**Research Interests: Reading neural codes**

**Current:** The brain contains billions of neurons. Its great computational power emerges because all of these neurons interact with each other. The consequences of these interactions results in many neurons encoding sophisticated and selective knowledge of the world, properties we apparently need for learning, recollecting and interacting with a sophisticated environment. This code exists as a series action potentials or ‘spikes’ produced by the neuron. Using microelectrodes that can record these spikes, we try to ‘crack’ the code of neurons in a structure called the hippocampus in rats learning and performing tasks requiring them to press keys in specific sequences. The hippocampus is the key brain structure associated with learning and memory. It appears to contain a running ‘map’ or where we are, what we just did, and what we plan to do next.


*Hippocampus* **26:** 601-623.


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Hippocampal activity can discriminate space and time. The polar plots labelled R1 to R9 show the selectivity of activity (expressed as a vector) associated with the rat responding to each of 9 responses in sequence. The rat’s ‘job’ was to press the right key (R1), the center key (R2) and the left key (R3) in order 3 times (R1-R9). Plots with only 1 black segment that form a radius indicate the activity produced a pattern of activity selective for *that* key in *that* order. Each possible response (there are 9) are coded by position on the circle. The 1st right, center and left responses basically produced unique patterns specific for that key in the 1st sequence. The vectors associated with R4 and R7 indicate the responses were selective, but did not discriminate between well between the 2nd and 3rd response to the right key. For perspective, if we randomly shuffled this same data, we get distributions such as the 3 ‘shuffled’ plots on the left. The discriminability disappears.
Past: I had previously engaged in reading neural codes in the early visual system, in a structure that receives directly from the retina known as the lateral geniculate nucleus (LGN). We presented short videos of animals at the zoo to awake monkeys, and then attempted to calculate backwards what the monkey was watching when the LGN spike occurred. Much of the data is still being analyzed. Whereas the rules by which spikes are produced appear pretty simple in the LGN, it turns out (like everything else about the brain) to be complex.


Past: Parkinson’s disease (PD) is a devastating disease of the motor system for which we have no cure. I have been involved with one approach to treating symptoms: deep brain stimulation (DBS). In most cases, it relieves many symptoms: tremor disappears, movement becomes easier and more fluid. When we record in the area targeted for placing the permanent stimulating electrode (subthalamic nucleus, STN), the activity is aberrant: dominated by high-frequency rhythmic activity. In some cases, we passed an array of 5 microelectrodes (4 horizontally displaced from 1 center electrode) through the STN as we passively moved the patient’s arm and/or leg. We found the expected ‘motor map’ of the contralateral musculature was not only fractured, but correlations among the different sites recorded were dynamic in a way that was far from random. It would appear that a consequence of the disease is that representations of the musculature which we assume are normally used for organizing coherent movement are completely perturbed (resulting in the observed tremor and rigidity [which is an elevation of muscle tone]). Thus, it makes sense that the high-frequency stimulation delivered by DBS used clinically is therapeutic (it effectively ‘turns off’ the brain area). No STN is better than a perturbed STN.


Weyand TG, Jacobs RU and Richter EO. Fractured and fluid somatotopy in the subthalamic nucleus of the Parkinsonian patient (in preparation).