Awakening after sugammadex: jumping to conclusions?

We read with interest the publication by Le Guen et al. regarding the effects of the reversal of neuromuscular blocking (NMB) drugs with sugammadex on bispectral index [1]. This is an important topic because neuromuscular blockade may have an effect on the patient's raw electroencephalogram (EEG), bispectral index and consciousness. As the authors write, one possible theory is related to a reduction in afferent inputs to the thalamocortical system [2]. Although we agree with the fact that NMB drugs should be antagonised before stopping hypnotics and antinociceptive agents, we disagree with Le Guen et al.'s conclusion, which cannot be inferred based on their findings.

We believe that the impact of electromyography (EMG) on the bispectral index might be understated in Le Guen's publication [1]. The monitor used for the study was the bispectral index monitor A-2000 XP with unilateral sensors. We use bispectral index monitors in our institution on a daily basis, and one of its limitations is the detection of EMG activity, which is not shown under 30 dB. Thus, the inference regarding the absence of artefacts during the emergence-phase might be false, and we are quite certain that EMG activity had an important role in increased bispectral index values, which would not necessarily translate to a 'lighter' depth of anaesthesia. As such, we suggest that other depth of anaesthesia monitoring should be used, such as monitors with density spectral array or monitors which can adequately quantify the presence of EMG activity.

In our opinion, another limitation is the consideration of cough and spontaneous movements as clinical signs of recovery of consciousness. These signs can also be associated with inadequate antinociception or neuromuscular blockade, even with an adequate level of hypnosis. A practical example is the placement of supraglottic airway devices without neuromuscular blockade, as supratherapeutic doses of hypnotics might be needed to keep the patient immobile even with an adequate level of hypnosis.

Regarding the clinical signs of awakening, the signs presented by the control group are not shown in Table 2, as mentioned in the text. Moreover, the authors mention that external stimulation was not allowed during the emergence phase. Therefore, we do not understand how responding to simple commands can be considered a sign of awakening.

We are also curious to know what was considered 'complete recovery' of consciousness. It would be very interesting to have access to the raw EEG before the emergence phase in the patients who had complete recovery of consciousness after the administration of sugammadex. Were delta and alpha-spindle activity present before antagonising the NMB drugs?

The authors also mention that propofol and remifentanil infusions were immediately stopped if any clinical sign of awakening was observed. Were the infusions stopped even when reversal of NMB drugs was incomplete?

Furthermore, we have some questions concerning the methodology applied and the findings reported. Can the authors provide any insight regarding the differences observed on bispectral index increments after T5 in the intervention group compared with T10 in the control group? Were post-tetanic counts/train-of-four values recorded during this period and was there any difference between the groups? Was there any statistically significant difference in bispectral index value between these two periods (intervention group T5 vs. control group T10)?

Once again, we congratulate the authors for performing a double-blind randomised crossover study regarding this relevant topic.

F. Salgado-Seixas
R. Pereira
M. Pereira
I. Madeira
Centro Hospitalar Universitário do Porto,
Porto, Portugal
Email: salgadoseixas@gmail.com

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