Correspondence

Defoaming Blood in the Operating Room

To the Editor:—In the operating room estimation of blood lost to the suction bottle is often inaccurate because of a layer of foam on top of the liquid. Commonly the amount of blood in the bottle is calculated by adding half the amount of foam to the actual amount of liquid, but this method is misleading if the layer of foam is unevenly distributed. We would like to remind our colleagues that several drops of caprylic alcohol will completely defoam the blood so that the level of fluid in the bottle can be determined more accurately.

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Depolarizing Neuromuscular Block

To the Editor:—Gissen and Nastuk, in their article on succinylcholine and decamethonium ("Comparison of Depolarization and Desensitization," ANESTHESIOLOGY 33:611, 1970), come to a conclusion that will occasion much surprise. They argue that "the most desirable neuromuscular blocking agent is one that produces maximal postjunctional membrane depolarization and little receptor desensitization. On this basis, our data indicate that SCh is superior to C10 for clinical application. Thus, although there is a wide gap between our experiments and clinical situations, we might conclude from our results that SCh rather than C10 is the relaxant drug of choice." The gap is wide indeed.

There is no clear evidence in their work to support the choice of SCh as a safer muscle relaxant for use in man at 37 C when this work was performed in frog muscles at 18–20 C.

It is well known that the actions of SCh and C10 are greatly dependent on type of muscle,[1] species,[2] and temperature.[3] The neuromuscular blockades induced by SCh and C10 in man[4] and other mammals[3] are more profound at low temperatures (28–30 C) than at 37 C; moreover, observations in man at low temperatures have indicated that "cooling prolonged the action of depolarizing drugs but the nature of the blockade did not appear to be affected however long the paralysis lasted"[4] (there was a phase I type of block only). Furthermore, it has been reported that temperature changes affect the effect of the transmitter differently in the frog[5] and in mammalian muscles.[6] Lowering the temperature in frog muscles—to below 20 C—reduces end-plate potential amplitude,[5] while the opposite effect is observed in mammalian end-plates when the starting point is 37 C.[6]

Finally, SCh and C10 depolarize the mammalian end-plate membrane by only 5 to 10 mv at 32 to 37 C,[7,8] as against 60 mv in frog muscles at 18–20 C according to Gissen and Nastuk. The minimal postjunctional depolarization observed in mammalian muscles does not appear to explain neuromuscular blockade,[7] and there are reasons to doubt that even the larger depolarization observed in frogs could explain this failure of transmission in amphibian muscle.[9]

I am surprised at the logic of Gissen and Nastuk in arriving at their final remark: "although there is a large gap between our experiments and clinical practice, the results in-