Anesthesiology
83:1382–1383, 1995

Myotonias and Masseter Spasm: Not Malignant Hyperthermia?

To the Editor:—The report by Vita *et al.*[1] correlates the inherited disorder myotonia fluctuans and its specific chromosomal abnormality with muscle rigidity produced by succinylcholine and with malignant hyperthermia (MH)-positive muscle contracture responses to halothane. The correlation weakens with their presumed logical tie to clinical episodes of MH. As the accompanying editorial[2] states, there is contradictory evidence for a tie to MH, and the anesthetic complications do not appear to be MH. Most of the evidence regarding myotonic disorders and MH do not support a connection, *i.e.,* a positive biopsy is not enough evidence by itself; there must be undisputed evidence for clinical episodes of MH.[3]

The criteria for what constitutes a "clinical episode of MH" seem to have become less certain. Rigidity *per se,* even that involving the entire body,[4] is insufficient evidence for MH, as has been recognized with succinylcholine and other choline derivatives in myotonia since Orndahl described it in the early 1950s.[5,6] Vita *et al.* admit that the clinical tie to MH is weak: "certain forms (of masseter spasm) do not progress to a recognizable hypermetabolic state." After the anesthetic has been aborted because of trismus, "there is usually little clinical evidence to distinguish which patients would have proceeded to a hypermetabolic response."

Goats inbred for myotonia congenita do not demonstrate MH when exposed to halothane and succinylcholine.[7] They demonstrate the characteristic rigidity and have an associated modest increase in carbon dioxide production and oxygen consumption related to the metabolic demands of the rigidity. Two human cases seem to contradict the findings in goats: Haberer *et al.*[8] report a boy with myotonia congenita who experienced severe rigidity, fever, and acidosis 7 h after an orthopedic procedure, with eventual arrest and CK of 11,000. Was this rhabdomyolysis that led to hyperkalemia? Patients with myotonia can exhibit greater hyperkalemia with exercise,[9] which may be a predisposing factor if excess muscle activity develops (related to their myotonia) and they cannot relax. In this case, the evidence for MH *per se* is not convincing, although the blood gas and temperature findings are consistent with MH. This boy had been given oral dantrolene preoperatively and what appear to be nontriggering agents, and the complications in the late postoperative period were severe. Saidman *et al.*[10] describe a young girl with presumed myotonia congenita who experienced typical MH and died. However, data from Moulds and Denborough on normal biopsy findings in patients with myotonia and their analysis of Saidman *et al.*'s case weaken the argument for a direct tie between MH and myotonia.[11] Further, a 1978 review on relaxants notes that the contracture produced by succinylcholine in normal patients (their fig. 2) is exaggerated in patients with myotonia.[12]

Several additional reports that include myotonia in their considerations state that the association with MH is either none, uncertain, or of uncertain extent.[13–17] In other words, the contracture produced by succinylcholine in those muscle groups affected by myotonia complicates the evaluation of the patient, and a cautious approach would include MH. This is an appropriate course of action early in the consideration of a case.

Myotonia should be considered "myotonias," an array of muscle disorders caused by a variety of genetic aberrations in chloride and sodium channels and perhaps yet to be discovered mutations in other ion channels.[18] Consequently, variation in clinical behavior to depolarizing stimulation by succinylcholine may be anticipated, in some cases making differentiation from "true MH" (also a multigenetic disorder) impossible in its early stages.

The findings in myotonia fluctuans appear to confirm this opinion, and the conclusion from the report of Vita *et al.* is that they have demonstrated a mutation associated with myotonia fluctuans and that this is one explanation for masseter spasm occurring with the use of succinylcholine.

Gerald A. Gronert, M.D.
Department of Anesthesiology
University of California, TB 170
Davis, California 95616

References

1. Vita GM, Olckers A, Jedlicka AE, George AL, Heiman-Patterson T, Rosenberg H, Fletcher JE, Levitt RC: Masseter muscle rigidity associated with glycine[1306]-to-alanine mutation in the adult muscle sodium channel α-subunit gene. ANESTHESIOLOGY 82:1097–1103, 1995
2. Iaizzo PA, Lehmann-Horn F: Anesthetic complications in muscle disorders. ANESTHESIOLOGY 82:1093–1096, 1995
3. Gronert GA: Controversies in malignant hyperthermia (editorial). ANESTHESIOLOGY 59:273–274, 1983
4. Heiman-Patterson T, Martino C, Rosenberg H, Fletcher J, Tahmoush A: Malignant hyperthermia in myotonia congenita. Neurology 38:810–812, 1988
5. Orndahl G: Myotonic human musculature: Stimulation with depolarizing agents. Acta Med Scand 172:739–751, 1962
6. Orndahl G: Myotonic human musculature: Stimulation with depolarizing agents: II. A clinico-pharmacological study. Acta Med Scand 172:753–765, 1962
7. Newberg LA, Lambert EH, Gronert GA: Failure to induce malignant hyperthermia in myotonic goats. Br J Anaesth 55:57–60, 1983
8. Haberer J-P, Fabre F, Rose E: Malignant hyperthermia and myotonia congenita (Thomsen's disease). Anaesthesia 44:166, 1989
9. Wevers RA, Joosten EMG, van de Biezenbos JBM, Theewes AGM, Veerkamp JH: Excessive plasma K+ increase after ischemic exercise in myotonic muscular dystrophy. Muscle Nerve 13:27–32, 1990
10. Saidman LJ, Havard ES, Eger EI: Hyperthermia during anesthesia. JAMA 190:1029–1032, 1964
11. Moulds RFW, Denborough MA: Myopathies and malignant hyperpyrexia. Br Med J 3:520, 1974

12. Mitchell MM, Ali HH, Savarese JJ: Myotonia and neuromuscular blocking agents. ANESTHESIOLOGY 49:44–48, 1978

13. Brownell AKW: Malignant hyperthermia: relationship to other diseases. Br J Anaesth 60:303–308, 1988

14. Lehmann-Horn F, Iaizzo PA: Are myotonias and periodic paralyses associated with susceptibility to malignant hyperthermia? Br J Anaesth 65:692–697, 1990

15. Lehmann-Horn F, Knorr-Held S: Muscle diseases relevant to the anesthetist (sic). Acta Anaesthesiol Belg 41:113–118, 1990

16. Neumann GG, Kopman AF: Dyskalemic periodic paralysis and myotonia. Anesth Analg 76:426–428, 1993

17. Russell SH, Hirsch NP: Anaesthesia and myotonia. Br J Anaesth 72:210–216, 1994

18. Rudel R, Ricker K, Lehmann-Horn F: Genotype-phenotype correlations in human skeletal muscle sodium channel diseases. Arch Neurol 50:1241–1248, 1993

(Accepted for publication September 15, 1995.)

Anesthesiology
83:1383, 1995

Detection of Carbon Monoxide with Mass Spectroscopy during Anesthesia

Peter H. Breen, M.D., F.R.C.P.C.
Department of Anesthesiology
University of California, Irvine, Medical Center
101 City Drive South
Orange, California 92613-1491

To the Editor:—I read with interest the case report in which Woehlck *et al.*[1] proposed that the detection of mixed halogenated agents during isoflurance anesthesia is a clinically useful sign to suggest the presence of carbon monoxide. The authors correctly note that clinical mass spectrometry cannot directly measure carbon monoxide because its molecular weight is the same as nitrogen and its fragmentation products are similar to those of carbon dioxide.

Yet, mass spectrometry may be helpful in the direct detection of changing carbon monoxide fractions in respiratory gas. In canine studies in which carbon monoxide poisoning was induced by injection of a molar amount of the gas into the inspired limb during closed-circuit anesthesia,[2] we monitored complete uptake of carbon monoxide from the circuit by mass spectrometry (model 6000, Ohmeda, Madison, WI). In that study, carbon monoxide in the circuit was qualitatively detected as spillover into and increase of the nitrogen channel (same molecular weight) and the carbon dioxide channel (conversion to C^{12} fragment). As complete pulmonary uptake of carbon monoxide occurred, the nitrogen and carbon dioxide mass spectrometry signals decreased to baseline levels.

References

1. Woehlck HJ, Dunning M, Gandhi S, Chang D, Milosavljevic D: Indirect detection of intraoperative carbon monoxide exposure by mass spectrometry during isoflurance anesthesia. ANESTHESIOLOGY 83: 213–217, 1995

2. Breen PH, Isserles SA, Westley J, Roizen MF, Taitelman UZ: Combined carbon monoxide and cyanide poisoning: A place for treatment? Anesth Analg 80:671–677, 1995

(Accepted for publication September 18, 1995.)

Anesthesiology
83:1383–1384, 1995

In Reply:—I agree with Breen that carbon monoxide may be detected and quantitated directly by mass spectrometry in certain situations. However, significant human toxicity may result from less than 1,000 ppm carbon monoxide.[1] This may be as much as 100 times less than the amount administered in the study by Breen *et al.*[2] The potential sources of interference with the technique of direct measurement of carbon monoxide by mass spectrometry includes changing levels of nitrogen and carbon dioxide as well as the presence of anesthetic agents and other gases. The changes in concentrations of nitrogen and carbon dioxide found in breathing circuits are likely to be many times greater than the amounts of carbon monoxide, which may result from anesthetic breakdown. An increase in indicated nitrogen may be due to the presence of carbon monoxide or the presence of nitrogen from air leaks, patient denitrogenation, or the