CORRESPONDENCE


In Reply—We appreciate the obviously careful reading of our paper by Drs. Brandon and Cook and are grateful to them for drawing our attention to two minor errors. First, in our discussion we stated that Weber et al. used a single twitch mode of nerve stimulation and that Choi et al. used electromyography to measure neuromuscular response. These authors and their methods were erroneously transposed and, in fact, Weber et al. used electromyography and Choi et al. used single twitch mode of nerve stimulation. Second, in table 1, the dibucaine number of patient 4 should be 72 and not 84 as printed.

We support the reanalysis which Brandom and Cook have made of their dose-response data. The weakness of the original analysis by Weber et al. was that they estimated an ED₅₀ value by extrapolation from values for ED₃₀, ED₅₀, and ED₇₀. This resulted in an underestimate of the ED₅₀. In our study, we derived the ED₅₀ value from data points covering the entire range of responses from 0% to 100% twitch depression. While it can be argued how best to treat 100% responses, we consider that an accurate determination of ED₅₀ requires data points both above and below 95% response. The revised estimate for ED₅₀ obtained by Brandom and Cook, 70 µg·kg⁻¹, after their reanalysis of the data of Weber et al. is similar to the value, 67 µg·kg⁻¹, which we reported in our paper.

The debate concerning the treatment of data points at the extremes of the dose-response curve is too complex to be covered in this forum. Briefly, we would take the position that extrapolation beyond one's data points is undesirable and that to estimate an ED₅₀ will inevitably require the inclusion of some points representing 100% twitch depression. If one has data only within the 20–80% range of response, then only the ED₃₀ should be reported. As we noted in our paper, published estimates of ED₅₀ are more uniform than are estimates of ED₃₀.

We would agree with Drs. Brandom and Cook that the manner by which variations in plasma cholinesterase activity and genotype affect the metabolism of and neuromuscular responses to mivacurium should be elucidated. Our data regarding neuromuscular responses in patients with abnormalities of plasma cholinesterase were purely descriptive and suggested that the pattern of response was inconsistent and was not the same as might be seen following succinylcholine administration. For the information of Drs. Brandom and Cook and others who may be interested, the substrate used in our study to estimate plasma cholinesterase activity was acetylthiocholine.

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REFERENCES


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Delayed Respiratory Depression Following Alfentanil

To the Editor—We were interested to read the report of delayed respiratory depression following administration of alfentanil. However, in contrast to the authors’ statement that this phenomenon had not been reported before, we wish to note that delayed respiratory depression following continuous infusion of alfentanil was first described by Lamarche et al. in 1984 and, later that year, two more cases of unexpected respiratory depression were reported after its use.

Delayed respiratory depression has also been described after administration of oxymorphone, and may occur after all opioids. The unstated theme of all these articles is that by whatever route of ad-

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