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REFERENCES


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 Brandon and Cook have made of their dose-response data. The weakness of the original analysis by Weber et al. was that they estimated an ED70 value by extrapolation from values for ED95, ED50, and ED10. This resulted in an underestimation of the ED70. In our study, we derived the ED70 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 ED70 requires data points both above and below 95% response. The revised estimate for ED70 obtained by Brandon 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 ED70 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 ED80 should be reported. As we noted in our paper, published estimates of ED70 are more uniform than are estimates of ED95.

We would agree with Drs. Brandon and Cook that the manner by which variations in plasma cholinesterase activity and genotype affect the metabolism of and neuromuscular responses to mivacurium chloride in humans should be elucidated. Our data regarding neuromuscular responses in the 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. Brandon 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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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-