CASE REPORTS

Unusual Cardiovascular Stability during Deep Halothane Anesthesia

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CASE REPORT

The patient, a 42-year-old woman, was scheduled for a resection of an adenocarcinoma of the sigmoid colon. She had been taking 0.5 mg digoxin daily for paroxysmal atrial tachycardia of eight years' duration. Results of preoperative physical examination were within normal limits. The patient weighed 59 kg; blood pressure was 110/80 mm Hg, pulse rate 74 beats/min. Electrocardiogram revealed rare atrial premature beats and nonspecific ST and T wave changes compatible with digitalis effect. Blood, urine and hematocrit were normal.

Premedication consisted of Demerol, 50 mg, and Vistaril, 50 mg, intramuscularly. Prior to induction of anesthesia, the blood pressure was 150/90 mm Hg, pulse rate 90/min. Following denitrogenation, 175 mg thiopental and 80 mg succinylcholine were given intravenously. The trachea was intubated and the flow of oxygen was reduced to 300 ml/min. Liquid halothane was injected into the expiratory breathing tube of the circle system at a rate calculated to equal the circuit and tissue uptake of a 59-kg patient. A cannula in the radial artery provided for intermittent arterial blood sampling for determination of *p*H, $Paco_2$, Pa_{O_2}, and halothane concentration. A blood sample was obtained, and tonometry revealed the blood/gas partition coefficient to be 2.5.

Following intubation, the pulse rate rose to 120/min, and ventricular bigeminy was evident. Within six minutes, the halothane concentration in arterial blood reached 12 mg/100 ml, and the bigeminy converted to a sinus rhythm of 70/min. The blood pressure varied between 100/80 and 90/70 mm Hg. When the peritoneum was opened 25 minutes after induction, the pulse rate was 90/min and the arterial blood halothane concentration was 32 mg/100 ml. The halothane concentration over the next 40 minutes varied between 33 and 35 mg/100 ml. The rate of administration of liquid halothane was reduced to 60 per cent of the predetermined dose. Seventy-six minutes after induction, the concentration of halothane in arterial blood was 29 mg/100 ml, and at 98 minutes, 23 mg/100 ml. Arterial *p*H and blood gases were analyzed at 36 minutes, 57 minutes, and 76 minutes. Pa_{O_2} values were 370, 310, 320 mm Hg; $Paco_2$ values 25.5, 26.2, 30.0 mm Hg; *p*H values 7.45, 7.43, 7.37. The Air-Shields ventilator–ventimeter remained on assist mode, and throughout this interval, the respirator assisted ventilation. Systolic blood pressure ranged between 90 and 120 mm Hg; diastolic, between 70 and 80 mm Hg; the pulse rate varied between 100 and 110/min.

When the halothane concentration in arterial blood rose above 32 mg/100 ml, the patient's face became congested, but good capillary filling prevailed.

During the remainder of the four-hour procedure, arterial halothane concentration ranged between 22 and 18 mg/100 ml. Blood pressure ranged from 120/90 to 90/70 mm Hg. Estimated blood loss was 600 ml, and fluid replacement consisted of 1,500 ml of crystalline fluids and 500 ml of whole blood.

At the time of extubation and transfer to the recovery room, the arterial blood concentration of halothane was 7 mg/100 ml, and the patient was unresponsive. However, within 20 minutes she was able to answer questions. Postoperatively, she experienced two episodes of paroxysmal atrial tachycardia with 2:1 block. On the ninth postoperative day, she was discharged.

DISCUSSION

An overdose of halothane results in cardiovascular depression and is manifested by hypotension, sinus bradycardia, and cardiac arrest.[1] Halothane is also a potent respiratory depressant.

For practical reason, it is important to establish the inspired or blood concentration of halothane which in man constitutes a relative or absolute overdose. Overdose, together with individual variability, can be established only by inferences from animal experiments and by case reports.

The arterial blood concentrations in 28 surgical patients receiving halothane–oxygen anesthesia were reported by Lowe and Beckman.[2] Three patients sustained cardiovascu-

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lar collapse at arterial concentrations between 21 and 34 mg/100 ml. Of the remaining four patients whose arterial blood halothane concentrations rose above 20 mg/100 ml, all had significant reductions in blood pressure. The highest arterial blood concentration without cardiovascular collapse was 28 mg/100 ml; this was associated with a 40 per cent decrease in systolic blood pressure.

The closed-system technique employing programmed injection of liquid halothane was found to produce a blood halothane concentration of 10–20 mg/100 ml, based on 59-kg body weight and a normal distribution of blood flow. With this technique, as well as with any technique employing halothane as the major agent, the patient's respiratory and cardiovascular responses are critical for evaluation of the appropriateness of the selected dose.

The case reported here had several interesting features. For one, although the patient had organic heart disease manifested by paroxysms of atrial tachycardia, with an electrocardiogram showing premature atrial contractions and digitalis effect, her only arrhythmia while under anesthesia occurred during induction, before a substantial blood concentration of halothane was achieved. Second, in spite of a sustained high concentration of halothane in the blood, blood pressure did not fall below 80 per cent of the previous day's value. Third, in the absence of atropine, the pulse rate did not fall below 60/min and, during most of the anesthetic course, remained between 90 and 110/min. Inspiratory effort, which was maximally assisted, was retained even at the patient's highest concentration of halothane in blood, the arterial Pa_{CO_2} never rising above 30 mm Hg. The only sign of circulatory instability was the facial flush which occurred when the halothane concentration rose above 32 mg/100 ml. Even in the presence of this flush there was brisk capillary filling.

In this case, the usual cardiovascular signs of depth of halothane anesthesia could not be relied upon. Furthermore, it has been demonstrated that one individual sustained a halothane blood concentration of 35 mg/100 ml without severe cardiovascular depression.

References

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Hyperthermia under Anesthesia with Regional Muscle Flaccidity

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The development of severe hyperpyrexia in patients under general anesthesia has recently been reported.[1, 2, 3] The etiology of the syndrome is still obscure, but it has been suggested that genetic factors may play a role,[4, 5] or that pathological biochemical mechanisms, such as the uncoupling of oxidative phosphorylation,[6, 7] may occur. Increased muscle tone with hyperpyrexia following the administration of succinylcholine has been a frequent finding.[8, 9, 10]

In a study undertaken to relate the degrees of muscle fasciculation and muscle pain after the administration of succinylcholine, as measured by an increase in muscle enzyme activity, there was a sevenfold increase in enzyme activity when halothane was used.[11] This enzyme, creatine kinase (creatine phosphokinase) is generally assumed to be elevated when muscle cells are injured. In conjunction with these biochemical findings the case reported here suggests that muscle enzyme function may be altered peripherally in susceptible patients by depolarizing muscle relaxants and/

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