Malignant Hyperpyrexia in the Cat

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Until recently, malignant hyperpyrexia secondary to halothane–muscle relaxant anesthesia seemed to plague only man and a strain of swine.[1,2] Then, last year, a case in a dog was reported.[3] The list of susceptible mammals must be lengthened once again, for we encountered malignant hyperpyrexia in a cat anesthetized with halothane and paralyzed with decamethonium.

A healthy unmedicated 4.2-kg adult male black cat was anesthetized with nitrous oxide, 67 per cent, and halothane, 1.5 per cent, for routine laboratory studies of the electroencephalogram. The trachea was intubated, the bladder catheterized, a femoral artery and vein cannulated, and the animal placed on a circulating warm water (38 C) blanket. Lactated Ringer's solution with 5 per cent dextrose was delivered at a rate of 7–10 ml/kg/hr.

Eighty minutes after anesthetic induction, mid-esophageal temperature was 37.8 C and end-expired CO_2 with spontaneous respiration was 6.5 per cent. At this point, decamethonium, 1 mg, was given, and mechanical ventilation at a rate of 28/min and 10 ml/kg tidal volume was begun. In our laboratory this minute volume ordinarily maintains end-expired CO_2 at about 4.5 per cent. The lungs were clear to auscultation and expanded well on inflation.

Ten minutes later body temperature had risen to 41 C, so the heating blanket was turned off; the end-expired CO_2 meter indicated 9.6 per cent. Thinking that nitrous oxide might be contributing to the elevated CO_2 reading, we changed the ventilatory gas mixture to halothane, 2 per cent, in oxygen. Convinced then that the CO_2 reading was correct, we increased tidal volume by 50 per cent. Blood pressure was 125/75 torr and heart rate 220/min; EKG showed bilobar T-waves and severely depressed S-T segments.

One hour after decamethonium, and with minute volume double that usually used, end-expired CO_2 was 8.5 per cent. Arterial blood values were: P_{CO_2} = 58 torr, P_{O_2} = 118 torr, and pH = 7.17. The mechanical ventilator was replaced in case it might be defective, but Pa_{CO_2} values did not decrease. Blood pressure began to decrease (70/40 torr), and heart rate increased to 420/min, with tall, pointed T-waves on the EKG.

One and a half hours after decamethonium, the electronic temperature gauge (Yellow Springs) indicated well past the maximum 42 C; a mercury thermometer inserted rectally read 44 C. The diagnosis of malignant hyperpyrexia now was obvious. Vigorous cooling with ice bags and massive hyperventilation were started, followed by sodium bicarbonate, 2.5 mEq/kg, given in divided doses. A second arterial blood sample drawn 15 minutes after the start of cooling showed: P_{CO_2} = 35 torr, P_{O_2} = 209 torr, pH = 7.19, with rectal temperature still 44 C.

Two hours after the initial dose of decamethonium the limb muscles were still flaccid (normally we repeat the C10 every 1 to 1 ½ hours). Rectal temperature remained 44 C, heart rate was 360/min, and blood pressure 30/20 torr; runs of bigeminal and isolated ventricular complexes appeared on the EKG. Plasma sodium and potassium values were 164 and 9.5 mEq/l, respectively (feline normals are 153 and 4.4 mEq/l, respectively). Profound cortical depression was evidenced by large slow EEG waves. Halothane was discontinued and ventilation continued with oxygen only. We then gave procaine, 10 mg/kg, intravenously. This briefly slowed heart rate to 210/min; as the blood pressure decreased from 30/15 to 25/15 torr, no further procaine was given.

After half an hour, cooling appeared to be effective, as the esophageal temperature returned to 42 C. Fifteen minutes later esophageal temperature was 40 C, CO_2 5 per cent,

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blood pressure 40/20 torr, and heart rate 240/min. A third blood-gas analysis showed: P_{CO_2} = 35 torr, P_{O_2} = 164 torr, pH = 7.22. The temperature then rapidly returned to normal. Blood pressure remained low, however, and barely increased after repeated doses of ephedrine, 1 mg/kg, intravenously.

Three hours after decamethonium we attempted to recreate events by restarting halothane, 2 per cent, and giving a second dose of decamethonium, 1 mg. But the temperature remained between 37 and 37.5 C. Subsequently the blood pressure became barely perceptible, the EKG turned grossly irregular, and the animal died. Unlike the 30 minutes or more of postmortem flaccidity seen in identically anesthetized animals, this cat developed extreme rigor within 10 minutes. This is similar to the reported case of malignant hyperthermia in the dog which developed rigor mortis within 2 minutes after cardiac arrest.

The clinical protocol described here follows that reported for the malignant hyperpyrexia syndrome in man and swine. To be sure, the syndrome must be extremely rare in the feline, as we have not seen it in the well over 500 cats anesthetized in like manner before. In fact, cats decrease their body temperature by several degrees Celsius when breathing halothane.[4] While the time course of the hyperthermic response would seem to incriminate decamethonium, we cannot with certainty exclude halothane or the halothane–decamethonium sequence as the precipitating factor.

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Estimating Allowable Hemodilution

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Deliberate hemodilution with noncellular colloid volume expanders avoids the risks and expense of replacing surgical blood loss with whole bank blood. The limit to such replacement is usually set by concern for oxygen transport as measured by hemoglobin or hematocrit. Some patients tolerate isovolemic hemodilution to hematocrit values as low as 25 per cent. For a given patient for whom one knows the preoperative blood volume, the hematocrit, and the extent to which one will permit hemodilution, serial hematocrits would guide replacement when large volumes are replaced. In the absence of repeated hematocrit or hemoglobin measurements, a variety of rules of thumb, of varied rational and physiologic bases, which are intended to guide the anesthesiologist's decision to begin whole blood replacement, are in use. A common one neglects the continuous decrease in hematocrit. This "oversimplified" rule increasingly underestimates the true hematocrit as loss continues, and can be improved upon. Our work consists of three parts: a) a model of the blood volume, permitting prediction of an allowable loss for a given decrement in hematocrit; b) a clinical verification of the

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Received from the Department of Anesthesia, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104. Accepted for publication July 12, 1974. Supported in part by USPHS Research Center Grant 5-P01-GM-15430-06, and Research Training Grant 5-T01-GM-00215-15 from the National Institute of General Medical Sciences, National Institutes of Health.