Counting the train-of-four twitch response: comparison of palpation with mechanomyography, acceleromyography, and electromyography. Comment on *Br J Anaesth* 2020; 124: 712–7

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Editor—Two very different variables can be derived from train-of-four (TOF) stimulation: the TOF ratio (the ratio of the amplitude of the fourth to the amplitude of the first response of the TOF), which is used to indicate whether full recovery from neuromuscular block has occurred, and the TOF count (i.e. the number of discernible twitches after TOF stimulation), which indicates the depth of neuromuscular blockade. Bowdle and colleagues[1] compared simultaneously two approaches to evaluating the TOF count: by subjective observer-based palpation and by using one of three different objective monitors (mechanomyography [MMG], acceleromyography [AMG], and electromyography [EMG]).

According to their results, MMG was more sensitive than subjective palpation for counting the twitches after TOF stimulation, whereas EMG was comparable with subjective palpation and AMG was less sensitive.[1] At first glance, this may come as a surprise. It is accepted that MMG, EMG, and AMG differ with regard to their measurement of the TOF ratio, and hence the TOF-ratio determined by different measurement methods should not be used interchangeably.[2,3]

However, Bowdle and colleagues[1] did not measure the TOF ratio, but rather assessed the TOF count. The objectively measured twitch response using MMG, EMG, or AMG should be the same, regardless of the equipment used for its detection. However, this was not the case in Bowdle and colleagues' report: the observation that objective AMG-based detection was less sensitive than subjective palpitation raises particular concern. Unfortunately, the authors did not comment on why they thought the AMG underestimated the TOF count.

One potential explanation for their findings may be the impact of movement artefacts on the different measurement methods. MMG and EMG are not affected by movement artefacts. MMG directly measures isometric contractile force and EMG assesses electrical activity during muscle contraction. Differences between these two methods when compared with subjective palpitation may be explained in this study by differences in its design. MMG and observer-based palpation were carried out on different arms whereas EMG and subjective palpitation were assessed on the same arm.[1] In contrast, AMG measures the acceleration of the muscle after stimulation of the corresponding nerve. Thus, movement of the respective muscle is a prerequisite of this technique. To avoid misinterpretation of the TOF count, AMG has to be able to differentiate between movements of the thumb after TOF stimulation and movement of the thumb for any other reason such as movement of the operating table. AMG devices normally have an in-built mechanism for such discrimination. When using a TOF-Watch acceleromyograph, responses below 3% of control twitch are not registered as a TOF response, but these small responses can still be detected by an observer.[4] This may explain the findings of Bhananker and colleagues,[5] who reported a discrepancy between the TOF count after subjective evaluation by an observer compared with objective detection using a TOF-Watch SX accelerographic monitor.

The TOFScan, a more recently developed AMG monitor, differentiates between twitch response and movement artifact by an in-built algorithm taking into account duration and direction of the movement (User's manual Jun 2020 V1.8.2; Thierry Bagnol, personal communication, July 2020; TOFScan, IDMED, Marseille, France). It would be of interest to know how the StimPod acceleromyograph used by Bowdle and colleagues discriminates between twitch response and movement artifact. This information could contribute to a better understanding of the results and their clinical relevance. Unfortunately, the authors did not yet share this information with the readers of their article.

Declarations of interest

TF-B was an associate editor of the *European Journal of Anaesthesia* from 2010–19. He is chair of the Research Committee of the European Society of Anaesthesia and Intensive care (ESAIC). TF-B has received funding from MSD to give international lectures.

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Visualising the pressure-time burden of elevated intracranial pressure after severe traumatic brain injury: a retrospective confirmatory study

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Editor—Elevated intracranial pressure (ICP) after severe traumatic brain injury (TBI) is an important cause of secondary brain injury, either by hypoperfusion because of decreased cerebral perfusion pressure (CPP), or by mechanical distortion leading to brain herniation.[1] The thresholds to treat elevated ICP in severe TBI (20 or 22 mm Hg) are based on epidemiological studies,[2,3] however, early application of aggressive measures to treat brief episodes of ICP elevations above 20 mm Hg have shown harm.[4] Moreover, the association between elevated ICP and outcome is not merely attributable to crossing a threshold, but depends upon the magnitude and the duration of intracranial hypertension. This has been demonstrated in a multicentre prospective European dataset (n=261) by Güiza and colleagues.[5] Using a three-dimensional visualisation technique, they showed that worse outcomes (taken at 6 months) could be explained by the interaction between the level of ICP elevation and the duration of the hypertensive episode, confirming a clinically intuitive concept. For instance, insults of high ICP, >30 mm Hg, seemed to be only tolerated for a short time (<8 min), whereas ICP>20 mm Hg leads, on average, to a poor outcome if sustained over 37 min. The ability to tolerate elevated ICP was decreased in children, when cerebrovascular autoregulation was absent, and when CPP was inadequate. To date, this visualisation technique has not been replicated outside of the prospective European dataset.[5]

We sought to confirm these findings by applying the same visualisation method on an independent patient cohort of 1112 severe TBI patients from Addenbrooke's Hospital (Cambridge, UK) collected between 1991 and 2017. Patient characteristics and management protocols have been described.[6] Because all data were extracted from the hospital records and fully anonymised, no data on patient identifiers were available, and therefore formal patient or proxy consent and institutional ethics approval were not required.

From minute-by-minute resolution data (time-averaged), ICP hypertensive episodes were defined as being above a given intensity threshold I, for at least a given duration D. For each pair of intensity and duration thresholds <I,D>, the average number of corresponding episodes per patient was calculated, separately in each 6-month Glasgow outcome scale (GOS) group.[5] Thereafter, the Pearson correlation between the average number of ICP episodes and GOS was calculated for each <I,D>, and colour-coded according to a predefined colour map (Fig. 1). In this way, a single time point with elevated ICP can contribute to multiple insults on the colour contour plot. For example, an ICP of 12.5 mm Hg for 5 min will contribute to the episode-outcome Pearson correlation for the ICP>10, 11, and 12 mm Hg intensity category, and for the >1, 2, 3, 4, and 5 min duration category. Multivariable logistic regression was used to study the effect of the amount of time in the red zone of these plots, with patient outcome (mortality and unfavourable outcome) after adjustment for age and initial Glasgow Coma Scale.

From the 1112 patients, 34 million ICP insults were identified. By plotting the relationship between the insult count for each <I,D> pair and the outcome, a three-dimensional colour-coded contour plot was obtained, similar to that of Güiza and