

**Gregory Allen, M.D., F.R.C.P.C.**

Director

North American Malignant Hyperthermia Registry  
Malignant Hyperthermia Association of the United States  
Sherburne, New York

Associate Professor of Anesthesia

College of Medicine

The Pennsylvania State University

Hershey, Pennsylvania

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*In Reply:*—Dr. Allen writes that we failed to take into account the muscle rigidity of the left arm in the clinical scale of Larach *et al.*,<sup>1</sup> leading to an underestimation of the likelihood of MH susceptibility. However, only tachycardia and muscle breakdown could be detected in this patient. We intentionally did not take into account the muscle rigidity of the left arm, because in the definition published by Larach *et al.*,<sup>1</sup> rigidity is designed as generalized rigidity or masseter muscle spasm. Our patient did not exhibit such clinical signs.

We agree with Dr. Allen that data from *in vitro* contracture tests are not used to rank a possible MH event. In our case, the clinical signs were mild (perhaps beneath the threshold of clinical detection?). Nevertheless, the laboratory tests were performed as soon as MH was suspected, as recommended by Dr. Allen. This case report is interesting, because mild clinical signs may be associated with life-threatening rhabdomyolysis, in the face of an otherwise unrecognized MH episode. In such a clinical setting, *in vitro* contracture tests are needed to determine MH susceptibility.

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## Is Lack of Statistical Power Always Evidence of Lack of Effect?

*To the Editor:*—I read with interest the paper by Pittman *et al.*<sup>1</sup> recently published in ANESTHESIOLOGY. The study in the article shows that rats undergoing 75-min middle cerebral artery occlusion during pentobarbital or propofol anesthesia, in doses sufficient to maintain electroencephalogram burst suppression, have similar neurologic and histologic outcomes. The study is well designed, and I think that this research is extremely important.

As stated in the article by the authors, the neurologic scores between the two groups were not significantly different. However, when neurologic scores are analyzed in detail (their fig. 2), animals treated with pentobarbital seem to have a better neurologic outcome. In fact, seven pentobarbital-treated animals had a neurologic score of 1 (as compared to one propofol-treated animal), and fewer pentobarbital-treated rats had a score of 2 (four *versus* seven) or a score of 3 (six *versus* nine). If we pool the results (0 to 1 *versus* 2 to 3 neurologic score), 10 animals treated with pentobarbital *versus* 3 animals treated with propofol had a "good" neu-

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**Lisiane Fierobe, M.D.**

Assistant Professor of Anesthesiology

Department of Anesthesiology and Critical Care

Hospital Bichat

Paris, France

lFierobe.bichat@in vivo.edu

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1. Larach MG, Localio AR, Allen GC, Denborough MA, Ellis FR, Gronert GA, Kaplan RF, Muldoon SM, Nelson TE, Ording H, Rosenberg H, Waud BE, Wedel DJ: A clinical grading scale to predict malignant hyperthermia susceptibility. ANESTHESIOLOGY 1994; 80:771-9

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rologic score (0 to 1, no deficit or left forelimb flexion only), and 10 *versus* 16 had a more severe hemiparesis (2 to 3 neurologic score).

The authors did not attempt to "force" the results in any direction. I also agree that histologic results (infarct areas) provide a better end point when considering the protective cerebral effects of any drug or treatment, or both. However, although the aforementioned differences are not statistically significant, they should be considered. The authors did not provide any correlation between histology (infarct area) and neurologic deficit. I would speculate that such scores are congruent, with smaller cerebral infarct size correlating with better neurologic outcome. However, if this is the case, I wonder which drug provides a better correlation.

**Concezione Tommasino, M.D.**

Assistant Professor of Anesthesiology

University of Milano

Milan, Italy

tommasino.concezione@hsr.it



## CORRESPONDENCE

## Reference

1. Pittman JE, Sheng H, Pearlstein R, Brinkhous A, Dexter F, Warner DS: Comparison of the effects of propofol and pentobarbital on neurologic outcome and cerebral infarct size after temporary focal ischemia in the rat. *ANESTHESIOLOGY* 1997; 87:1139-44

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**In Reply:**—Dr. Tommasino raises the issue whether a different statistical analysis might offer a different conclusion regarding the relative effects of propofol and pentobarbital on neurologic outcome from transient middle cerebral artery occlusion in the rat. We agree that visual inspection of figure 2 in Pittman *et al.* suggests this possibility.<sup>1</sup> However, the statistical test (Mann-Whitney U test-Wilcoxon's rank sum) used to compare neurologic scores between groups was chosen *a priori*. This decision was, in retrospect, quite reasonable. Our study<sup>1</sup> was not designed to detect a difference between groups for the correlation between infarct volume and neurologic score. Therefore, we recommend that no conclusions be drawn from these values. Nevertheless, as requested, for propofol, Kendall's  $\tau = 0.51$  (95% confidence interval, 0.21 to 0.81) and, for pentobarbital, Kendall's  $\tau = 0.31$  (95% confidence interval 0.01 to 0.60). Perhaps the correlation is better for the propofol group. But, because our experiment was not designed to test differences in correlation between the two groups, we truly do not know. The data presented in figure 2 of Pittman *et al.*<sup>1</sup> suggest, however, that a more comprehensive approach to the neurologic examination may be of value in defining potential differences among groups. This is being explored in our laboratory and by other groups.

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### David S. Warner, M.D.

Professor

Departments of Anesthesiology, Neurobiology, and Surgery

Duke University Medical Center

Durham, North Carolina

warne002@mc.duke.edu

### Franklin Dexter, M.D., Ph.D.

Associate Professor

Department of Anesthesia

University of Iowa College of Medicine

Iowa City, Iowa

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## A Method for Measuring Carbon Dioxide at the Tracheal Stoma

**To the Editor:**—End-tidal carbon dioxide monitoring is a standard used during general anesthesia and monitored anesthesia care. Nasal cannulas commonly are used to deliver supplemental oxygen and to sample carbon dioxide for the monitoring of respiratory rate and rhythm in patients undergoing sedation for surgical procedures. Monitoring end-tidal carbon dioxide in patients with tracheal stomas may be difficult when using standard tracheal collar oxygen supplementation because no adaptation for the carbon dioxide sample line is readily available. We present a device for the monitoring of carbon dioxide in patients with tracheal stomas who are undergoing operative procedures that necessitate intravenous sedation.

Taking a standard tracheal collar, a 6-inch piece of corrugated oxygen tubing, a BODAI suction safe swively endotracheal suction connector (Sontek Medical, Hingham, MA), and an 8-French pediatric suction catheter, we fabricated a simple device to monitor carbon dioxide at the tracheal stoma (fig. 1). The pediatric suction catheter is placed through the BODAI suction device and threaded into the corrugated tubing until it rests at the skin edge of the tracheal stoma. The end-tidal carbon dioxide sample line is attached

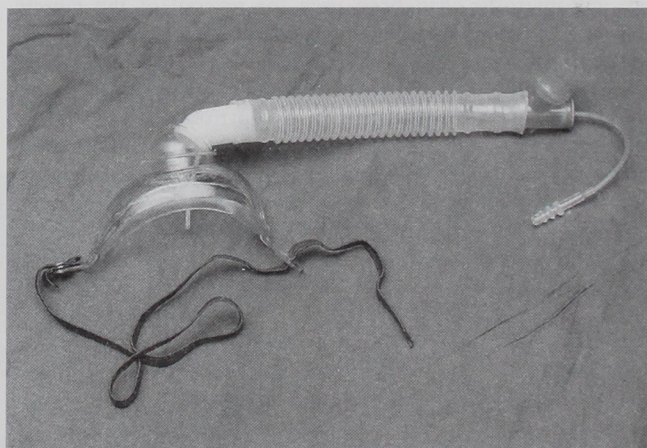


Fig. 1. Assembled airway equipment.