Heart block after induction of anesthesia in a child

THOMAS E. SCHULTZ, CRNA, MS
Glasgow, Montana

A case of complete heart block occurred after induction of halothane anesthesia in a previously healthy child. The patient underwent repair of an umbilical hernia under general anesthesia. After a standard halothane, nitrous oxide, and oxygen mask induction, complete heart block was noted on the electrocardiographic monitor. Atropine and 100% oxygen were administered, and sinus tachycardia resulted. With the immediate stabilization of the patient's condition, the surgical team agreed to proceed with the case. After deepening of the level of anesthesia, first with halothane and then with desflurane and easy intubation of the trachea, complete heart block again was noted. Oxygen was administered at 100%, sinus tachycardia resumed, the case was canceled, and the patient emerged from anesthesia without further incident. The patient had an uneventful recovery and was discharged to home.

Key words: Bradycardia, halothane, heart block, pediatric.

Introduction

Bradycardia is a well-known adverse effect of halothane anesthesia in children. A case of heart block that seemed to be caused by surgical stimulation rather than purely by anesthesia was reported in a child during anesthesia.1 This is a report of complete heart block in a previously healthy child who received halothane.

Case summary

The patient was a 3-year-old boy with a history of an asymptomatic umbilical hernia. He was scheduled to undergo an elective repair of the hernia on the morning of surgery, was admitted to the outpatient surgery department. During the preoperative history and physical examination, a mild pectus excavatum was noted. The patient was alert and calm. Heart sounds were regular without murmurs, clicks, or rubs, and the lungs were clear. With the exception of the hernia and the pectus excavatum, the physical examination findings were normal. A negative surgical and anesthesia history was reported along with no history of familial anesthesia complications. He was receiving no medications. The preoperative hemoglobin level was 12.2 g/dL.

In the operating room, electrocardiography, pulse oximetry, and noninvasive blood pressure monitors were applied. After baseline vital signs were obtained, a calm, cooperative mask induction was performed using halothane with 70% nitrous oxide in oxygen. The halothane was administered in 0.5% increments every 2 to 3 breaths until a 4% inspired concentration was achieved. After a satisfactorily deep level of anesthesia was obtained and easy controlled mask ventilation was achieved, the operating room nurses were instructed to gain intravenous access. A sudden onset of complete heart block occurred 9 minutes after the start of the inhalation induction and before venipuncture. No recording of the dysrhythmia was obtained as efforts to abolish the heart block were the priority. The halothane and nitrous oxide were immediately turned off, and the patient was ventilated with 100% oxygen. Atropine, 0.4 mg, was adminis-
tered intramuscularly in the left anterior thigh while a 22-gauge intravenous line was started by the surgeon. Within a minute, the complete heart block was abolished and sinus tachycardia was noted.

Once the patient’s condition was stabilized, the decision to proceed was made. The patient’s anesthetic level was deepened initially with 4% inspired halothane. Because of the patient’s initial response to halothane, after 2 ventilations or approximately 5 seconds, the halothane was turned off, and 8% desflurane was administered for approximately 20 seconds. After a satisfactorily deep level of anesthesia and easy mask ventilation was achieved, the patient was gently intubated with a 5.0 uncuffed endotracheal tube.

Complete heart block again was noted (Figure 1). The patient was ventilated with 100% oxygen, the case was canceled, and the patient’s family physician was summoned for consultation. The patient’s cardiac rhythm spontaneously converted to sinus tachycardia (Figure 2), and the patient emerged from anesthesia and was extubated without further complications.

An electrocardiogram done in the postanesthesia care unit was normal. After discharge, the patient was referred to a pediatric cardiologist. The workup included a 12-lead electrocardiogram, an echocardiogram, and 2 Holter monitor studies. After evaluation and further consultation with an electrophysiology specialist, no evidence of conduction abnormalities or underlying cardiovascular disease was found.

**Discussion**

Bradycardia in children usually is caused by hypoxemia, disease, inhalational agents such as halothane, or the surgery itself. Another well-known cause of bradycardia and dysrythmias in children is succinylcholine administration for laryngoscopy. In the present case, because the history and the cardiology workup revealed no underlying disease, cardiac disease was not the cause of the dysrhythmia.

Throughout the induction, the patient’s color remained pink, the oxygen saturation via the pulse oximeter consistently read 100%, and his blood pressure was stable before the heart block. Therefore, hypoxemia was not a factor.

Halothane, in the absence of adjuvant drugs or surgical stimulation, produces negative inotropic and chronotropic effects. Clinically, however, bradycardia is a well-known adverse effect of halothane in children. It is reasonable, for the present case, to assume that the bradyarrhythmia was drug induced. Since succinylcholine was not used, the only drugs the patient received were inhalational agents.

Rowe and Garbin were the first to report complete heart block in a child during abdominal surgery. The episode in the present case, however, differs because it occurred before surgical stimulation. There was no painful stimulation that could have caused a vagotonic reaction as the operating room nurses had not attempted venipuncture at the time of the heart block. Rowe and Garbin believed that the atrioventricular conduction abnormalities were related to traction of the abdominal contents in the case they reported.

Because the patient in the present case was free of cardiac disease, hypoxemia, and surgical stimulation, halothane is implicated as the cause of the complete heart block. Atlee and Rusy suggested that halothane-induced arrhythmias could be a result of impaired conduction, as they found halothane to slow conduction from the atria to the bundle of His. In the present case, the child most likely suffered a preponderance of parasympathetic activity or drug-induced depression of impulse conduction due to the induction dose of halo-

---

**Figure 1. Second episode of complete heart block**

![Graph showing electrocardiogram](image-url)
than. This previously healthy child experienced an episode of complete heart block after induction of halothane anesthesia in the absence of adjuvant drugs, hypoxemia, or surgical stimulation. To my knowledge, this has not been reported in the literature.

Epilogue

The patient returned 4 months later for repair of the umbilical hernia. After a preoperative oral dose of 0.02 mg/kg dose of atropine, he was induced lightly with halothane, intubated after administration of rocuronium, and underwent an umbilical herniorrhaphy without incident. He was discharged from the hospital the same day.

REFERENCES


AUTHOR

Thomas E. Schultz, CRNA, MS, is the chief of Anesthesia at Frances Mahon Deaconess Hospital in Glasgow, Montana.

ACKNOWLEDGMENTS

I thank Ann Williams, MD, FACS, Dennis Rugeic, MD, and the medical staff at Frances Mahon Deaconess Hospital for their editorial assistance and encouragement while preparing this manuscript, and Janet Plant for assistance with the literature search.