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Original Contribution

Supraventricular tachycardia during pediatric anesthesia: a case series and qualitative analysis☆,☆☆,★

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Abstract

Study Objective: To perform a qualitative analysis of noncardiac patients who developed suspected intraoperative supraventricular tachycardia (SVT) during general anesthesia.

Design: Retrospective database analysis and chart review.

Setting: Operating room of a university-affiliated children's hospital.

Measurements: The records of children without cardiac disease who received general anesthesia at The Children's Hospital of Philadelphia from July 1998 through June 2011 were reviewed. Patients with heart rate values above 180 beats per minute were identified, as were specific medications or key words in the free-text fields of the anesthesia records that would be indicative of a tachyarrhythmia. Each case was reviewed by at least two authors; each patient was assigned a diagnosis classification of "highly suspicious" or "unlikely" SVT. The highly suspicious SVT cases were examined in detail to determine the specific aims.

Main Results: 36 subjects out of a total of 285,353 anesthetics administered during the study period were suspected by the anesthesia care team to have had an episode of intraoperative SVT: 22 were "highly suspicious" events, and 14 were "unlikely" events. The highly suspicious SVT events occurred in all phases of anesthesia, and none led to any hemodynamic instability. Effective treatments included vagal maneuvers, pharmacologic antiarrhythmics, or no treatment if the event resolved spontaneously before treatment. Six patients had outpatient follow-up and three received antiarrhythmic medications to control ongoing SVT.

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Conclusions: SVT during the intraoperative period in noncardiac pediatric patients was uncommon. When it occurred, it was not associated with clinically significant patient morbidity. For some patients, the anesthesia unmasked a predisposition for re-entrant SVT and those patients remained on maintenance antiarrhythmic therapy following discharge home.

1. Introduction

Arrhythmias occasionally occur during pediatric anesthesia. When halothane was used as the primary inhalational agent for pediatric anesthesia, approximately 5% of anesthetized children developed intraoperative arrhythmias [1]. Halothane sensitizes the heart to catecholamines and, especially in the presence of hypercapnia, demonstrates proarrhythmic properties that are predominantly ventricular in origin [2]. Since the widespread adoption of sevoflurane for inhaled induction in children, and desflurane for maintenance of general anesthesia, there has been the occasional development of supraventricular tachycardia (SVT) in previously healthy, anesthetized children.

The aim of this study was to perform a qualitative analysis of patients who developed episodes thought to be consistent with SVT during administration of general anesthesia, since the time that halothane was discontinued in 1998. The primary goal of the analysis was to determine the characteristics of these patients, determine possible causes of the tachycardic episodes, and investigate their short-term and long-term outcomes, including treatment. A secondary goal was to determine the reliability of the diagnosis provided by the anesthetic care team at the time of the episode by comparing the clinical circumstances with the classic diagnostic criteria for pediatric SVT.

2. Materials and methods

Permission was obtained from the Institutional Review Board (IRB) of the Stokes Research Institute of The Children's Hospital of Philadelphia to screen all anesthetic records of noncardiac patients who received general anesthesia from June 1998 through June 2011, to find episodes of tachycardia labeled by the anesthesia care team as "SVT". Requirement for written consent was waived by the IRB.

The reviewed data were contained in an electronic anesthetic record database (CompuRecord; Philips Medical Systems, Andover, MA, USA) that captures all anesthetic information, vital signs every 15 seconds, and any recorded free-text. Patients believed to have had an intraoperative SVT episode were identified by searching for: 1) patients with heart rate (HR) values above 180 beats per minute; 2) relevant administered medications (eg, adenosine, esmolol, labetalol, propranolol, lidocaine, and amiodarone); or 3) keywords from free-text narrative events suggestive of SVT during the anesthetic (including misspellings) that included the terms "SVT", "PSVT", "supraventricular", "arrhythmia", "dysrhythmia", "fib", "fibrillation", "flutter", "ice", "tachycardia", "vagal maneuver" and "Valsalva". All records with any one of these three criteria were included for screening analysis.

For each SVT anesthetic record identified, the perioperative records were examined in detail to obtain demographic information, medical diagnosis and comorbidities, surgical procedure, anesthetic agents, description of event, treatment, consequences of event, and outcomes on any subsequent follow-up appointments. At least two investigators, including one pediatric cardiac anesthesiologist and one pediatric cardiologist, reviewed the anesthetic and inpatient medical records of each included patient to further categorize the events and determine the likelihood of the diagnosis.

A qualitative analysis was performed by examining the clinical features of all reported SVT patients and assigning to each a diagnosis classification of "highly suspicious" or "unlikely". Characteristics of "highly suspicious" SVT events included a clearly documented and sudden increase in HR above baseline levels, and clear evidence of termination of the event with vagal or pharmacologic treatment, or by withdrawal of an inciting physical stimulus such as a central line catheter or guidewire. Further evidence accumulated after the intraoperative phase of the event that could substantiate a diagnosis classification included capturing the SVT on a 12-lead electrocardiogram (ECG) and diagnostic opinions of the consulting pediatric cardiologist, if obtained. Statistical analyses of the results are solely descriptive.

3. Results

Of 285,353 total anesthetics in noncardiac patients over the study period, 36 anesthetic records contained the indication of a possible SVT. Of these, 22 patients (age ± SD = 6.6 ± 5.56) met the criteria for a "highly suspicious" event (Table 1). Five of these highly suspicious episodes were later confirmed by 12-lead ECG. For some of these "highly suspicious" events, the precise cause of the SVT episode could not be discerned. However, 4 of these patients had central line catheter or wire manipulation sometime (three immediately) prior to developing SVT, but no such preceding event was noted in the remaining 18 patients. One

Table 1 Clinical characteristics of patients who developed supraventricular tachycardia (SVT)

Subject	Age (yrs)	Comorbidities *	Diagnosis	Procedure	Anesthetic during event
1	9	28 wks premature, RAD	Leg length discrepancy	Tibia & fibula osteotomies	Desflurane
2	4	None	Appendicitis	Laparoscopic appendectomy	Sevoflurane
3	9	None	Appendicitis	Laparoscopic appendectomy	Desflurane
4	< 1	Prematurity, RAD, anemia	Bowel obstruction	Exploratory laparotomy	Desflurane
5	12	Depression	Ulnar fracture	ORIF	Desflurane
6	8	None	Leukemia	Central line placement	Desflurane
7	< 1	None	Pyloric stenosis	Laparoscopic pyloromyotomy	Sevoflurane
8	15	None	Anterior mediastinal mass	Resection mass	Desflurane
9	12	Prematurity, possible arrhythmia in infancy	Hypertrophic scar	Scar revision	Desflurane
10	2	Prematurity, BPD, GERD, developmental delay	Neuroblastoma	Central catheter placement	Desflurane
11	5	Hearing loss	OSA	T&A	Isoflurane
12	3	OSA	Ureteral reflux	Ureteral reimplant	Desflurane
13	13	None	Femur fracture	Intramedullary nail	Desflurane
14	13	Previous volvulus repair	Small bowel obstruction	Exploratory laparotomy	Desflurane
15	5	None	Tonsil hypertrophy	T&A	Isoflurane
16	< 1	None	Hyperinsulinemia	Broviac placement	Isoflurane
17	3	Chronic nausea, hydronephrosis	Neuroblastoma	Central line placement	Desflurane
18	14	Diabetes	Hepatic failure	Liver transplant	Isoflurane
19	< 1	Tachypnea, anemia	Hyperinsulinemia	Pancreatectomy	Desflurane
20	< 1	None	Pyloric stenosis	Laparoscopic pyloromyotomy	Desflurane
21	16	RAD	Biliary dyskinesia	Laparoscopic cholecystectomy	Desflurane
22	3	None	Tonsil hypertrophy	T&A	Desflurane

Subject	Age (yrs)	Event characteristics	Treatment	Consequences of event	Follow up
1	9	Abrupt on/off HR increase to 194 bpm during procedure	None	None	None
2	4	Two episodes abrupt on/off HR increases to 213 bpm at induction	Carotid massage	Planned admission converted to telemetry unit	Lost to follow-up
3	9	Abrupt HR increase to 220 bpm lasting two min at end of procedure	Adenosine	Planned admission converted to telemetry unit	Lost to follow-up
4	< 1	Abrupt atrial flutter, HR to 240 bpm during procedure, maybe related to catheter placement	Placement of esophageal echocardiogram probe	Telemetry during planned admission to ICU	None
5	12	Increase in HR to 140 bpm lasting 30 min, 20 min after intubation, before start of procedure	Adenosine	Procedure canceled, unplanned telemetry admission	SVT on Holter monitor as outpt, then lost to follow up.
6	8	Abrupt HR increase to 200 bpm, unrelated to line placement	Unspecified vagal maneuvers	None	None
7	< 1	Multiple, abrupt HR increases to 220 bpm, lasting two min before intubation & after extubation	Adenosine, esmolol	Planned admission converted to telemetry unit	Followed by cardiology, given nadolol and flecainide
8	15	Abrupt on/off HR increase from 80 to 160 bpm during procedure	Phenylephrine	None	None
9	12	Abrupt HR increase to 175 bpm lasting one min at induction	Carotid massage	None	None
10	2	Abrupt on/off HR increase to 214 bpm during wire placement	Carotid massage	None	None

(*continued on next page*)

Table 1 (*continued*)

Subject	Age (yrs)	Event characteristics	Treatment	Consequences of event	Follow up
11	5	Two episodes of abrupt HR increases to 210 bpm during procedure; SVT confirmed by 12-lead ECG in PACU	Propranolol	Unplanned admission to telemetry	None
12	3	Multiple abrupt on/off HR 230 bpm greater than 30 sec, after extubation; SVT confirmed by 12-lead ECG in PACU	Adenosine in PACU	Planned admission converted to telemetry unit	Lost to follow-up
13	13	Abrupt onset HR 180 bpm, unresponsive to carotid massage, labetalol, esmolol at end of procedure	Two doses of adenosine	Planned admission converted to telemetry unit	Lost to follow-up
14	13	Abrupt on/off HR increase to 220 bpm during procedure; SVT confirmed by 12-lead ECG; normal echocardiogram	Adenosine	Telemetry during planned admission to ICU	None
15	5	Abrupt on/off HR increase to 210 bpm during procedure	None	Unplanned admission to telemetry	Outpt Holter normal, no treatment
16	< 1	Abrupt on/off HR increase to 207 bpm during line placement	Carotid massage	None	None
17	3	Abrupt on/off HR increase to 220 bpm during procedure; junctional ectopic tachycardia confirmed by 12-lead ECG	Adenosine	Telemetry during planned admission to ICU	Outpt Holter normal, no treatment
18	14	Abrupt on/off HR increase to 190 bpm near end of case, twice	Carotid massage	None	None
19	< 1	HR increase to 220 bpm at end of case, with abrupt decrease	Unspecified vagal maneuvers	None	None
20	< 1	Abrupt on/off HR increase to 220 bpm during induction; SVT confirmed by 12-lead ECG	Unspecified vagal maneuvers	Planned admission converted to telemetry unit	SVT on outpt Holter, digoxin
21	16	Abrupt on/off HR increase to 186 bpm during case, twice	Adenosine	Planned admission converted to telemetry unit	Lost to follow-up
22	3	Abrupt on/off HR increase to 215 bpm at induction and emergence; procedure canceled	Unspecified vagal maneuvers	Unplanned admission to telemetry	SVT on outpt Holter, nadolol

HR=heart rate, bpm=beats per minute, ICU=intensive care unit, outpt=outpatient, ECG=electrocardiograph, PACU=Postanesthesia Care Unit.

* RAD=reactive airway disease, ORIF=open reduction & internal fixation, BPD=bronchopulmonary dysplasia, GERD=gastroesophageal reflux disease, OSA=obstructive sleep apnea, T&A=adenotonsillectomy.

child with SVT after induction had their procedure canceled; however, during the rescheduled procedure two days later, the patient continued to demonstrate intermittent SVT that was hemodynamically stable and did not require additional treatment.

The characteristics of the anesthetics in the highly suspicious SVT patients were similar to the typical case volume (Table 2). The SVT events did not appear to predominantly occur in any particular phase of anesthesia, but it appeared that some of the events were temporally related to some type of physical stimulus, such as tracheal intubation or surgical manipulation.

Treatments of SVT included vagal maneuvers (n=12), and pharmacologic antiarrhythmics (n=11). Some children had more than one attempted treatment. Two patients had termination of the suspected SVT event without any treatment. In the 12 patients who initially were treated with a vagal maneuver, 9 successfully converted the SVT to sinus rhythm. Six of the 12 vagal maneuvers were described as carotid massage; the others were not specifically documented on the anesthetic record. Adenosine was the first-line medication in 8 patients, one of whom required a second dose. One child with suspected SVT that not resolve with administration of adenosine had resolution by insertion of a transesophageal echocardiography probe. Other medications used to convert SVT included esmolol, phenylephrine, and propranolol; one additional child received digoxin postoperatively in the intensive care unit (ICU).

In 8 patients with highly suspicious SVT, no change was noted in their postoperative disposition. However, 14 had escalation of care: 7 patients required an admission change from a regular inpatient unit to a telemetry unit; three patients who were originally planned for same-day discharge were admitted to a telemetry unit; and three patients who were originally

Table 2 Anesthetic characteristics of patients with highly suspicious supraventricular tachycardia (SVT)

Primary surgical service		
	General surgery	13
	Orthopedics	3
	Otorhinolaryngology	3
	Urology	1
	Plastics	1
	Transplant	1
SVT event occurrence *		
	induction	6
	maintenance	13
	emergence	4
Induction agent		
	sevoflurane	10
	thiopental sodium	7
	propofol	4
	fentanyl	1
primary anesthetic during SVT episode		
	desflurane	16
	isoflurane	4
	sevoflurane	2

* One child had SVT during induction and again during emergence.

planned for ICU admission were switched to a telemetry ICU bed. Three patients were discharged taking oral antiarrhythmic medications as a result of continuing postoperative SVT.

At hospital discharge, 11 patients were not recommended to have follow-up by a cardiologist. Of the 11 patients who were recommended to have outpatient follow-up with a cardiologist, 6 were lost to follow-up (one of whom demonstrated SVT on Holter monitoring), two showed no abnormalities on Holter monitoring, and three demonstrated SVT on a Holter monitor and were continued on antiarrhythmic medications. No patient was subsequently found to have any other type of inherent cardiac abnormality.

4. Discussion

On review of all anesthetic records of children anesthetized in the post-halothane era, we found 36 children without known heart disease who were suspected of developing SVT during general anesthesia. Detailed qualitative analysis showed that 22 of these events were "highly suspicious" of a reentrant type of SVT. None of these patients had known preexisting cardiac disease, and none had clinically important sequelae from the SVT during their hospitalization. Three patients were started on pharmacologic antiarrhythmics before hospital discharge, and remained on medication because they continued to demonstrate episodes of SVT on Holter monitoring. There were no unique identifiable patient characteristics or anesthetic agents that were associated with the development of SVT; however, the anesthetic records of some patients indicated that the SVT episodes were temporally related to some type of physical stimulation such as tracheal intubation, guidewire manipulation, or surgical stimulus.

Treatments that successfully terminated the SVT events included vagal maneuvers and antiarrhythmic medications; in some cases there was spontaneous resolution of the event. In the two cases of highly suspicious SVT that resolved without treatment, it was the suddenness and degree to which HR increased and decreased that convinced the investigators that the diagnosis was accurate. Vagal maneuvers consisting primarily of carotid massage appeared to be successful in most cases in which it was attempted as the first treatment method. (This mechanism might be implicated in our patient with SVT, which terminated during insertion of the echocardiography probe.) Increasing vagal tone slows conduction through the AV node [3]. Physical maneuvers that increase vagal tone and terminate SVT include carotid massage, Valsalva maneuver, and application of ice to the face [4]. The comparative efficacy and complications of these different methods have not been studied in anesthetized pediatric patients.

Adenosine was used as the primary pharmacologic treatment in most cases, in accordance with Pediatric Advanced Life Support (PALS) guidelines [5]. Adenosine 0.1 mg/kg given via rapid intravenous bolus is recommended as a first-line pharmacologic treatment of SVT and has terminated SVT in anesthetized children [6]. Additional medications that terminate SVT in the literature and were used in our patients included phenylephrine, esmolol, and propranolol [7]. Amiodarone and digoxin are also successful in the termination of resistant SVT [8].

Digoxin and calcium channel blockers should be used with caution. Digoxin should be avoided if there exists the possibility of Wolff-Parkinson-White (WPW) syndrome. Atrioventricular (AV) node blockade in the setting of an atrial arrhythmia with WPW may allow for rapid conduction down the accessory pathway and result in ventricular fibrillation. Although verapamil can effectively treat pediatric SVT, its use in neonates has been associated with hemodynamic decompensation [9].

A unique limitation of this case series was the difficulty of determining in hindsight whether the patient's tachycardia represented a true reentrant SVT. Supraventricular tachycardia is a broad term used to identify a tachyarrhythmia of supraventricular origin that is often indistinguishable from sinus tachycardia on a surface ECG (Table 3). A diagnosis of sinus tachycardia is based on the morphology of the P waves, age-specific HR values, and the clinical situation during presentation. Sinus tachycardia is characterized by upright P waves in leads I, II, and aVF followed by a QRS complex with a rate-appropriate PR interval (ie, PR interval shortens as HR accelerates). Sinus tachycardia occurs commonly in the operating room during induction of general anesthesia and tracheal intubation, and at times of heightened surgical stimulation or pain. Although age-related limits of normal HRs in children exist, it is difficult to extrapolate these data to the anesthetized child. In the cases reported here, we

Table 3 Supraventricular tachycardias classified by origin

Origin	Classification	Lead II P wave ECG findings	Clinical characteristics
Sinus node			
	Sinus tachycardia	Upright	Heart rate (HR): infants < 220 bpm; children <180 bpm; varies during general anesthesia
Atrium			
	Atrial fibrillation	Variable fibrillation waves	Irregular rhythm, variable rate
	Atrial flutter	Saw tooth flutter waves	Regular QRS, rate depends on degree of block (2:1, 3:1)
	Ectopic atrial tachycardia (EAT)	Rapid single morphology P wave different from sinus, may not be visible	Variation in HR 120–30 bpm, may have slow on/off period
	Multifocal atrial tachycardia (MAT)	Rapid variable morphology P wave different than sinus, may not be visible	Variation in HR 120–300 bpm, may have slow on/off period
AV junction			
	AV reentrant tachycardia (AVRT)	Retrograde P wave following the QRS	Regular HR 150–300 bpm, abrupt on/off period, respond to vagal maneuver/drugs
	AV nodal reentrant tachycardia (AVNRT)	Downward facing P wave following QRS, may not be visible	Regular HR 150–300 bpm, abrupt on/off period, respond to vagal maneuver/drugs
	Junctional ectopic tachycardia	No visible P waves or P waves not associated with QRS	Variation in HR 120–250 bpm, may have slow on/off period

assumed that a presumptive diagnosis of SVT was made by the anesthesia care team based on the suddenness of the tachycardia and the highest rate achieved. On retrospective review, cases felt to be unlikely or of low suspicion for SVT often had documentation of age-appropriate HR values; in most cases, the high HR values seemed to increase over the course of several minutes in the face of an explainable catecholamine surge, and then gradually resolved. In our highly suspicious SVT patients, the diagnosis was later confirmed by ECG in some, but most remained highly suspicious based on the absolute rate attained, the suddenness of appearance, and the disappearance with traditional treatment methods.

Although SVT is commonly used to describe either AV reentrant tachycardia (AVRT) or the AV nodal reentry type of tachycardia (AVNRT), it is often used to label multiple arrhythmias originating from above the ventricles. These also include atrial fibrillation, atrial flutter, ectopic atrial tachycardia (EAT), multifocal atrial tachycardia (MAT), and junctional ectopic tachycardia (Table 3). Atrial fibrillation and atrial flutter may be diagnosed using lead II of the surface ECG. Atrial fibrillation has a characteristically irregular R-to-R distance and noted fibrillation waves. This arrhythmia is rare in healthy children, although it may occur acutely with wire stimulation or in situations of atrial dilatation. Atrial flutter has a distinctive "saw tooth" P-wave pattern on lead II of a surface ECG. None of our reported cases resembled atrial fibrillation or flutter. Ectopic atrial tachycardia and MAT are rarely seen in healthy pediatric patients, and most often after cardiac surgery. Ectopic atrial tachycardia is the result of an increased automaticity of a non-sinus atrial focus, or multiple foci in the case of MAT. Similarly, in junctional ectopic tachycardia the focus is often high in the AV node.

These arrhythmias can be difficult to distinguish from other forms of SVT on a surface ECG in lead II alone. The *P*-wave of EAT is a different configuration from that seen in sinus tachycardia. There may be evidence of varying degrees of AV block, as with atrial flutter. The P-wave of MAT may have a varying morphology. Ectopic atrial tachycardia and MAT often have gradual onset and offset which, along with the rate, can help distinguish them from other forms of SVT. Ectopic atrial tachycardia is classified as MAT if there is more than one focus that can be seen as multiple P-wave morphologies. These arrhythmias are diagnoses of exclusion; failure of vagal maneuvers or of adenosine to terminate the SVT, slower onset, and the clinical scenario assist in diagnosing these arrhythmias.

In otherwise healthy children, AVRT or AVNRT account for most episodes of SVT. In an attempt to determine the true nature of each SVT event, we considered a variety of criteria that increased or decreased our suspicion for one of these types. These criteria included a sudden on-off appearance, HR above age-appropriate normal limits, and termination of the event with a vagal maneuver or administration of a pharmacologic agent.

Additional limitations of this study, which are common to all retrospective reviews, are errors of data reporting and collection. Potential cases of SVT were identified using a query of electronic medical records for terms in narrative description or medications that might have been used to treat SVT. This methodology may have missed some cases of unrecognized SVT with HRs within the limits that are considered normal; or it may have missed suspected SVT cases that did not have any of the matching criteria within the data fields of the electronic record.

While sevoflurane may have fewer proarrhythmic properties than other volatile anesthetics, isolated cases of

sevoflurane-associated arrhythmias have been reported. These reports are suggestive of a tachyarrhythmia of supraventricular origin or the prolongation of the QT interval, as opposed to those of ventricular origin seen with halothane [10–13].

In conclusion, most cases of intraoperative SVT were transient and responded to vagal maneuvers or antiarrhythmic medications. However, some children demonstrated for the first time a propensity for SVT, and were discharged home receiving maintenance antiarrhythmic therapy.

In Memoriam

This article is dedicated to the memory of Chad Cripe, who recently passed away at the age of 45 after a year-long battle with brain cancer. Chad was an exceptionally gifted CRNA, who then went on to medical school later in life. While balancing the responsibilities of school and three young children, Chad graduated from Hahnemann Medical College, the University of Pennsylvania Anesthesiology Residency, and a fellowship in Pediatric Cardiac Anesthesia at The Children's Hospital of Philadelphia, where he remained as a faculty member for several years. Chad was a rising academic and clinical star who always kept his sense of humanity while caring for the sickest patients. We will miss him dearly.

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