocrine (pituitary adrenal hormones) responses.

Functionally, we assume continuous or intermittent abnormal signal at the peripheral auditory system or midbrain auditory pathways. Depending on cognitive-emotional initiated value and the availability of attentional resources lead to conscious awareness perceiving of the neutral Tinnitus which can cognitively interpret as suspicious and contingents with emotional appraisal (US) such as feel of fear. The negative reaction (e.g. fear) is the learned response to neutral Tinnitus. Fear individually can trigger the feel of fear in a positive feedback loop, therefore continuing perceiving Tinnitus contingent with feel of fear reinforces the negative reaction. Furthermore Tinnitus negative reaction is being weakened by the both fearing in the absence of perceiving tinnitus, and perceive Tinnitus without triggering the feel of fear. Development of neuroplasticity in MGN (2), LA and auditory primary cortex are exhibited dynamic molecular neuron modification in brain which can cause reciprocal psychiatric comorbidities such as anxiety, stress, phobias and/or depression symptoms.