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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References

1. Tautz TJ, Urwyler A, Antognini JF, Riou B: Case scenario: Increased end-tidal carbon dioxide. A diagnostic dilemma. ANESTHESIOLOGY 2010; 112:440–6
2. Kwetny IM, Finucane BT: Negative arterial to end-tidal carbon dioxide gradient: An additional sign of malignant hyperthermia during desflurane anesthesia. Anest Analg 2006; 102:815–7
3. Larach MG, Localio AR, Allen GC, Denborough MA, Ellis FR, Gronert GA, Kaplan RF, Muldoon SM, Nelson TE, Ording H: A clinical grading scale to predict malignant hyperthermia susceptibility. ANESTHESIOLOGY 1994; 80:771–9
4. Larach MG: Should we use muscle biopsy to diagnose malignant hyperthermia susceptibility? ANESTHESIOLOGY 1993; 79:1–4

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All Valve Malfunctions Are Not the Same

To the Editor:

We congratulate Tautz and colleagues[1] 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 reinspired first, followed by fresh gas without carbon dioxide. The capnometer thus displays a sluggish inspiratory downstroke (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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References

1. Tautz TJ, Urwyler A, Antognini JF, Riou B: Case scenario: Increased end-tidal carbon dioxide: A diagnostic dilemma. ANESTHESIOLOGY 2010; 112:440–6
2. van Genderingen HR, Gravenstein N, van der Aa JJ, Gravenstein JS: Computer-assisted capnogram analysis. J Clin Monit 1987; 3:194–200

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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.