Pulseless Ventricular Tachycardia During Office-Based Anesthetic in a Four-Year-Old Child

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A 4-year-old 16-kg boy presented for full mouth dental rehabilitation in a private pediatric dental office. The patient had no significant previous medical history. Upon sevoflurane induction by a dentist anesthesiologist, the patient converted from normal sinus rhythm to pulseless ventricular tachycardia. Advanced cardiac life support protocol was initiated. After 2 automatic external defibrillator shocks were delivered in conjunction with epinephrine administration, the patient returned to normal sinus rhythm. The patient was transported via emergency medical service paramedics to a local children’s hospital emergency room where he was observed uneventfully for 24 hours prior to discharge.

Key Words: Pulseless ventricular tachycardia; Cardiac arrest; Pediatric anesthesia; Advanced cardiac life support.

PREOPERATIVE ASSESSMENT

A 4-year-old boy was scheduled to undergo dental rehabilitation in a private dental office by means of a general anesthetic, the patient’s first. Three days prior to the appointment, after reviewing the patient’s medical record, the dentist anesthesiologist placed a phone call to the parents. The patient was reported to have a speech impediment and to be taking cetirizine as needed for seasonal allergies. No other significant history or medical findings were reported. There was also no family history of complications under general anesthesia. Guidelines regarding nothing by mouth were reviewed with both parents. The reported speech impediment was determined not to be secondary to oral anatomy or physiology. During auscultation, the patient’s lungs were clear and normal S1 and S2 cardiac sounds with regular rate and rhythm were appreciated. The patient’s weight was 16 kg and height 99 cm. The patient presented with a Mallampati class 1 (MP1) airway, although a complete airway evaluation was not done due to the poor cooperation of the patient. The anesthetic plan was reviewed with the parents who had no additional questions prior to consenting to general anesthesia for their son.

The parents were allowed to accompany their son for his rapid mask induction (8% sevoflurane, 50% N2O 50% O2). Following the induction, the parents left the operatory as had been previously discussed during the consent process. Standard American Society of Anesthesiologists monitors were placed, including an electrocardiogram (ECG), a pulse oximeter, an automated blood pressure cuff, and a temperature probe. With the patient breathing spontaneously, the eyes were then closed and protected with paper tape. The patient’s head was wrapped prior to placement of a shoulder roll. Intravenous (IV) access was established in the right antecubital fossa via a 22-gauge catheter. A warmed 4.5-mm Parker uncuffed nasoendotracheal tube was placed uneventfully.

DAY OF SURGERY

A routine preoperative evaluation was performed, which included another review of the patient’s medical history and focused anesthetic physical examination. No new historical findings were noted. The reported speech impediment was determined not to be secondary to oral anatomy or physiology. During auscultation, the patient’s lungs were clear and normal S1 and S2 cardiac sounds with regular rate and rhythm were appreciated. The patient’s weight was 16 kg and height 99 cm. The patient presented with a Mallampati class 1 (MP1) airway, although a complete airway evaluation was not done due to the poor cooperation of the patient. The anesthetic plan was reviewed with the parents who had no additional questions prior to consenting to general anesthesia for their son.

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There was a grade 1 view of the vocal cords and the intended endotracheal tube position was confirmed via visualization, auscultation, and sustained capnographic end-tidal CO$_2$. A dental floss ligated throat pack was then positioned, and the pediatric dentist was informed that surgical treatment could be initiated. Following the placement of the throat pack, the anesthetic gas was switched to 1.5% isoflurane and assisted intermittent positive pressure ventilation was started as the patient was hypoventilating/apneic from the induction.

Between 1 and 2 minutes later, while surgical staff was attempting to obtain the first intraoral radiograph, the dentist anesthesiologist noted the pulse-oximeter waveform had been lost. After repositioning the pulse-oximeter probe from toe to finger, a wide complex ventricular tachycardia at a rate of 220 on the ECG was noted. Immediately, the pediatric dentist and assistants were asked to step away from the patient. No pulse was detected from the right brachial or carotid arteries. American Heart Association Advanced Cardiac Life Support (ACLS) protocol was now initiated. All anesthetic gases except oxygen, delivered at 10 L/min, were discontinued. One dental assistant was directed to call emergency medical services (EMS) via a 911 call, while the second assistant was asked to hold the automatic external defibrillator (AED) for the dentist anesthesiologist. Concomitantly, the pediatric dentist was instructed to give 1 positive pressure breath via the anesthesia circuit bag every 8 seconds. The anesthesiologist continued to run the code, including starting chest compressions at a rate of 100/min. Predrawn epinephrine, 100 µg (0.00625 mg/kg), was administered by IV push as this dose was immediately available (the ACLS dose of 0.01 mg/kg, 160 µg, was not delivered). The AED was placed using pediatric pads and after rhythm analysis, a shock was delivered. This particular AED, Phillips Heartstart brand, delivers a preset dose of 50 J (3.125 J/kg for this patient) for each shock with the pediatric pads. The patient remained in pulseless ventricular tachycardia. Two more minutes of CPR continued and was followed with the administration of a second IV bolus of 100 µg of epinephrine. During cardiopulmonary resuscitation, pulse-oximeter waveform was noted following each chest compression, with a reading of approximately 85%. End tidal CO$_2$ was also noted on the monitor. The patient was then defibrillated via AED a second time and subsequently converted to normal sinus rhythm.

The patient’s vital signs immediately after the second defibrillation were as follows:

- heart rate: 120 beats/min;
- blood pressure: 124/77 mm Hg; and
- oxygen saturation: 100%.

EMS paramedics arrived approximately 10 minutes after the 911 call had been placed. The patient appeared to be clinically stable in all parameters. The case history was reviewed with the paramedics, while the patient was prepared for transport utilizing the original endotracheal tube. The anesthesiologist determined to continue to sedate the patient with propofol to optimize controlled transport to the nearby children’s hospital emergency room. Transferring the patient from the office to the ambulance consumed about 15 minutes, with an additional 15 minutes for transport to the emergency room. The dentist anesthesiologist continued to have primary responsibility for airway maintenance until the patient was accepted by emergency room personnel.

At the hospital, initial laboratory testing, including a chemistry 8 panel and complete blood count, was obtained and soon interpreted as within normal limits. A 12-lead ECG showed no signs of ST segment elevation or depression, and only a minor QT(c) prolongation of 460 ms. Troponin levels were less than 0.01. A chest x-ray showed slight peripheral interstitial edema. The patient soon emerged from anesthesia enough to be uneventfully extubated. A transthoracic echocardiogram revealed a left ventricular ejection fraction of 69.5% and no abnormal findings. Subsequent testing was uniformly within normal limits, including urinalysis, serial arterial blood gases, and complete blood count.

The patient was discharged after overnight observation and recovered without any adverse sequelae.

**DISCUSSION**

Cardiac arrest remains a relatively rare, albeit serious event during general anesthesia. The etiology of cardiac arrhythmias, specifically ventricular in origin, remains complicated and multifactorial. Causes can include physiologic or anatomic abnormalities, as well as injury to various organ systems, and can also be the result of certain medications. In an otherwise healthy patient in whom normal sinus rhythm converts to an arrhythmia, a quick investigation into each of the potential causes of the abnormal rhythm, including monitor dysfunction, should be initiated. In the case described herein, the patient sustained no traumatic event, thus eliminating trauma as a precipitating factor. The patient’s preoperatively reported speech defect as well as antihistamine use were also ruled out as preexisting factors leading to the cardiac arrest. No known anatomic abnormalities were noted, and cardiac auscultation was normal, likely implying normal valvular anatomy. Although not common, sevoflurane, especially in higher doses, has been reported to cause ventricular dysrhythmias as well as QT
prolongation based on reports from the World Health Organization, the Pediatric Perioperative Cardiac Arrest Registry, and various case reports. Sevoflurane-induced dysrhythmia seems the most likely explanation given the subsequent work-up below.

**Laboratory Blood Work**

In this particular case, laboratory values showing normal results for blood work, including the initial chemistry panel and the subsequent serial arterial blood gases and complete blood count, essentially ruled out electrolyte disturbance as the cause of the spontaneous ventricular tachycardia.

**Hospital Cardiac Monitoring**

A transthoracic echocardiogram performed for the patient during his stay at the hospital was normal. Interestingly, a 12-lead ECG showed no ST elevations or depressions, despite approximately 5 minutes of chest compressions and 2 AED delivered shocks. External trauma on the heart frequently, but not always, results in ST changes should the patient successfully convert back to normal sinus rhythm. Another postarrest finding worthy of note was the normal troponin level of <0.01 ng/ml despite the cardiac arrest and aggressive chest compression which can cause at least a minor troponin elevation due to the abnormal CPR trauma, in this case involving 2 defibrillations and 2 sequences of chest compression. A subsequent troponin level approximately 12 hours after admission was found to be at 0.01 ng/ml, indicating a minor, but insignificant elevation.

**Medications**

The patient was prescribed cetirizine (Zyrtec) as needed for seasonal allergy symptoms, but had not taken it in more than 1 month. He was, therefore, on no known medications at the time of the cardiac arrest. The patient’s parents also subsequently reaffirmed that, to the best of their knowledge, he had not ingested any other medication.

The anesthetic induction included 8% sevoflurane in a 50% N₂O/O₂ mixed gas flow at 6 L/min. After induction, the patient’s heart rate ran as high as 160 beats/min in sinus rhythm. However, as he progressed from stage 2 into stage 3 of anesthesia, his heart rate slowed to approximately 120 beats/min, at which time the straightforward intubation, without any associated hypoxia, took place.

Few reports of arrhythmias following sevoflurane induction exist. These papers describe conversion to supraventricular tachycardia, polymorphic and monomorphic ventricular tachycardia, and bradyarrhythmia occasionally progressing to supraventricular tachycardia. Situations such as reported herein, although documented, appear to be incredibly rare.

**Anatomic**

Occasionally, cardiac anatomic variations exist that can induce ventricular, junctional, and supraventricular arrhythmias. These types of anomalies are difficult to determine preoperatively unless the patient has given some clinical clue of symptomatic cardiac pathology. Additionally, with no apparent abnormal findings on this patient’s ECG or echocardiogram, an anatomic variation seems unlikely.

**CONCLUSION**

Dentist anesthesiologists must be ready to reasonably treat even uncommon emergencies in office-based settings. Urgent and potentially emergent situations can include anything from an unanticipated difficult airway to intraoperative malignant hyperthermia to silent aspiration intraoperatively or in the recovery area. Cardiac arrest, such as has been reported herein, has even been reported as secondary to IV or epidural catheter placement.

Dentist anesthesiologists, who have been providing office-based general anesthesia since December 1844, are well aware of the challenges faced in such environments even as our sister anesthesia professions are only recently beginning to discuss out of the operating room experiences. The unique challenges faced by this anesthesiology niche are best met with optimal vigilance and preparation.

**REFERENCES**


