Testing brains with burst suppressions

Distinguishing burst suppression (BS) pattern from a “continuous EEG” pattern is perhaps the first and easiest to learn by junior fellows as they start working in units with neurological intensive care (Westhall et al., 2015). Detection of BS is considered the hallmark of severe compromise in brain function, and in the context of brain damage, it is traditionally considered to signify a poor prognosis (Westhall et al., 2016; Hofmeijer et al., 2014). Recognition of BS is important for neurologists when treating status epilepticus using anesthesia that is titrated to keep the EEG at the “BS level”, between the levels of continuous EEG and inactive EEG.

Traditionally, recognition of BS used to be based on observing a constant background pattern that does not change over time or does not react to patient handling, and it is hence considered “unreactive”. Long term EEG monitoring of patients with acute brain injury has changed the clinical perception of BS, which is now viewed as a brain state that usually changes over time. For instance, prognosis after acute asphyxia (van Rooij et al., 2005) or cardiac arrest (Oh et al., 2015) can be done from counting the hours from the initial insult to the evolution of EEG from the BS pattern to recovery of continuous EEG pattern (comprehensively reviewed by Hofmeijer and van Putten, 2016). Conceivably, such dynamics in brain states also imply that BS is not a discrete, unitary state, but the functional brain status can vary substantially within a broader category of BS states. Recent clinical studies have, indeed, provided computational evidence for quantifiable dynamics in BS that may predict clinical outcomes (Iyer et al., 2014; van Putten et al., 2015; Hofmeijer et al., 2014).

A general feature in neurological assessments is “reactivity” to exogenous stimuli, which can be testing of various reflexes, or response to handling and commands. In comatose patients, these observations are combined in an assessment scale, such as the commonly used Glasgow Coma Scale (GCS). However, these scales are not very sensitive in discriminating between classes of patients in deeper comatose states, such as those with BS pattern in their EEG.

The study of Nita et al. (2016) in this issue of Clinical Neurophysiology takes an important step in this direction. They show that BS pattern in the EEG after traumatic brain injury may react to exogenous stimulations, which is delivered here using the conventional photic stimulation (PS) available in all standard EEG devices. In their work, Nita et al. delivers PS at 1 Hz for a period of one minute, and they find that the global field power of EEG is significantly increased during PS as compared to the immediately preceding baseline period. This “BS reactivity” was found in all five pediatric patients with different etiologies of brain trauma, and the level of their “BS reactivity” depended on the baseline BS, as well as the clinical level of patient’s coma. For a neurophysiologist, it was especially interesting that PS did not affect the heart rate which was used to exclude the possibility of a more general arousal. Hence, the EEG-recorded BS reactivity likely discloses a global feature of cortical brain function that cannot be approached using other clinical measures in these patients.

Several prior studies have examined BS reactivity in deeply anesthetized humans as well as in experimental animal models (referenced in Nita et al.). While those earlier findings are compatible with the observations by Nita et al., it is important to recognize that they don’t directly compare with the clinically relevant group of traumatic BS more than at the level of EEG phenomenology. The work of Nita et al. studying BS in genuinely brain injured patients is opening a novel pathway for future clinical studies. It shows that recognition of BS pattern in the EEG is just the start, and the cortical function can be readily probed deeper by using simple methods that are already available at bedside.

Future studies will be warranted to address some key properties of BS reactivity on its way to more standardized clinical practice. First, larger patient populations with more diverse etiologies and with more different age groups will be needed. The aim here should be to both establish the general utility of BS reactivity, as well as to establish quantitative norms for different ages and etiologies. Second, it would be interesting to learn whether BS reactivity is global, as shown in the study of Nita and colleagues. One could also expect that different sensory modalities (PS, auditory, tactile) might trigger region specific responses, which by themselves have further information value in differential diagnostics in the severely brain damaged patients. Third, the evolution of BS reactivity over time will need further exploration, akin to the already employed monitoring of cortical reactivity using conventional evoked potential paradigms (Fisher et al., 2016; Koenig and Kaplan, 2015). Finally, the method of computing BS reactivity by Nita et al. was relatively simple, though appropriated for a proof of concept study. The future studies can take the computational challenge to the next level and seek to define more adaptive, interactive, and even automated (Liberman et al., 2013) ways to quantify BS reactivity to make its estimate directly available for the bedside practitioner.
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References


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