THE SOUND OF SILENCE – DECRYPTING THE NEURAL CODE OF TINNITUS

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It is widely believed that perceptual units may be represented in Hebbian cell assemblies, i.e., in neuronal connectivity that forms in response to the frequent co-activation of neurons. Hebb's prediction was that learning strengthens synaptic connectivity through LTP and subsequent morphological synaptic alterations. Strong internal connectivity within a cell assembly assures that it will be fully activated when a sufficient share of its elements is activated (ignited). Assuming that once activated, a cell assembly will remain active for a short period of time and that it contains at least some ten thousand neurons, its activation will produce a measurable magnetoencephalographic (MEG) signal. The perception of permanent sound, such as is the case for tinnitus, would require periodic reactivation of the respective cell assemblies. The cortico-limbic as well as the thalamo-cortical interplay produces slow waves, which may sustain such neocortical high frequency (gamma) activity and thus the ongoing perception of sound. We have shown that the spectra of tinnitus sufferers are indeed characterized by enhanced slow wave activity (Weisz et al., PloS Medicine, 2005). Subsequently, we investigated this hypothesis further by analyzing spontaneous magnetocortical activity recorded from subjects with and without perception of tinnitus (Weisz et al., 2006). Epochs were extracted around slow wave (2-7 Hz) activation peaks (determined via Hilbert transform) and submitted to wavelet analysis. A first important finding is that slow waves modulate high frequency activity in a resting state, reflected in an enhancement of gamma band activity. Overall the proportion of gamma to the entire power spectrum is enhanced in tinnitus subjects relative to normal hearing controls. If this altered high frequency activity is supposed to be the underlying neuronal correlate of perception, then it should also be related to the laterality of tinnitus. This was particularly the case for activity around 55 Hz, which was more dominant on the contralateral side of the ear in which tinnitus is perceived in unilateral cases and bilaterally equal in case of bilateral tinnitus.

We conclude that tinnitus arises, when transient or permanent hearing loss produce a type of cortical/subcortical reorganization that result in a permanent driving of cell assemblies with auditory representational functions.