from entering the operative field. However, several small shards did manage to escape, either through the mesh or through the rear of the light assembly, and were later found on the floor of the operating room. Also, the concomitant loud noise of the explosion could have startled any member of the operating team and caused an uncontrolled motion. Fortunately, no injury resulted to the patient or to any member of the operating room team.

This case is an example of how a seemingly benign intervention during surgery, the use of a warming light, has the potential to provoke a hazardous situation even in the face of a built-in safety feature.

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Resistance to *d*-Tubocurarine Following Denervation

To the Editor:—I would like to offer a different interpretation of the data presented in the excellent paper by Hogue *et al.*[1] concerning denervation-induced resistance to *d*-tubocurarine (*d*Tc). The data clearly show that 1) the ED_{95} to *d*Tc is decreased in partially denervated legs; 2) the number of acetylcholine receptors (AChRs) is increased in these partially denervated muscles; and 3) there is a correlation between ED_{95} and AChR number. The seminal point of the paper, as reflected by the title, is that the increased AChR number is responsible for the resistance to *d*Tc. The discussion also cautiously indicates that factors other than increased number of receptors may contribute to the resistance. This point may have been underemphasized.

Increased AChR number is not a strong explanation for the resistance, for the following reasons. First, visual inspection indicates that the relationship between the ED_{95} and AChR number is not very strong when only the data from the affected leg is considered. The statistics presented by the author do not analyze this but rather analyze the correlation between AChR number and ED_{95} when data from control and affected limb are pooled together. Thus, an increase in receptor number in particular may not mediate the resistance but may be just one of several effects on nerve and muscle produced by partial denervation. Second, the increased receptor number is poorly correlated to the ED_{50}. Moreover, the ED_{50} is in fact not significantly changed by the partial denervation. If, as suggested by the authors, the per cent occupancy of the AChR by *d*Tc was decreased because of the increase in AChR, then the ED_{50} should be affected in the same manner as the ED_{95}. However, the data presented are inconsistent with this.

It may be that the action of partial denervation important to *d*Tc resistance is not AChR quantity but AChR quality. Following denervation, not only the number of receptors increases but also the subunit composition of the receptors changes.[2] The new denervation-induced AChRs differ from adult junctional receptors in having embryoniclike channel properties[3] and altered sensitivities to acetylcholine and *d*Tc.[2] Mediation of the resistance to *d*Tc by embryoniclike AChRs incorporated at or near the junction may explain the weak correlation to receptor number as well as the difference in effect of partial denervation on ED_{50} and ED_{95}.

Of course, other explanations for the denervation-induced resistance to *d*Tc exist. The authors (and reviewers) are to be commended for presenting results and a discussion that are open to reinterpretation.

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In Reply:—We appreciate Dr. Storella's thoughtful review and appropriate comments on our report relating increased dose requirements for *d*-tubocurarine (*d*Tc) to increases in nicotinic acetylcholine receptors (AChR).[1] Dr. Storella highlights several points that deserve discussion. As he mentions, while the dose to achieve 95% twitch depression (ED_{95}) in the partially denervated animals was higher than controls, the ED_{50} between the groups did not reach significance. Likewise, while the correlation of ED_{95} and AChR was strong, the relationship was weaker for ED_{50} and AChR.

Complete denervation results in the conversion of mature to immature form of AChR. The immature differs from the mature AChR in subunit composition, binding affinities for ligands, and electrophysiologic and immunological characteristics.[2–4] The degradation half life (t½) is also different. In the mature AChR the t½ is ~8 days.[5] The t½ accelerates