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Postanesthesia Evaluation of Neuromuscular Function

To the Editor:

The American Society of Anesthesiologists' recently published Practice Guidelines for Postanesthetic Care[1] contains a statement that is at best puzzling and at worst I believe sends the wrong message to the anesthesia community. To quote: "Assessment of neuromuscular function primarily includes physical examination and, on occasion, may include neuromuscular blockade monitoring."

There is now overwhelming evidence that traditional bedside or clinical tests of neuromuscular function such as headlift, tidal volume, tongue protrusion, and others are very insensitive tests for the detection of residual neuromuscular weakness.[2–5] To cite just one recent study "a reliable clinical test for detection of significant residual block... will probably remain elusive."[6] Thus one must ask what clinical signs the Task Force is referring to when they recommend a "physical examination"?

The answer to the problem of postoperative residual neuromuscular block lies not with a postanesthesia evaluation, but with intelligent intraoperative monitoring of neuromuscular function ideally with a quantitative monitor.

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(Accepted for publication May 22, 2013.)

In Reply:

We thank Dr. Kopman for his comments regarding the Practice Guidelines for Postanesthetic Care.[1] This guideline document consisted of an update rather than a comprehensive revision of the 2001 version[2] and examined new evidence from literature, surveys, and other sources as applied to the existing evidence model. Of note, there were no changes to the recommendations. Had we obtained substantive new findings as applied to the original evidence linkages, we would likely have proceeded with a full revision and had the opportunity to reconsider the issue raised by Dr. Kopman.

Regarding traditional bedside or clinical tests of neuromuscular function, we agree with Dr. Kopman that this area does straddle the topics of intraoperative and postoperative care, and our literature search focused primarily on postoperative care. In this case, our findings were observational as opposed to Category A (randomized controlled trial) evidence and believe that more research is needed in this important area. These observational studies did indicate that neuromuscular blockade monitoring is effective in detecting neuromuscular dysfunction. We also agree that intraoperative monitoring of neuromuscular function (ideally with a quantitative monitor) would be valuable, particularly during emergence and recovery.

As with all of the American Society of Anesthesiologists (ASA) evidence-based practice parameters, the ASA endeavors to conduct an exhaustive literature search and invites comments and contributions from Task Force members, expert consultants, and other contributors during the several months the preapproval draft is posted on the internet. Though no queries similar to those raised by Dr. Kopman were received when the draft of this document was available for comment, we plan to again review these Guidelines in the future and will consider the query at that time. Again, we thank Dr. Kopman for his thoughtful and informative letter indicating his concerns.

Jeffrey H. Silverstein, M.D.,* Jeffrey L. Apfelbaum, M.D., Richard T. Connis, Ph.D., David G. Nickinovich, Ph.D.; on behalf of the American Society of Anesthesiologists Task Force on Postanesthetic Care. *Mount Sinai School of Medicine, New York, New York. jeff.silverstein@mssm.edu

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A Plea for the Cautious Use of Droperidol

To the Editor:

I read with interest the recent retrospective investigation by Nuttall *et al.*[1] regarding the safety of low-dose droperidol.

Apparently to justify the use of droperidol at the Mayo Clinic, Nuttall *et al.* noted the minutes of an offsite Food and Drug Administration meeting where an employee stated that droperidol's black box warning does not apply to the use of low doses of this drug.* At the same time, they quoted the following bold print excerpt from droperidol's extensive boxed warning: "Cases of QT prolongation and/or torsade de pointes have been reported in patients receiving droperidol at doses at or below recommended doses."† These two positions seem to be at odds. Also, they did not mention the statement from the boxed warning that if the QTc is prolonged, droperidol should not be administered or that the package insert states that it should be used with extreme caution if the patient has any significant heart disease.

In their report, Nuttall *et al.* listed several patients who were known to have prolonged QTc intervals and even episodes of ventricular tachycardia before receiving droperidol who either died or developed ventricular tachycardia within 48 h of the droperidol administration. The administration of droperidol to these patients is in complete violation of the package insert. On the basis of their failure to uncover the unequivocal development of torsades de pointes, the authors concluded that droperidol was in no way contributory to these outcomes. They do admit that their retrospective study may not have uncovered such arrhythmias in part because of the inability to capture brief episodes of torsades and other problems. Apparently, the patients did not undergo the mandatory 2–3 h electrocardiogram monitoring called for by the package insert, and it was not clear that the droperidol was administered after the failure of other antiemetics as is also called for by the package insert.

My view is that droperidol's published boxed warning and package insert are more definitive than comments made at a meeting which cannot necessarily be taken as the official position of the Food and Drug Administration. Consequently, I do not believe that the statement in this article entitled "What We Already Know about This Topic" that the drug's warnings do not apply to low doses is correct.

In summary, the boxed warning and package insert for droperidol must be carefully read, and its cautionary information closely adhered to in order to more safely use this potentially dangerous medication.

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In Reply:

I thank the Editor for the opportunity to respond to the comments put forth by Dr. Sosis regarding our article.[1] We did not justify the use of droperidol at the Mayo Clinic based on minutes of an offsite Food and Drug Administration meeting. We actually performed a large retrospective safety study which was published in the journal Anesthesiology in 2007.[2] Droperidol was added back to our formulary after that study. I noted that droperidol was frequently being used by my colleagues. We performed our second retrospective safety study to determine whether this behavior was safe. We found no evidence of polymorphic ventricular tachycardia (VT) associated with the use of this drug.

As noted in our article, there were eight patients who died after droperidol administration. All of the eight patients who died were on palliative care and died of their disease. There were four patients with documented VT, but all four patients had previous cardiac conditions: two had preexisting internal cardiac defibrillators, three had episodes of VT before receiving droperidol, and another had preexisting hypertrophic obstructive cardiomyopathy and underwent a septal myectomy. None of the above-mentioned patients had a prolongation of the QT. The back box states "Cases of QT prolongation and/or torsade de pointes, some fatal, have been reported in patients receiving droperidol at doses at or below recommended doses. All patients should undergo a 12-lead electrocardiogram before administration of droperidol to determine whether a prolonged QT interval (*i.e.*, QTc >440 ms for males or 450 ms for females) is present. Do not administer droperidol if there is a prolonged QT interval. Droperidol is contraindicated in patients with known or suspected QT prolongation, including patients with congenital long QT syndrome. Administer droperidol

* Available at: www.fda.gov/ohrms/dockets/ac/03/transcripts/4000T1.DOC. Accessed February 18, 2013.

† Available at: http://www.americanregent.com/documents/Product19PrescribingInformation.pdf. Accessed February 18, 2013.