PERSPECTIVE

# Saccadic premotor burst neurons in rhesus monkeys and histochemical correlates of their firing patterns

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**ABSTRACT**

Bursting, also known as burst firing, is a very diverse general phenomenon of neuronal activation patterns in the central nervous system and spinal cord in which periods of rapid action potential spiking are followed by periods of quiescence much longer than typical inter-spike intervals. Bursting is thought to be im-portant in the operation of robust central pattern generators, neural code transmission, and some neuropathologies such as epilepsy. Bursting research, both directly and in relation to how it participates in other neural phenomena, has been very popular since the beginnings of cellular neuroscience and is closely related to the fields of neural synchronization, neural coding, plasticity, and attention.

**Key Words:** *Neural synchronization*

## INTRODUCTION

Bursts are named according to the number of discrete action potentials they contain: a doublet is a two-spike burst, a triplet three, and a quadruplet four. Bursters are neurons that are intrinsically prone to bursting behavior, and this tendency to burst may be a result of the environment or the cell's phenotype. Bursting behavior of brainstem premotor Burst Neurons (BNs) is required for saccade initiation and calibration. Voltage-gated potassium channels, low-voltage-activated calcium channels, and Hyperpolarization-Activated Cyclic Nucleotide-Gated (HCN) channels are all important regulators of bursting in neurons. As a result, it was hypothesised that ion channels with fast kinematics are required for BN firing patterns and rapid saccade velocities. Ion channel expression patterns, on the other hand, have yet to be confirmed. Confirmation would not only validate the neuromimetic model predictions for saccade generation in the brainstem, but it would also support current views that channelopathies can cause saccade disorders in humans. In both BN populations, we found high expression of Kv channels, which allow rapid firing, as well as HCN1 and 2, which allow post-inhibitory rebound bursting.

Furthermore, in terms of calretinin immunoreactivity, PGD was discovered to host multiple neuron groups. Our findings provide histochemical evidence to support models that post-inhibitory rebound promotes bursting in BNs. Furthermore, our findings support the idea that histochemical examination of functional groups within the brainstem saccadic circuitry can provide information about electrophysiological firing properties. This breakthrough contributes significantly to the understanding of channelopathies in saccadic disorders. Human histological studies will confirm this approach for saccadic disorders in the future.

Fast eye movements, such as saccades or the quick phases of optokinetic reflexes, as well as vestibular forms of nystagmus, are accomplished by high-frequency bursting of motoneurons supplying extraocular muscles. The burst component of motoneuron firing originates from input of premotor burst neurons located in the Paramedian Pontine Reticular Formation (PPRF) for horizontal eye movements and in the Rostral Interstitial Nucleus of the Medial Longitudinal Fascicle (RIMLF) for vertical/torsional eye movements, according to anatomical tract-tracing and electrophysiological recording experiments in monkeys. While Burst Neurons (BNs) of the RIMLF are found throughout the nucleus's wing-like shape, mid-sized BNs of the PPRF are found bilaterally intermingled with neurons of various sizes and shapes belonging to non-oculomotor systems, and are thus frequently identified with the assistance of a microscope.

Both populations of burst neurons are excitatory and use glutamate/aspartate as transmitters. Contralateral motor nuclei, on the other hand, receive simultaneous inhibitory burst signals during conjugate saccadic eye movements. For horizontal eye movements, Inhibitory Premotor Burst Neurons (IBNs) are located in the nucleus Paragigantocellularis Dorsalis (PGD), and for vertical/torsional eye movements, they are presumably located in the interstitial nucleus of Cajal. Along with IBNs, the interstitial nucleus of Cajal contains multiple neuronal populations that require further delineation and identification. IBNs in the horizontal system are thought to use glycine as a transmitter, whereas IBNs in the vertical system are thought to use GABA.

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Monosynaptic and simultaneous innervation of extraocular moto neurons by premotor EBNs and IBNs is required for synchronous and precise saccadic eye movement initiation and generation. The histochemical signature of premotor Burst Neurons (BNs) in macaque monkeys was investigated in relation to their intrinsic membrane properties that facilitate bursting at high firing rates. Ion channel profiles of two premotor BN populations with different anatomical connections and transmitter content were investigated to rule out any specific biases in their physiological profiles. Within the constraints of this study, neither of the BN populations differed in their ion channel expression profiles. All BNs expressed Kv1.1 and Kv3.1, a combination required for fast-firing, as well as low-voltage-activated ion channels, regardless of whether they had an excitatory or inhibitory effect or whether they were horizontal or vertical components of the saccadic circuitry. This lends support to the BN PIR hypothesis. Furthermore, the discovery of these ion channel protein expression profiles in BNs of the saccadic system of the macaque monkey opens the door to post-mortem investigation of altered phenotypes in human pathology cases with eye movement disorders. The histochemical signature of premotor Burst Neurons (BNs) in macaque monkeys was investigated in relation to intrinsic membrane properties that facilitate bursting at high firing rates. The proof of concept was investigated by examining the ion channel profiles of two premotor burst neuron populations with distinct anatomical connections and transmitter content. The main findings were that BNs can be distinguished by immunohistochemistry of per neuronal nets and non-phosphorylated neuro-filaments; the IBN area in PGD contains multiple neuronal populations that can be distinguished by calretinin immunostaining; and finally, both BN populations express voltage-gated potassium channels that correspond to their fast-firing characteristics.

They also express HCN1 and HCN2, as well as Cav3.2-3.3 channels, supporting the PIR-bursting hypothesis of BNs. Overall; these findings provide histochemical evidence as well as indirect information on the physiological properties of BNs. This evidence can be used to test hypotheses derived from clinical data using mathematical models. Furthermore, this study marks an important step forward in the investigation of BN channelopathy as a cause of eye movement symptoms in disorders such as PSP, opsoclonus, and NPC in pathological post-mortem human brainstem tissue.