Previous computational models have been proposed to suggest the underlying biophysical mechanisms theoretically. Similar to [4, 6], we interpret burst suppression as the consequence of interaction of two different time-scaled processes linked though a gating variable. It was pointed out in [7] that the slow system is not only associated with direct perturbations but also coupled to neuronal activity through auto-regulatory mechanism. On this basis, an extended version of [6] was developed in [2], where the slow time scale of burst suppression was attributed to activity-dependent synaptic depletion. In this paper, we have introduced an extension of the Wilson-Cowan mean field model that includes dynamics for metabolic support and homeostasis. The simulations show that the model proposed is able to produce physiological responses similar to that one can observe in the EEG under deep anesthesia. With anesthetic (propofol) induction, neuronal activity experiences transition from continuous oscillations to bistable dynamics (burst suppression patterns), and eventually isoeletricity for higher concentration. The model robust and exhibits quasi-periodicity with explicit inclusion of stochastic factors.

More importantly, the model may help explain the spatio-temporal feature of burst suppression [3, 5]. The globalness of burst onset may arise though some broader effect such as altered neural metabolic dynamics. The inhomogeneity of burst suppression occurs when the global bursts are compromised by network heterogeneity, in the cortical area (parameter mismatch in individual columns, for example due to different levels of sensitivity to the anesthetics [1]). At the same time, inter-column couplings lead to local burst synchronization (locally overcome the network heterogeneity). This local burst synchronization, however, is less likely to grow global if the cortical network only depends on the short-ranged neuronal couplings. This also reconciles with the results in [2].

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