

# **CME** Anesthesia-Related Cardiac Arrest in Children: Update from the Pediatric Perioperative Cardiac Arrest Registry

Sanjay M. Bhananker, MD, FRCA\*

Chandra Ramamoorthy, MD†

Jeremy M. Geiduschek, MD\*

Karen L. Posner, PhD\*

Karen B. Domino, MD, MPH\*

Charles M. Haberkern, MD,  
MPH\*

John S. Campos, MA\*

Jeffrey P. Morray, MD‡

**BACKGROUND:** The initial findings from the Pediatric Perioperative Cardiac Arrest (POCA) Registry (1994–1997) revealed that medication-related causes, often cardiovascular depression from halothane, were the most common. Changes in pediatric anesthesia practice may have altered the causes of cardiac arrest in anesthetized children.

**METHODS:** Nearly 80 North American institutions that provide anesthesia for children voluntarily enrolled in the Pediatric Perioperative Cardiac Arrest Registry. A standardized data form for each perioperative cardiac arrest in children  $\leq 18$  yr of age was submitted anonymously. We analyzed causes of anesthesia-related cardiac arrests and related factors in 1998–2004.

**RESULTS:** From 1998 to 2004, 193 arrests (49%) were related to anesthesia. Medication-related arrests accounted for 18% of all arrests, compared with 37% from 1994 to 1997 ( $P < 0.05$ ). Cardiovascular causes of cardiac arrest were the most common (41% of all arrests), with hypovolemia from blood loss and hyperkalemia from transfusion of stored blood the most common identifiable cardiovascular causes. Among respiratory causes of arrest (27%), airway obstruction from laryngospasm was the most common cause. Vascular injury incurred during placement of central venous catheters was the most common equipment-related cause of arrest. The cause of arrest varied by phase of anesthesia care ( $P < 0.01$ ). Cardiovascular and respiratory causes occurred most commonly in the surgical and postsurgical phases, respectively.

**CONCLUSIONS:** A reduction in the proportion of arrests related to cardiovascular depression due to halothane may be related to the declining use of halothane in pediatric anesthetic practice. The incidence of the most common remaining causes of arrest in each category may be reduced through preventive measures.

(Anesth Analg 2007;105:344–50)

**T**he Pediatric Perioperative Cardiac Arrest (POCA) Registry was formed in 1994 to study the causes and outcomes from perioperative cardiac arrests in anesthetized children. The initial findings of the POCA registry were reported by Morray et al. (1). Medication-related cardiac arrests, particularly those due to the cardiovascular depressant effects of halothane, were the most common, and often occurred in American Society of Anesthesiologists (ASA) physical status 1–2 children younger than 1-year-of-age. Over

the last decade, halothane use has declined in favor of sevoflurane. Given that sevoflurane has been reported to cause less bradycardia (2,3) and myocardial depression (4,5) compared to halothane, the profile of cardiac arrest in anesthetized children may have changed as well. We therefore analyzed the cases submitted to the POCA Registry in the 7 yr since the original report to investigate the causes and outcomes from perioperative cardiac arrests in children.

## **METHODS**

This study was approved by the University of Washington IRB. The POCA Registry was formed in 1994. Data were collected from voluntary enrollment of institutions in the United States and Canada that provide anesthetic care to children. This study includes cases submitted by enrolled institutions in 1998–2004. During this time, an average of  $68 \pm 9$  (range, 58–79) institutions was enrolled each year in the POCA registry. Seventy-two percent of these institutions were university-affiliated hospitals, 16% community hospitals, 4% government or military hospitals, and 9% other.

The data collection process has been described in detail (1). Briefly, a designated representative from each

From the \*Department of Anesthesiology, University of Washington School of Medicine, Seattle, Washington; †Department of Anesthesiology, Stanford University School of Medicine, Palo Alto, California; and ‡Department of Anesthesiology, Phoenix Children's Hospital and Valley Anesthesiology Consultants, Phoenix, Arizona.

Accepted for publication April 19, 2007.

Supported in part by the American Society of Anesthesiologists (ASA), Park Ridge, IL, as part of the Closed Claims Project.

All opinions expressed are those of the authors and do not necessarily reflect those of the ASA.

Address correspondence and reprint requests to Sanjay Bhananker, MD, FRCA, Department of Anesthesiology, Harborview Medical Center, 325 Ninth Avenue, Box 359724, Seattle, WA 98104-2499. Address e-mail to kdomino@u.washington.edu.

Copyright © 2007 International Anesthesia Research Society

DOI: 10.1213/01.ane.0000268712.00756.dd

participating institution submitted a standardized data form for all cases of cardiac arrest (defined as the administration of chest compressions or as death) that occurred in children 18 yr or younger during administration of, or immediate recovery from, anesthesia. Neonatal resuscitations and resuscitations in the pediatric intensive care unit or on the ward were excluded.

The standardized data form included patient demographic information, pre-arrest status, surgical procedure, personnel involved in anesthetic care, anesthetics, techniques and monitors used, antecedent events, the immediate cause of arrest, and details of the resuscitative effort. Possible causes for cardiac arrest were previously defined (1). The institutional representatives were asked to assess the contribution of anesthesia, surgery, and underlying patient disease to causation and outcome after cardiac arrest as none, minor, major, or total (1). A narrative summary was also requested to specify the sequence of events and causal relations associated with the cardiac arrest and to provide any relevant information not included in the standardized data collection form.

Outcome was assessed using a modification of a 10-point severity of injury scale (6) applied 24 h after the arrest and at the last clinical evaluation if the patient survived more than 24 h (1). Autopsy findings, if available, were also included.

All cases were submitted anonymously, identified only by a one- to five-digit number assigned by the institutional representative, thus precluding identification of the patient, anesthesia provider(s), or submitting institution. All data forms were reviewed for consistency and completion. If data were missing from the data form, an alert was sent out to all POCA Registry institutional representatives requesting information for the case as identified by the one- to five-digit number.

Two or more members of the POCA Registry Steering Committee (SMB, JMG, CMH, JPM, CR) reviewed all data forms and categorized each cardiac arrest as anesthesia-related, not anesthesia-related, or unknown according to previously published definitions (1). Cases of inability to wean from cardiopulmonary bypass were categorized as not related to anesthesia when there were no anesthesia-related problems in the pre-bypass period. Cases of arrest due to hemorrhage were classified as anesthesia-related if anesthesia personnel could have adequately replaced blood loss or if arrest was due to metabolic consequences of massive transfusion (e.g., hyperkalemia, hypocalcemia). All arrests due to presumed cardiovascular cause, unclear etiology, and all medication-related arrests received a second review by three Steering Committee members (SMB, JPM, CR). Agreement by two of three reviewers was required to change the institutional assignment of etiology of arrest.

All statistical analysis was restricted to cases designated by the Steering Committee as anesthesia-related. Categorical data were analyzed using Fisher's

**Table 1.** Patient Characteristics in Anesthesia-Related Arrests

	1998–2004 <i>n</i> = 93	1994–1997 <sup>a</sup> <i>n</i> = 150
ASA physical status <sup>b</sup>		
1	13 (7)*	23 (15)*
2	34 (18)	27 (18)
3	79 (42)	56 (37)
4	53 (28)	41 (27)
5	11 (6)	3 (2)
Emergency	40 (21)	31 (21)
Age		
<1 mo	21 (11)	22 (15)
1–5 mo	41 (21)	42 (28)
6–11 mo	12 (6)*	19 (13)*
12 mo–5 yr	58 (30)	47 (31)
6–18 yr	60 (31)†	20 (13)†

Percentages in parentheses may not sum to 100% due to rounding.

ASA = American Society of Anesthesiologists.

<sup>a</sup> Previously published data used with permission (Murray, et al. *Anesthesiology* 2000;93:6–14. © Lippincott Williams & Wilkins).

<sup>b</sup> Cases with missing data excluded.

\*  $P < 0.05$  1998–2004 vs 1994–1997 by Z-test.

†  $P < 0.01$  1998–2004 vs 1994–1997 by Z-test.

exact test with the Z test to compare proportions between time periods (1994–1997 and 1998–2004). Factors predicting mortality from arrest were analyzed with multivariate forward stepwise logistical regression with odds ratios and 95% confidence intervals reported. Variables with univariate likelihood ratio  $\chi^2$  values significant at  $P < 0.05$  were included in the multivariate analysis. Significance was defined as values of  $P < 0.05$ .

## RESULTS

In the 7-yr period from 1998 to 2004, the POCA Registry received 397 reports of perioperative cardiac arrest in children. One-hundred-ninety-three of these arrests (49%) were judged as related to anesthesia. Cases unrelated to anesthesia were predominantly ASA physical status 4–5 patients (70%) and ASA E status (44%). In nearly one-quarter of these cases (23%), the arrest episode was due to failure to wean from cardiopulmonary bypass. Seventeen percent were associated with uncontrolled surgical hemorrhage. The present analysis focuses on the 193 anesthesia-related cardiac arrests.

Three-quarters (75%) of the anesthesia-related arrests occurred in patients of ASA physical status 3–5 (Table 1). There were proportionately fewer ASA physical status 1 patients in 1998–2004 arrests (7%) compared to 1994–1997 (15%,  $P < 0.05$ ). There were also proportionately fewer patients under 1-yr-of-age due to a decline in the 6–11 mo group ( $P < 0.05$ , Z-test) and more patients 6–18 yr in 1998–2004 ( $P < 0.01$ , Table 1).

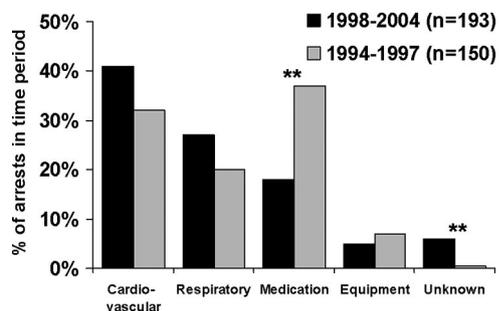
Causes of arrest are shown in Table 2, with a comparison of the two time periods in Figure 1. Compared to 1994–1997, the time period 1998–2004 had fewer arrests related to medication ( $P < 0.01$ , Fig. 1), and more for which a specific cause could not be

**Table 2.** Causes of Cardiac Arrest 1998–2004

Cause	<i>n</i> = 193 No. (% of 193)
Cardiovascular	79 (41)
Hypovolemia associated with blood loss	23 (12)
Electrolyte imbalance	10 (5)
Hypovolemia (nonhemorrhage)	5 (3)
Air embolism	4 (2)
Other CV	11 (6)
Presumed CV unclear mechanism	26 (13)
Respiratory	53 (27)
Airway obstruction—laryngospasm	11 (6)
Airway obstruction—other	5 (3)
Inadequate ventilation or oxygenation	9 (5)
Inadvertent or premature extubation	7 (4)
Difficult intubation	4 (1)
Esophageal or endobronchial intubation	3 (2)
Bronchospasm	4 (2)
Pneumothorax	2 (1)
Aspiration	2 (1)
Other	1 (1)
Presumed respiratory, unclear mechanism	5 (3)
Medication	35 (18)
Halothane-induced CV depression	9 (5)
Sevoflurane-induced CV depression	6 (3)
Other single medication <sup>a</sup>	9 (5)
Medication combination	7 (3)
Allergic reaction	2 (1)
Intravascular injection of local	2 (1)
Equipment	9 (5)
Central catheter	5 (3)
Kinked or plugged ET tube	2 (1)
Peripheral IV catheter	1 (1)
Breathing circuit	1 (1)
Multiple events	3 (2)
Miscellaneous	2 (1)
Unknown	12 (6)

CV = cardiovascular; ET = endotracheal; IV = intravenous.

<sup>a</sup> Noninhalation agents.



**Figure 1.** Cause of arrest: causes of anesthesia-related cardiac arrest in 1998–2004 compared to 1994–1997. Data from 1994 to 1997 previously published and used with permission (Murray, et al. *Anesthesiology* 2000;93:6–14, © Lippincott Williams & Wilkins). Multiple and miscellaneous other causes (3% 1998–2004 vs 4% 1994–1997) not shown. **\*\****P* < 0.01, 1998–2004 vs 1994–1997 by Z test.

identified. In 1998–2004, there were no differences in major cause of cardiac arrest by age.

In 1998–2004, cardiovascular causes of arrest accounted for the highest proportion of anesthesia-related

**Table 3.** Anesthesia-Related Factors in Cardiac Arrests from Hypovolemia Due to Blood Loss 1998–2004 (*n* = 23)

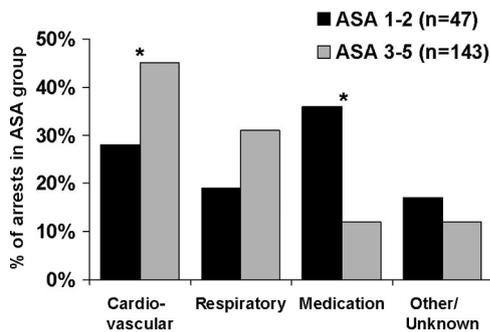
Underestimation of blood loss	11 (48)
Inadequate peripheral venous access	5 (22)
Central venous catheter not present or not transduced	5 (22)
Arterial catheter not present or malfunctioning	4 (17)
Underestimation of pre-existing hypovolemia or anemia	3 (13)
Not enough help available to treat blood loss	3 (13)
Delay in getting blood from blood bank	3 (13)
Hypocalcemia not appreciated, or undertreated	3 (13)
Development of coagulopathy	2 (9)

Percentages in parentheses may sum to >100% due to multiple factors in some cases.

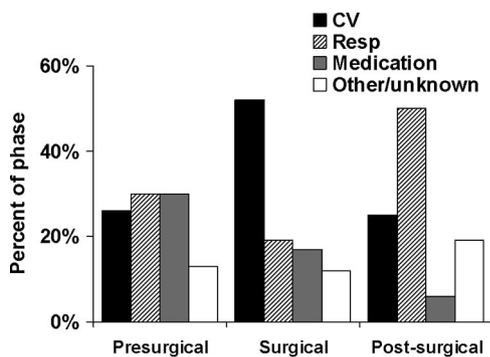
arrests (41%, *n* = 79; Table 2, Fig. 1). Among these, the most common identifiable single cause was hypovolemia related to blood loss (*n* = 23, 12% of anesthesia-related arrests). The majority of these arrests resulted from blood loss occurring during either spinal fusion cases (*n* = 9) or craniotomy/craniectomy (*n* = 7). Anesthesia-related factors that contributed to arrest from hypovolemia due to blood loss are shown in Table 3. The most common anesthesia-related factors were under-estimation of blood loss (*n* = 11, 48%), inadequate peripheral IV access, (*n* = 5, 22%), and central venous catheter not present or not transduced (*n* = 5, 22%). Eight of the 10 arrests associated with electrolyte imbalance involved patients who suffered hyperkalemic arrest secondary to transfusion of stored blood. In 26 cases of cardiovascular arrests, an exact cause could not be determined. The majority (*n* = 21) of these 26 children were ASA physical status 3–5, often as a result of congenital heart disease (*n* = 9).

Respiratory events were responsible for 27% (*n* = 53) of all cardiac arrests (Table 2, Fig. 1). Airway obstruction from laryngospasm (*n* = 11) was the most common respiratory event. Seven of these patients developed laryngospasm in the postsurgical phase (*n* = 6 during emergence, *n* = 1 during transport), while four had laryngospasm during induction of anesthesia. All patients had cyanosis, a decrease in oxygen saturation to <85% and bradycardia before arrest. Four patients were treated with IV succinylcholine and atropine, while two patients who developed laryngospasm during induction were treated with IM succinylcholine. All patients recovered successfully without permanent sequelae.

Medication-related arrests accounted for 18% (*n* = 35) of all arrests (Table 2, Fig. 1), and were more common among ASA physical status 1–2 patients (36%) than ASA physical status 3–5 patients (12%, *P* < 0.05, Fig. 2). Nine of the medication-related arrests were associated with halothane-related cardiovascular depression, while sevoflurane accounted for six arrests. The “other single medication” category includes three patients who arrested from hyperkalemia after succinylcholine administration and two who had transient asystole after neostigmine administration (with



**Figure 2.** Cause of arrest by American Society of Anesthesiologists (ASA) Physical Status (1–2 vs 3–5) in the 1998–2004 period. ASA Physical Status was unknown in three cases. \* $P < 0.05$  between proportions of ASA 1–2 vs ASA 3–5 patients with medication- and cardiovascular-related arrests by Z test.



**Figure 3.** Cause of arrest by phase of care. Presurgical period = preinduction and induction; surgical phase = maintenance of anesthesia; postsurgical phase = emergence, transport, or recovery. Three cases of “other” phase are excluded.  $P < 0.001$  Fisher’s exact test (Monte Carlo resampling method).

appropriate doses of glycopyrrolate administered simultaneously).

Five percent of cardiac arrests ( $n = 9$ ) were related to equipment (Table 2, Fig. 1). Half of these ( $n = 5$ ) were secondary to central venous catheter complications, including injuries related to needle, guidewire, or catheter insertion (i.e., pneumothorax, hemothorax, and hemopericardium). Other central catheter-related complications included bradycardia upon central catheter advancement and hypotension secondary to cessation of inotrope delivery resulting from inadvertent central catheter removal. Other known or unknown causes or no identifiable cause accounted for the remaining 9% ( $n = 17$ ) of cardiac arrests.

Most cardiac arrests occurred during anesthesia maintenance (58%). Nearly one-quarter (24%) occurred in the preinduction or induction phase and 19% occurred during emergence, transport or recovery. The cause of arrest varied by phase of anesthesia care ( $P < 0.01$ , Fig. 3). Half (52%) of arrests during anesthesia maintenance were cardiovascular in origin, and half (50%) of the arrests in the postoperative phase (emergence, transport, recovery) were attributable to respiratory causes.

There was a significant association between surgical procedure and cause of cardiac arrest ( $P < 0.001$ , Table 4). As expected, respiratory causes were the most common cause of arrest in patients undergoing airway surgery (49%), while cardiovascular causes were the most common cause of arrest in cardiac, neurosurgery, and spine surgery. Nearly three-quarters (71%) of the arrests occurring in patients undergoing neurosurgery or spine surgery were cardiovascular in origin.

Most patients had no injury resulting from the arrest episode ( $n = 118$ , 61%). Upon final assessment, there were 10 patients (5%) with temporary sequelae and 10 (5%) with permanent nonfatal sequelae. Mortality after anesthesia-related cardiac arrest was 28% ( $n = 55$ ). Patients who died were more likely to be sick (91% ASA physical status 3–5) and undergoing emergency procedures (36% ASA E) compared with patients who survived ( $P < 0.01$  for both factors,  $\chi^2$ ). Age was not predictive of mortality. Multivariate analysis suggests an increased odds of death for ASA physical status 3–5 (odds ratio, 3.6;  $P = 0.013$ ) and emergency surgery (odds ratio, 2.8;  $P = 0.007$ ) compared to other pediatric patients experiencing anesthesia-related cardiac arrest (Table 5).

## DISCUSSION

This report updates the changing profile of cardiac arrest in anesthetized children as reported to the POCA Registry in the years 1998–2004. Compared to an earlier period (1994–1997), cardiac arrests resulting from medication-related causes (primarily cardiovascular depression from halothane) declined, and arrests from cardiovascular causes were the most common. Of the known cardiovascular causes of cardiac arrest, hypovolemia from blood loss and the metabolic complications of transfusion (usually hyperkalemia) were most common.

The decreased number of arrests associated with the inhaled anesthetics probably relates to the declining use of halothane in pediatric anesthesia practice in favor of newer anesthetics, particularly sevoflurane.<sup>1</sup> Sevoflurane has been reported to have less potential for producing bradycardia (2,3) and myocardial depression (4,5) in infants and children than does halothane. Halothane also causes more hypotensive episodes than sevoflurane in children with congenital heart disease (7). However, it is important to recognize that sevoflurane is also a cardiac depressant; arrests related to these effects have been identified in this report and our earlier report (1), as well as by others (8). The decline in 1998–2004 in the proportion of ASA physical status 1–2 children and infants less than 1-year-of-age may also be ascribed to the declining use of halothane, since halothane-induced cardiovascular depression tended to occur in previously healthy children under 1-year-of-age.

The primacy of cardiovascular causes of cardiac arrest in 1998–2004 is consistent with a review of

**Table 4.** Causes of Arrest for Various Types of Surgeries (1998–2004, *n* = 193)

Cause of arrest	Airway/ENT surgery ( <i>n</i> = 39)	Cardiovascular procedure ( <i>n</i> = 38)	Neuro- or spine surgery ( <i>n</i> = 35)	All other surgery ( <i>n</i> = 81)
Respiratory	19 (49)	6 (16)	0 (0)	28 (35)
Cardiovascular	8 (21)	17 (45)	25 (71)	29 (36)
Medication	9 (23)	7 (18)	4 (11)	15 (19)
Other, none or unknown	3 (8)	8 (21)	6 (17)	9 (11)

Values inside parentheses indicate percentages.

ENT = ears, nose, throat.

*P* < 0.001 Fisher's exact test (Monte Carlo resampling method).

**Table 5.** Factors Associated with Mortality

Factor	Univariate		Multivariate	
	OR (95% CI)	<i>P</i>	OR (95% CI)	<i>P</i>
ASA physical status				
3–5 ( <i>n</i> = 143)	4.379 (1.628–11.778)	0.003	3.592 (1.313–9.830)	0.013
1–2 ( <i>n</i> = 47)	Reference		Reference	
Emergency surgery				
Emergency ( <i>n</i> = 40)	3.314 (1.604–6.870)	0.001	2.820 (1.330–5.903)	0.007
Nonemergency ( <i>n</i> = 151)	Reference		Reference	

Multivariate model Nagelkerke *R*<sup>2</sup> = 0.133.

OR = odds ratio; CI = confidence interval; ASA = American Society of Anesthesiologists.

pediatric anesthesia malpractice claims from the 1990s, which demonstrated a high proportion of claims related to cardiovascular events (9). In the current study, the most common cardiovascular cause of arrest was hypovolemia, usually due to blood loss, often during either spinal fusion or craniotomy/craniectomy. Hypovolemia and hyperkalemia associated with massive blood transfusion were the most common causes of arrest in noncardiac procedures in a recent report from the Mayo Clinic, although these were not all assessed as anesthesia related (10). In the current report, cases of cardiac arrest due to blood loss were deemed anesthesia-related when the anesthesiologist could possibly have prevented the arrest (Table 3). The pediatric anesthesiologist involved in these high-risk cases should carefully consider the use of invasive monitors and large bore peripheral IV catheters. Central catheters provide useful information, and can be inserted more safely with techniques such as ultrasound guidance. Close attention must be paid to anticipating, estimating, and replacing blood loss. Serial hematocrits, blood gases, and coagulation studies should be used to guide fluid and transfusion therapy. Other strategies to reduce blood loss and the need for transfusion include autologous transfusions, intraoperative blood salvage, antifibrinolytics, and control of arterial blood pressure (11,12).

Transfusion-related hyperkalemia results from the administration of old blood, as potassium concentration in stored blood increases linearly with time. Irradiation of blood is indicated when family members donate blood, or when the recipient is immunocompromised and transfusion-related graft versus host disease is a risk. Irradiation dramatically accelerates the leakage of

potassium from red cells into serum. The risk of developing serious hyperkalemia with rapid transfusion of stored (13,14) and/or irradiated blood (15,16) in pediatric patients is real, especially in neonates or infants in whom one or more blood volumes are transfused. Rapid transfusion using hand-held syringes via small caliber catheters (23 gauge or smaller) of packed red blood cells stored <2 wk may also result in hyperkalemia (17). Use of fresh red blood cells (rather than whole blood), and saline washing of irradiated red cells may help in reducing the incidence of transfusion-associated hyperkalemia (18). It is prudent to monitor serum potassium levels and treat hyperkalemia before serious dysrhythmias occur.

Respiratory events accounted for 27% (*n* = 53) of all arrests. While a variety of events contributed to this category (Table 2), arrest due to laryngospasm was most common (*n* = 11). In other studies, laryngospasm had an incidence of 0.1% (19,20) to 1.2% (21). Pediatric Closed Claims analysis from the 1990s also showed that airway obstruction including laryngospasm was the most common respiratory damaging event (9). In the current study, none of the arrests from laryngospasm resulted in death, though three patients developed negative pressure pulmonary edema.

Cardiac arrest due to laryngospasm may be preventable. In two of the cases of laryngospasm that occurred during induction prior to establishment of IV access, cardiac arrest from hypoxia occurred before the onset of muscle relaxation from IM succinylcholine. This may have been prevented had IV administration of muscle relaxant been possible, or if tracheal intubation had been performed earlier. Early IM (or submental

with digital massage) administration of succinylcholine, before the onset of bradycardia, may help in relieving the spasm before its progression to cardiac arrest. In six cases, laryngospasm occurred during emergence; in four of these cases, extubation occurred with the patient partially but not fully awake, suggesting that a delay in timing of extubation might have been preventive.

Equipment-related arrests accounted for 5% ( $n = 9$ ) of all arrests. Half of these were due to injury to lung parenchyma or to a vascular structure during central venous catheter insertion. The Closed Claims Project has reported that nearly half of the injuries due to central venous catheter insertion were potentially preventable with the use of ultrasound guidance or pressure waveform monitoring (22). Verghese et al. (23) have shown external ultrasound guidance of central venous catheter placement is superior to landmark guidance for improving speed of insertion and reducing the incidence of complications.

The association between cause of arrest and phase of the anesthetic likely relates to the most common etiologies of arrest in each category. Cardiovascular arrests often resulted from blood loss or blood replacement that occurred during the maintenance or surgical phase of an anesthetic. Respiratory-related arrests often occurred in patients with an unprotected airway during emergence or recovery (Fig. 3).

In this report, mortality after anesthesia-related cardiac arrest was 28%, similar to the 26% in our initial report from the POCA Registry (1). The only factors predictive of mortality after cardiac arrest were ASA physical status and emergency surgery. Both have been reported as risk factors for anesthesia-related cardiac arrest in children (24,25) and both were reported as being predictive of death after arrest in the first report from the POCA Registry (1). That ASA physical status is predictive of death after cardiac arrest in anesthetized children is another validation of this scoring system as an assessment tool for anesthetic risk. Emergency surgery as a predictor of mortality after anesthesia-related arrest may be related to patient factors (e.g., complexity or acuity of the patient's illness) or to environmental factors (e.g., reduced support staff or ancillary services during evenings or nights).

The POCA Registry has several methodologic weaknesses, as previously described (1). First, enrollment into the registry and reporting of cases is purely voluntary, and under-reporting is likely. Selection bias is also likely, and highly sensitive cases might not get reported. Self-reporting may also lead to inaccuracies in the data and under-estimates of the anesthetic-related elements of an arrest.

Second, the institutions participating in the POCA Registry are not representative of all North American pediatric surgical locations. Participation in the POCA Registry is skewed toward university-affiliated children's hospitals, whereas a large, but unquantified,

percentage of pediatric anesthesia services in North America are delivered in smaller community hospitals. Nonetheless, the lessons learned from our findings could be applied to all institutions providing anesthesia for children.

Third, the inclusion criteria for the POCA Registry are initiation of chest compressions or death. The Registry does not include cases in which a cardiac arrest was successfully treated with defibrillation but not chest compressions. Similarly, patients who developed hypoxia leading to severe bradycardia and who received chest compressions were included in our analysis, whereas those in whom the bradycardia resolved with restoration of adequate oxygenation were not.

Fourth, the data were gathered retrospectively, often by a person not directly involved in the anesthetic care; lack of concurrent first-hand information could lead to inaccuracies.

Finally, we cannot provide incidence figures for perioperative pediatric cardiac arrests. Although we received limited demographic data for the total number of pediatric anesthetics provided for various age groups and ASA physical status, these data are not complete from the time period of this paper (i.e., some institutions have submitted case reports but not the demographic data and *vice versa*).

Summary: In the 7-year period from 1998 to 2004, the proportion of cases of inhalation agent-related cardiovascular depression resulting in cardiac arrest was less than in the 1994–1997 time period. In contrast, cardiovascular causes of arrest, particularly hypovolemia due to blood loss and transfusion-related hyperkalemia, were proportionately more common. Laryngospasm was the most common cause of respiratory-related arrests, and lung and vascular injury from insertion of central venous catheters was the most common source of equipment-related arrests. For each of these causes of cardiac arrest in anesthetized children, preventive measures may be effective in reducing the incidence.

#### ACKNOWLEDGMENTS

*The authors thank the institutional representatives who participated in the POCA Registry and submitted cases for review. Their identities remain anonymous for purposes of confidentiality. The authors gratefully acknowledge the support and encouragement of Frederick W. Cheney, MD. The authors also thank Lynn Akerlund for her expert assistance in manuscript preparation.*

#### REFERENCES

1. Morray JP, Geiduschek JM, Ramamoorthy C, Haberkern CM, Hackel A, Caplan RA, Domino KB, Posner K, Cheney FW. Anesthesia-related cardiac arrest in children: initial findings of the Pediatric Perioperative Cardiac Arrest (POCA) Registry. *Anesthesiology* 2000;93:6–14
2. Sarner JB, Levine M, Davis PJ, Lerman J, Cook DR, Motoyama EK. Clinical characteristics of sevoflurane in children. A comparison with halothane. *Anesthesiology* 1995;82:38–46

3. Johannesson GP, Floren M, Lindahl SG. Sevoflurane for ENT surgery in children. A comparison with halothane. *Acta Anaesthesiol Scand* 1995;39:546–50
4. Holtzman RS, van der Velde ME, Kaus SJ, Body SC, Colan SD, Sullivan LJ, Soriano SG. Sevoflurane depresses myocardial contractility less than halothane during induction of anesthesia in children. *Anesthesiology* 1996;85:1260–7
5. Wodey E, Pladys P, Copin C, Lucas MM, Chaumont A, Carre P, Lelong B, Azzis O, Écoffey C. Comparative hemodynamic depression of sevoflurane versus halothane in infants: an echocardiographic study. *Anesthesiology* 1997;87:795–800
6. Brunner EA. Analysis of anesthetic mishaps. The National Association of Insurance Commissioners' closed claim study. *Int Anesthesiol Clin* 1984;22:17–30
7. Russell IA, Miller Hance WC, Gregory G, Balea MC, Cassorla L, DeSilva A, Hickey RF, Reynolds LM, Rouine-Rapp K, Hanley FL, Reddy VM, Cahalan MK. The safety and efficacy of sevoflurane anesthesia in infants and children with congenital heart disease. *Anesth Analg* 2001;92:1152–8
8. Gobbo Braz L, Braz JRC, Modolo NSP, do Nascimento P Jr, Brushi BAM, Raquel de Carvalho LR. Perioperative cardiac arrest and its mortality in children. A 9-year survey in a Brazilian tertiary teaching hospital. *Paediatr Anaesth* 2006;16:860–6
9. Jimenez N, Posner KL, Cheney FW, Caplan RA, Lee LA, Domino KB. An update on pediatric anesthesia liability: a closed claims analysis. *Anesth Analg* 2007;104:147–53
10. Flick RP, Sprung J, Harrison TE, Gleich SJ, Schroeder DR, Hanson AC, Buenvenida SL, Warner DO. Perioperative cardiac arrests in children between 1988 and 2005 at a tertiary referral center: a study of 92,881 patients. *Anesthesiology* 2007;106:226–37
11. Tobias JD. Strategies for minimizing blood loss in orthopedic surgery. *Semin Hematol* 2004;41:145–56
12. Weldon BC. Blood conservation in pediatric anesthesia. *Anesthesiol Clin North America* 2005;23:347–61
13. Brown KA, Bissonnette B, MacDonald M, Poon AO. Hyperkalaemia during massive blood transfusion in paediatric craniofacial surgery. *Can J Anaesth* 1990;37:401–8
14. Brown KA, Bissonnette B, McIntyre B. Hyperkalaemia during rapid blood transfusion and hypovolaemic cardiac arrest in children. *Can J Anaesth* 1990;37:747–54
15. Hall TL, Barnes A, Miller JR, Bethencourt DM, Nestor L. Neonatal mortality following transfusion of red cells with high plasma potassium levels. *Transfusion* 1993;33:606–9
16. Fukuoka Y, Ishiyama T, Oguchi T, Nonaka A, Kumazawa T. [Hyperkalemia after irradiated blood transfusion]. *Masui* 1999;48:192–4
17. Miller MA, Schlueter AJ. Transfusions via hand-held syringes and small-gauge needles as risk factors for hyperkalemia. *Transfusion* 2004;44:373–81
18. Barcelona SL, Thompson AA, Cote CJ. Intraoperative pediatric blood transfusion therapy: a review of common issues, Part I: hematologic and physiologic differences from adults; metabolic and infectious risks. *Paediatr Anaesth* 2005;15:716–26
19. Rolf N, Cote CJ. Frequency and severity of desaturation events during general anesthesia in children with and without upper respiratory infections. *J Clin Anesth* 1992;4:200–3
20. McConachie IW, Day A, Morris P. Recovery from anaesthesia in children. *Anaesthesia* 1989;44:986–90
21. Olsson GL, Hallen B. Laryngospasm during anaesthesia. A computer-aided incidence study in 136,929 patients. *Acta Anaesthesiol Scand* 1984;28:567–75
22. Domino KB, Bowdle TA, Posner KL, Spittell PH, Lee LA, Cheney FW. Injuries and liability related to central vascular catheters: a closed claims analysis. *Anesthesiology* 2004;100:1411–18
23. Verghese ST, McGill WA, Patel RI, Sell JE, Midgley FM, Ruttimann UE. Ultrasound-guided internal jugular venous cannulation in infants: a prospective comparison with the traditional palpation method. *Anesthesiology* 1999;91:71–7
24. Tiret L, Nivoche Y, Hatton F, Desmonts J, Vouc'h G. Complications related to anaesthesia in infants and children: a prospective survey of 40,240 anaesthetics. *Br J Anaesth* 1988;61:263–9
25. Keenan RL, Boyan CP. Cardiac arrest due to anesthesia: a study of incidence and causes. *JAMA* 1985;253:2373–7