

Value of Mass Spectrometry in Early Diagnosis of Malignant Hyperthermia

To the Editor:—The fact that CO₂ production is markedly elevated in malignant hyperthermia (MH) was observed first in the swine model¹ and later in human patients.^{2,3} In 1981 Triner and Sherman⁴ suggested that continuous measurement of expired CO₂ could be useful for early diagnosis of MH in susceptible patients. In October 1984 two cases were reported⁵ of susceptible patients who had MH develop while end-tidal CO₂ tension (PET_{CO₂}) was being monitored with a capnometer. The following case illustrates when the mass spectrometer was instrumental in the diagnosis of an episode of MH.

A 4-year-old, 18-kg boy presented for repair of facial lacerations. He had no significant medical history and no family history of any anesthetic difficulties. Induction and intubation with succinylcholine were uneventful, and anesthesia was maintained with halothane, nitrous oxide, and oxygen. Monitoring included precordial stethoscope, BP cuff, ECG, rectal temperature, and the mass spectrometer.* After two hours of surgery, the patient suddenly had tachypnea develop, with deep rapid respiration, tachycardia from 120 to 170 bpm, and a rise in PET_{CO₂} from 30 to 90 mmHg over 10 min. The breathing circuit was intact and air exchange unimpeded. The mass spectrometer display panel revealed normal values for inspired and expired oxygen, an absence of nitrogen and inspiratory and expiratory values appropriate for the administered concentration of halothane. The temperature was 37° C, having risen from 35.2° C over the prior 60 min. The clinical situation was that of rapidly worsening hypercarbia, in spite of the patient's attempt to improve CO₂ elimination by increasing minute volume. The presumptive diagnosis of MH was made, and halothane and nitrous oxide were discontinued, ventilation continuing with 100% oxygen. Immediately upon turning off the halothane, the heart rate, PET_{CO₂}, and respiratory rate began to decrease, returning within 10 min to near baseline levels. Arterial blood gas values were pH_a 7.28, Pa_{CO₂} 56.2 mmHg, Pa_{O₂} 403.8 mmHg, and BE -1.4 mmEq/l. Dantrolene sodium 4 mg/kg was given iv over the next 2 h. The

CPK was 2310 U/L (nl (35–232 U/L). The patient continued to improve and recovered without sequelae.

In our case the presenting signs, tachycardia and tachypnea, were nonspecific and the mass spectrometry data helped to narrow the differential diagnosis. The adequate expired halothane concentration made an inadequate plane of anesthesia unlikely. The adequate inspired and expired oxygen concentrations made hypoxia unlikely. The high PET_{CO₂} values were helpful in identifying a hypermetabolic process as the etiology of the tachypnea and tachycardia.

The critical importance of early recognition and treatment in reducing the mortality and morbidity of MH is widely recognized. This case illustrates that the mass spectrometer can be a powerful tool in the early diagnosis of MH.

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* System for Anesthetic and Respiratory Analysis, Allied Health Care Products, Inc.