3. Martin RW, Bashein G, Zimmer R, Sutherland J: An endoscopic micromanipulator for multiplanar transesophageal imaging. Ultrasound Med Biol 12:965–975, 1986
4. Martin RW, Bashein G: Measurement of stroke volume with three-dimensional transesophageal ultrasonic scanning. ANESTHESIOLOGY 70:470–476, 1989
5. Abel MD, Nishimura RA, Callahan MJ, Rehder K, Ilstrup DM, Tajik AJ: Evaluation of intraoperative transesophageal two-dimensional echocardiography. ANESTHESIOLOGY 66:64–68, 1987

(Accepted for publication January 29, 1991.)

Anesthesiology
74:959, 1991

In Reply:—Dr. Bashein and Dr. Martin have devised an ingenious solution to the difficulties of imaging the left ventricle in dogs using transesophageal echo techniques. Although surgery is required, the experimental model that they have developed should provide useful insights into the problems of transesophageal imaging for the detection of myocardial ischemia.

One important caveat is the requirement for thoracotomy to exploit the Bashein/Martin model. It is well recognized that thoracotomy induces abnormalities of cardiac motion at least as delineated by transthoracic echocardiography. Various explanations have been offered for these abnormalities, including alterations in intrathoracic rotational and translational motion, regional ischemia, effects of cardioplegia, and cardiopulmonary bypass.[1,2] These thoracotomy-induced alterations may introduce an additional variable into studies of ischemic dyskinesis and must be considered by researchers performing transesophageal echos in dogs using the Bashein/Martin thoracotomy model.

RICHARD E. KERBER, M.D.
Professor of Medicine

BYRON F. VANDENBERG, M.D.
Assistant Professor of Medicine

Cardiovascular Division
Department of Internal Medicine
The University of Iowa
Iowa City, Iowa 52242

REFERENCES

1. Kerber RE, Litchfield R: Postoperative abnormalities of interventricular septal motion: Two-dimensional and M-mode echocardiographic correlations. Am Heart J 104:263–268, 1982
2. Lehmann KG, Lee FA, McKenzie WB, Barash PG, Prokop EK, Durkin MA, Ezekowitz MD: Onset of altered interventricular motion during cardiac surgery: Assessment by continuous intraoperative transesophageal echocardiography. Circulation 82: 1325–1334, 1990

(Accepted for publication January 29, 1991.)

Anesthesiology
74:959–960, 1991

Hazard with Warming Lights

To the Editor:—Safety features found on equipment are often unappreciated, especially if they function only on rare occasions. We would like to call attention to an operating room situation where the protective feature of a heating lamp most likely prevented a serious untoward event. However, this safety feature alone may not always offer full protection against the failure conditions for which it was designed.

A 21-yr-old man was undergoing retroperitoneal lymph node dissection for a metastatic testicular seminoma. All measures were taken to preserve his body heat, including the use of an Emerson 96-HB warming light (J. H. Emerson Co., Cambridge, MA). This device supplies radiant heat by means of two 250-W infrared producing light bulbs. A safety feature of the warming lights consists of a double layer of wire mesh around the sides of each bulb and a single layer on the front. The wire mesh is made from strands 0.5 mm in diameter and consists of a grid 6 × 6 cm.

During the case, the warming light was directed at the head of the patient from a distance of 72 cm, as determined by an integral measuring rod. The light was positioned so that the bulbs were directly below an intravenous (iv) fluid bag and administration set on an IV pole (fig. 1). During one of the frequent replacements of the iv fluid bag, several drops of residual solution fell onto one of the heat-producing light bulbs. The resultant thermal shock on the glass bulb caused it to explode. The wire mesh around the bulb prevented glass shards

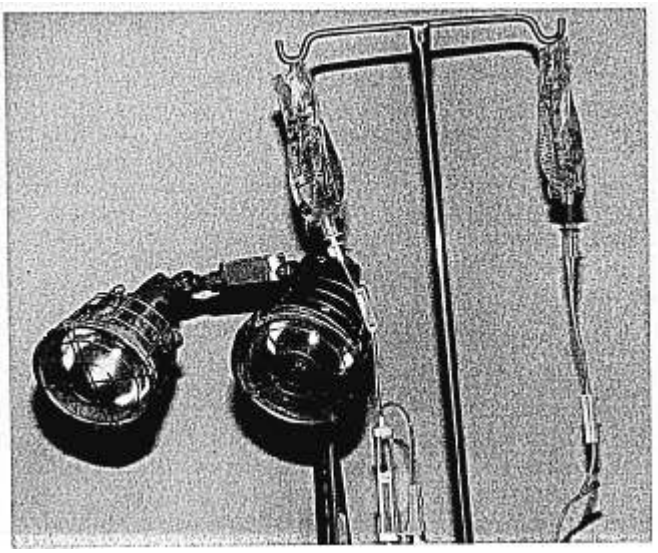

FIG. 1. Bag of fluid hanging directly over warming lights. Note protective wire mesh over the front of the lights.

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.

MARC I. LEVINE, M.D.
Resident in Anesthesiology

ALVIN WALD, PH.D., C.C.E.
Technical Director

Department of Anesthesiology
Columbia-Presbyterian Medical Center
630 West 168th Street
New York, New York 10032

(Accepted for publication January 29, 1991.)

Anesthesiology
74:960, 1991

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.

ROBERT J. STORELLA, JR., PH.D.
Department of Anesthesiology
Hahnemann University
Broad and Vine
Philadelphia, Pennsylvania 19102-1192

REFERENCES

1. Hogue CW, Itani MS, Martyn JAJ: Resistance to *d*-tubocurarine in lower motor neuron injury is related to increased acetylcholine receptors at the neuromuscular junction. ANESTHESIOLOGY 73: 703–709, 1990
2. Lorkovic H: Sensitivity of rodent skeletal muscles to dicholines: Dependence on innervation and age. Neuropharmacology 28: 373–377, 1989
3. Schuetze SM, Role LW: Developmental regulation of nicotinic acetylcholine receptors. Ann Rev Neurosci 10:403–457, 1987

Anesthesiology
74:960–961, 1991

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 ($t_{1/2}$) is also different. In the mature AChR the $t_{1/2}$ is ~8 days.[5] The $t_{1/2}$ accelerates