Correspondence

Neuromuscular Block with Succinylcholine and Decamethonium

To the Editor—R. H. deJong and F. C. Freund report the characteristics of the neuromuscular block with succinylcholine and decamethonium in man in Anesthesiology 28: 583, 1967. In this paper deJong 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.

deJong 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 2-
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 deJong 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.

Joannes H. Karis, M.D.
Ronald L. Katz, M.D.
Aaron J. Gissen, M.D.
Department of Anesthesiology
Presbyterian Hospital
New York, N. Y.

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-
THE PHASE OF THE BLOCK. Under the conditions of our study—clinical anesthesia—we were unable to observe a block with “phase I” characteristics, i.e., well-sustained tetanic response and lack of post-tetanic facilitation.

We believe that the nature of the block (i.e., depolarization or non-depolarization block) can be distinguished only by recording of the transmembrane potential of the postsynaptic cell. We thus stand firm on our conclusion that under clinical conditions, and using evoked muscle responses to nerve stimulation as the testing method, one is unable to distinguish “phase I” from “phase II” neuromuscular block.

RUDOLPH H. DE JONG, M.D.
FELIX G. FREUND, M.D.
Department of Anesthesiology
University of Washington
School of Medicine
Seattle, Washington

Urinary Epinephrine and Norepinephrine During Innovar-Nitrous Oxide Anesthesia

To the Editor:—I was surprised at some of the opinions expressed in the paper by Ciesecck et al. (Anesthesiology 28: 701, 1967), regarding urinary epinephrine and norepinephrine during Innovar-nitrous oxide anesthesia in man, as compared to our findings in dogs in which assays were done on blood only. I should like to comment on the following points of interest:

1. Twelve patients divided into three groups containing five, four and three people, with six males and six females (distribution not specified) undergoing three different operations for variable periods of surgery with three different agents, hardly lend themselves to a reliable comparative study. Furthermore, this study can hardly be compared to our two studies, even though we used dogs, since each animal was tested in an identical way three times and we used at least ten animals (males) in the same procedure in one of the papers they cited, and, in the other, each animal was first induced with thiopental and four different conditions were tested—again in at least ten dogs for each study. In addition, we established “normal” values and range of error for our assay procedure. Our animals didn’t receive meperidine, secobarbital, atropine or rapid intravenous infusions of 5 per cent dextrose in water and lactated Ringer’s solution, which might affect the assay values in a variety of ways.

2. The Innovar dose they used probably would not have put their patients “to sleep.” Anxiety during induction of anesthesia with nitrous oxide may therefore have been an important reason for the rise in epinephrine in the urine of these patients, if that occurred.

3. The number of observations they made was too small for statistical analysis. A P value of < 0.01 indicates that the changes observed were probably entirely due to chance, which was probably the truth. Incidentally, could the authors have meant P < 0.01?

4. The authors do not state whether they took into account the total volume of urine removed from the bladder at each of the four time intervals. I would guess that a good part of the rise in epinephrine in the urine after catheterization was performed (after induction of anesthesia) may have originated during the induction with nitrous oxide in the Innovar group and may not be due to the Innovar at all. A rise in epinephrine due to surgery in a lightly-anesthetized patient is more likely but, in this case, hard to believe.

5. I believe that the amount of epinephrine assayed is so close to the lower limit of detection, by the method used, that one would be hard put to give credence to the changes they observed unless their recovery tests were exceptionally good.

6. The authors point out that the blood pressure and blood gases remained virtually normal with all three anesthetics. Then, why do they impute a stable blood pressure to the