Why do we need a First-Person Neuroscience?

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Higher brain functions are first-person inner sensations to which only the owner of the nervous system has access. Let's take the case of memory. We create internal sensations of partial sensory features of the item whose memory is getting retrieved in its absence. The internal sensation of memory changes in response to slight changes in cue stimuli, indicating that memory results from using units of internal sensations induced in response to the changing cue stimuli through some unknown computational process. By what mechanism and at what nodal points does the cue stimulus induce the units of internal sensations of memory of the associatively learned item?

During memory retrieval, only the cue stimulus is present, which has to evoke the first-person inner sensation of memory of the learned item. When the cue stimulus moves through its route, how can it induce inner sensations? For this to occur, it is expected that an interaction between the cue stimulus and learned item should have taken place along the route of the cue stimulus (at locations where the cue stimulus would have met the stimulus of the item whose memory is retrieved) at the time of learning. The only way this can occur is by a change occurring at the locations of convergence of those two stimuli at the time of associative learning**. We need to identify a specific location, a learning-inducible change and a mechanism by which the cue stimulus make use of the learning-induced change to induce first-person inner sensations.** Theoretical examination shows that it can be satisfied by the formation of a LINK between the postsynaptic terminals (dendritic spines or spines) at which the converging inputs arrive (Ref.1). This is expected to occur between the spines of different neurons as a rule (exceptions can be present) due to reasons that a) mean inter-spine distance is more than the mean spine diameter (Ref.2) and the associatively learned stimuli need to maintain their specific motor outputs, which requires different neuronal pathways after the convergence of the associatively learned stimuli. The cue stimulus can reactivate the LINK and activate the inter-LINKed postsynaptic terminal that was originally activated by the item whose memory is now getting induced.

Can activation of a postsynaptic terminal through the LINK (inter-postsynaptic LINK) from a lateral direction induce units of inner sensation? Since the postsynaptic terminal is CONTINUOUSLY getting depolarized all the time by neurotransmitter molecules, any instantaneous lateral activation of the postsynaptic terminal is expected to induce a "cellular hallucination" that the activity is arriving from its presynaptic terminal. This matches with the expectations for the first-person inner sensations of the mind (Ref.3). This takes place only at a narrow range of frequencies of the oscillating extracellular potentials. The lateral spread of activity through the LINK is expected to contribute to one of the vector components of this oscillation. In order to identify the sensory content of this cellular hallucination, it is required to make a first-person approach from the inter-LINKed postsynaptic terminal towards the sensory receptor level to identify the minimum sensory stimuli that can activate this inter-LINKed postsynaptic terminal. Several sets of sensory stimuli are expected to be induced at one inter-LINKed postsynaptic terminal. Since cue stimulus induces such units of inner sensations at several locations, it is required to discover the computational process that integrate these units of internal sensations to obtain the matching partial internal sensory feature for the associatively learned item. In summary, the main step in this process is the retrograde extrapolation from the inter-LINKed postsynaptic terminal towards the sensory receptor level. This constitutes an approach from a first-person frame of reference (Ref.4). These show that a first-person neuroscience is needed to study the mechanism of induction, computations of the units of inner sensations and the disease processes resulting from either loss (reversal of LINKs) or gain (non-specific LINKs) of function states.

References

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