

study was unfortunately overlooked by Drs. Eikermann and Chamberlin. Indeed, we provided evidence that propofol anesthesia desynchronized the circadian rhythms of rest-activity and body temperature in rodents in the experimental condition of either alternation of light/dark² or constant darkness.⁴

By citing the study of Sessler *et al.* of 1991,⁵ they create the impression that anesthetic exposure does not affect the circadian rhythms in humans. It must be pointed out that this first study was unable to demonstrate any effect in five human volunteers. Sessler *et al.*⁵ acknowledged that such data did not exclude an effect that could be missed. Indeed, the shifts in acrophase were +1.2, +2.1, -0.7, -1.6, and -0.7 h in the five subjects. Drs. Eikermann and Chamberlin have once again overlooked our previous study (cited in the article) that demonstrated a desynchronization of the circadian rest-activity rhythm after propofol anesthesia in patients.⁶ When dealing with clinical studies, as we clearly state in the discussion of our article, one has to be cautious in drawing conclusion from merely one or two studies. Further studies are necessary to specify the magnitude of anesthetic effects on human circadian rhythms.

In contrast to the concerns of Drs. Eikermann and Chamberlin, we find that data in this field support the concept that either the “gears” and/or the “hands” of the clock might be influenced by propofol administration. It is premature to eliminate the importance of the concept of gears or hands at this early stage, as they suggest. However, we agree with them that a more profound understanding of the mechanism is an important question. Future studies should rigorously examine the effects of anesthesia on the complex pathways involved in the regulation of the clock. To this end, we are currently pursuing further experiments on the effect of anesthesia on some of these pathways (*i.e.*, the expression of clock genes within the suprachiasmatic nucleus, the melatonin release by the pineal gland).

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Clinical Usefulness of the Muscle Contracture Test: Time to Reevaluate?

To the Editor:

In a recent article, Tautz *et al.*¹ discussed the use of a muscle contracture test for diagnosis of malignant hyperthermia (MH) susceptibility. They correctly noted that there is a 2% chance that contracture testing will incorrectly mislabel an MH-susceptible individual as normal. It is difficult to believe that any of my colleagues will expose a patient to a 2% risk of severe complications by giving triggering anesthetics. In my opinion, the fact that a patient has been evaluated for MH will be a strong indication for using nontriggering anesthesia management. Tautz *et al.* stated that there is a consensus among experts that a person who has had contracture testing and who is labeled not susceptible can safely receive triggering anesthetics. Unfortunately, they did not provide references to any written guidelines or consensus statements on that issue.

Tautz *et al.* mentioned another reason for using the contractility test: the fact that for certain patients (children, severe asthmatics, and patients with difficult airways) potent inhaled anesthetics are useful. Yes, they are useful. But how safe are they? Is 98% safe enough when we have 100% safe nontriggering alternatives available? Most biopsy centers do not perform these tests for children under 5 yr old.* In the very few situations in which inhalation induction is the safest approach (*e.g.*, acute epiglottitis or a difficult pediatric airway), the anesthesiologist should be prepared to monitor and treat a possible MH crisis regardless of the patient's test results.

I recall consulting one of my own patients regarding contracture testing after an MH crisis.² According to the clinical grading scale, this patient's likelihood of MH was “almost certain.”³ The patient and family were informed, and the patient was advised to wear a Medic Alert bracelet. However, I felt uneasy recommending a procedure that was very expensive (\$6,000 USD), invasive (need for another anesthesia, relative disability for 3–4 days), and burdensome (3-h flight to nearest biopsy center), with no clear benefits for the patient. In their article, Tautz *et al.* wrote: “We cannot fault a clinician who wishes to give a nontriggering anesthetic to a person who has had contracture testing and who is not susceptible to MH.” Thank you for not blaming me for playing

* www.mhaus.org. Accessed March 19, 2010.

it safe! But perhaps we should fault an anesthesiologist who unnecessarily canceled elective procedures because he or she was uncomfortable anesthetizing the patients before their MH status had been established by biopsy. The debate about the usefulness of the muscle contracture test has had a long history.⁴ In our era of evidence-based medicine and cost-effective analyses, should we not also reevaluate muscle biopsy testing for MH?

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All Valve Malfunctions Are Not the Same

To the Editor:

We congratulate Tautz and colleagues¹ on an insightful case presentation of malignant hyperthermia and systematic analysis of increased end-tidal carbon dioxide. We write to clarify a detail in their analysis that may be misunderstood.

The capnograph/meter is an essential tool for deciphering the etiology of increased carbon dioxide during anesthesia. As a point of clarification, inspiratory and expiratory valve malfunctions in anesthesia breathing circuits do not result in identical capnograms, as shown in figure 1 of the article. The capnogram in the upper left panel of this figure shows increased carbon dioxide with increased inspiratory baseline. Although this is accurate for a stuck expiratory valve, the capnogram of a stuck inspiratory valve is actually quite different, because there is a dampening of the inspiratory downstroke on the capnogram, which does in fact get to zero.²

Consider a circuit with the inspiratory valve removed. In this scenario, the exhaled breath with carbon dioxide-rich gas is exhaled about equally into both limbs of the breathing circuit; therefore, about half of the exhaled tidal volume partially fills the inspiratory limb. With the next breath, the carbon dioxide-rich gas from the inspiratory limb is re-inspired first, followed by fresh gas without carbon dioxide. The capnometer thus displays a sluggish inspiratory down-

stroke (or a β angle greater than 90°).² The inspiratory baseline will therefore return to zero during the second half of inspiration. These capnogram differences may seem subtle but can be critical in the identification of machine fault etiologies.

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In Reply:

Dr. Kwetny argues that contracture testing has limited usefulness in the management of patients who might be susceptible to malignant hyperthermia (MH). As a biologic test, 98% sensitivity is commendable. Very few commonly used diagnostic screening tests approach that level of accuracy. We formulate anesthesia plans on a daily basis using tests with much poorer positive predictive value (e.g., electrocardiogram, echocardiogram, creatinine, hematocrit).

Furthermore, contracture testing has been a useful tool to identify genetic mutations in 60–80% of MH families. Because the number of identified causative mutations in MH families has increased over the past decade we now can offer noninvasive and less expensive genetic testing to many MH families.

In addition, we disagree that a nontriggering anesthetic is 100% safe (e.g., propofol infusion syndrome, awareness). Volatile anesthetics have real and unique benefits. We wonder whether, because of his belief that a nontriggering anesthetic is 100% safe, Dr. Kwetny provides nontriggering anesthetics to all of his patients, regardless of MH status.

What is most disturbing is the reticence not to consider the test at all and label a patient MH-susceptible based solely on clinical criteria, especially when those criteria are minimal. Permitting patients to be labeled MH-susceptible by individual clinicians who might not have the requisite expertise can subject that patient and his or her family to the hardship of finding clinicians who will care for them.

We counsel numerous patients referred for potential testing with vague personal or family history of potential MH. These are patients who have tried to obtain anesthetic care in the community and have been told that they cannot be anesthetized until they have been tested for MH. We are at a loss to explain why so many anesthesiologists are reluctant to provide nontriggering anesthetics before a biopsy procedure, especially if that is exactly what they will provide after the biopsy.