Seizure focus or epileptic network? Role of inhibitory control in seizure spread

Abstract: The clinical practice of epilepsy surgery is founded on the idea that seizures arise from a definable seizure source that is restricted to a small neocortical or mesial temporal brain region. However, the appearance of EEG recordings often belies this simple model, and has led to an alternative proposal that seizures originate from distributed, potentially large-area networks. Previously, our group demonstrated that small seizure foci can be present despite apparent large-area EEG signatures, using a combination of animal studies and microelectrode recordings of human seizures. The key observation is that synaptically transmitted, excitatory barrages travel outward from the locus of seizing brain, triggering a powerful inhibitory response and creating an EEG signature that may be indistinguishable from that recorded from the seizing brain territory. Using this dual-territory model, we now explore the long-range dynamics of seizure spread and discuss implications for seizure localization in epilepsy surgery procedures.

Biography: Dr. Schevon is Associate Professor of Neurology in the College of Physicians and Surgeons at Columbia University. Prior to her medical training, she studied electrical engineering and computer science, and worked in VLSI design at AT&T Bell Laboratories. She attended medical school at the University of Pennsylvania, and completed an epilepsy fellowship at Columbia in 2004. Dr. Schevon’s research focuses on linking the cellular processes that occur during seizures to their expression in microelectrode and clinical EEG recordings, in order to inform the clinical process of seizure localization.