

Comparison of two forms of epileptiform activity in spinal cord

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Blockade of spinal inhibitory synaptic transmission with bicuculline and strychnine produces strong synchronous motor activity in the isolated neonatal rat spinal cord that can be blocked with the ionotropic glutamate receptor antagonists CNQX and APV. With the addition of K⁺ channel blockers TEA and 4-AP, a robust metabotropic glutamate receptor-dependent synchronous bursting re-emerges. Here we compared these two burst patterns using ventral root recordings and intracellular recordings of interneurons. Following addition of TEA/4-AP, bursts are generally of slower frequency and smaller amplitude than the bic/strych bursting, although the duty cycle (burst length/period) remains constant. We then compared latency measures of burst onset in 4 different motor roots during bic/strych induced activity. We identified delays consistent with a propagating burst that traveled in both rostrocaudal and side-to-side axes. Burst initiation site varied between animals and occasionally between burst cycles indicating that no specific neural population was responsible for its genesis. In comparison, in any given animal, the synchronous activity that re-emerged after TEA/4-AP was characterized by increased propagation delays and higher variability in burst order implying a more flexibly coupled network, though the site of burst genesis generally remained the same. Intracellular recordings from interneurons revealed; (i) neurons not recruited during bursting, (ii) neurons recruited only during stronger bursts, (iii) neurons recruited during both bic/strych and TEA/4-AP bursting, and (iv) neurons recruited during only one of the two burst types. We conclude that the spinal cord can organize paroxysmal network activity by multiple mechanisms, that subsets of neurons are associated with bursting and that different neurons can be recruited at different times. Studies are underway to further clarify the organization of these circuits.

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