Correspondence

Neuromuscular Block with Succinylcholine and Decamethonium

To the Editor—R. H. de Jong and F. G. Freund report the characteristics of the neuromuscular block with succinylcholine and decamethonium in man in Anesthesiology 28: 583, 1967. In this paper de Jong and Freund come to the conclusion that the neuromuscular block produced by succinylcholine and decamethonium has from the onset the electromechanical characteristics of a phase II block. This, as they state, is in contradiction to work published earlier by Burns and Paton, Churchill-Davidson et al., Krul et al., and Katz et al.

After publication of their paper, we attempted to duplicate their results, using the same type of nerve stimulator, tektronic oscilloscope, force displacement transducer, and the same pattern of stimulation, but did not succeed. We observed, as reported before, that both succinylcholine and decamethonium initially cause a depolarizing (phase I) block and after increasing doses and time a desensitization (phase II) block. One may wonder how such a marked difference in observations, or at least their interpretation, can be explained.

de Jong and Freund induced anesthesia after a sleep dose of thiopental with a nitrous oxide-oxygen-halothane mixture which was carried to sufficient depth to permit endotracheal intubation. The halothane concentration was then maintained between 0.8 and 1.5 per cent for 45 minutes before control records were taken. Since they found the tetanic tension ratio and the increase in post-tetanic facilitation similar to those found in unanesthetized man, they concluded that “neuromuscular transmission is apparently not affected by halothane in the concentrations used for clinical anesthesia.”

In the same issue of Anesthesiology, Katz and Gissen showed that halothane in concentrations between 1 and 2 per cent does not decrease the twitch height in man. However, if \( d \)-tubocurarine was added, the magnitude and duration of its neuromuscular blocking action was increased. This, in our opinion, conclusively shows that halothane does affect the neuromuscular transmission process, even though at these concentrations of halothane its effect, if used alone, is not evident. This is understandable if one realizes that under normal circumstances a large margin of safety exists to assure neuromuscular transmission. It has been shown in vitro that halothane can depress the neuromuscular transmission process to a point at which the recorded endplate potential after nerve stimulation would not reach the critical membrane potential and no action potential would be propagated. It is therefore possible that the disparity of results obtained by de Jong and Freund compared to those of earlier investigators could be explained by a difference in experimental conditions.

In conclusion we are not willing to disregard all previous work which has conclusively shown that succinylcholine and decamethonium initially produce a depolarizing (phase I) block and with increasing doses and time a desensitization (phase II) block.

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To the Editor—We appreciate the opportunity to reply to the comments by Drs. Karis, Katz and Gissen. We too were surprised by our results, and therefore carefully double-checked them before publication.

Drs. Karis et al. rightly point out the possibility that the administration of halothane may have affected our results. But they provide no evidence that halothane alters the quality of the block. Neither did we find evidence that halothane alters—at least qualita-