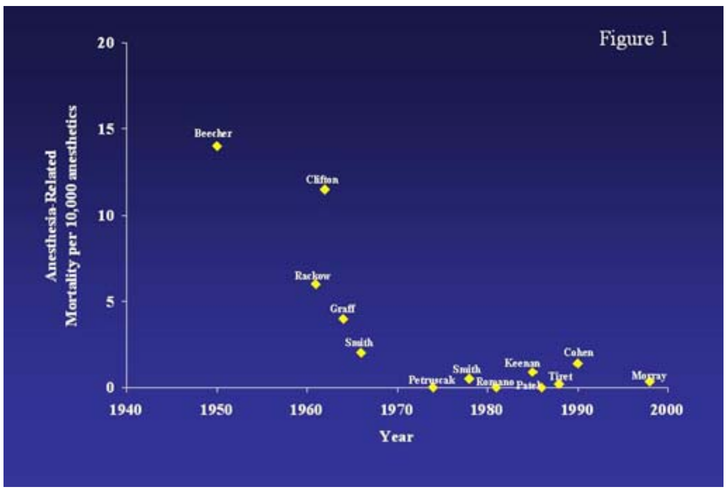
Unexpected Cardiac Arrest In The Anesthetized Child

Jeffrey Morray, MD

Historical Background

Outcomes for anesthetized children have improved over the past 50 years, as reflected in anesthesia-related mortality rates from studies published since the seminal study of Beecher and Todd in 1954.[1] (Figure 1) Large series in the past decade from France, Canada and the United States have reported anesthesia-related mortality rates of 0.2,[2] 0.36,[3] and 1.4[4] per 10,000 anesthetics, respectively. Several series of outpatient anesthetics in healthy children have shown mortality rates of zero.[5,6]



Despite advances in pediatric anesthesia, unexpected cardiac arrests still occur. The risk of anesthesia related cardiac arrest appears to be inversely proportional to age, with our youngest patients at the highest risk.[2,3] Of all cases of cardiac arrest submitted to the POCA Registry, 55% were less than one year of age.[4] Any relation between age and risk results in large measure from the impact of underlying patient disease. The relationship between ASA physical status and the rate of complication in the French series is shown in Table 1.[2] Similarly, in Keenan and Boyan's study, cardiac arrest occurred three times more often and death twice as often in the ASA physical status 3 or 4 patients than in the ASA physical status 1 or 2 patients.[7] In the POCA study, death following anesthesia-related cardiac arrest was predicted most strongly by ASA physical status, though emergency surgery was also predictive (Table 2).[4] When ASA physical status was accounted for, age alone was not predictive of death.

Table 1: Relation between ASA physical status and the rate of complication[2.]

ASA Physical status	Rate of complication per 10,000 anesthetics*
1	4
2	34
3	116
4-5	164

* p< 0.001

Table 2: Multivariate analysis of predictors of mortality[4]

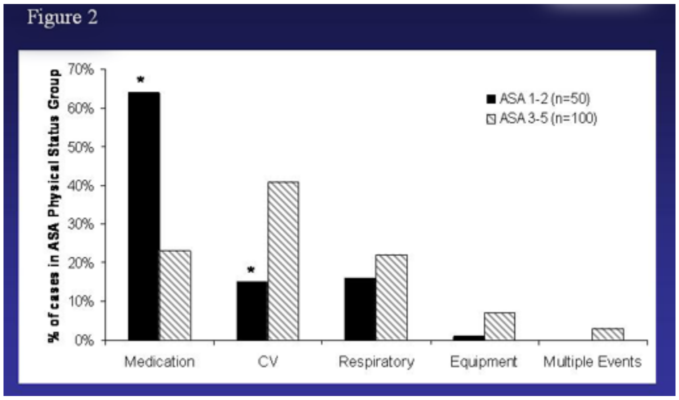
Factor	Odds Ratio	95% Confidence Ratio	Estimated Coefficient	P
ASA physical status 3-5	12.99	2.9 - 57.7	2.56	0.007
Emergency Surgery	3.88	1.6 - 9.6	1.35	0.0036

Causes Of Anesthesia-Related Cardiac Arrest

Along with a decline in the incidence of anesthesia-related cardiac arrest and death has come a change in the profile of causes of arrest. Forty years ago, airway obstruction and aspiration were more frequent, often from the lack of use or inappropriate use of endotracheal tubes. With increased use of muscle relaxants, inadequate ventilation became a relatively more common respiratory complication than airway obstruction or aspiration. In the last decade, respiratory complications have become relatively less common and cardiovascular complications more common, perhaps because of the universal use of pulse oximetry and capnometry.[8]

In the initial 150 cases of anesthesia-related cardiac arrest submitted to the POCA Registry, medication-related events were most common, accounting for 37% of all arrests.[4] Cardiovascular depression from halothane, alone or in combination with other drugs, was responsible for two thirds of all medication-related arrests. Intravascular injection of local anesthetic during administration of a caudal block resulted in cardiac arrest in five patients.

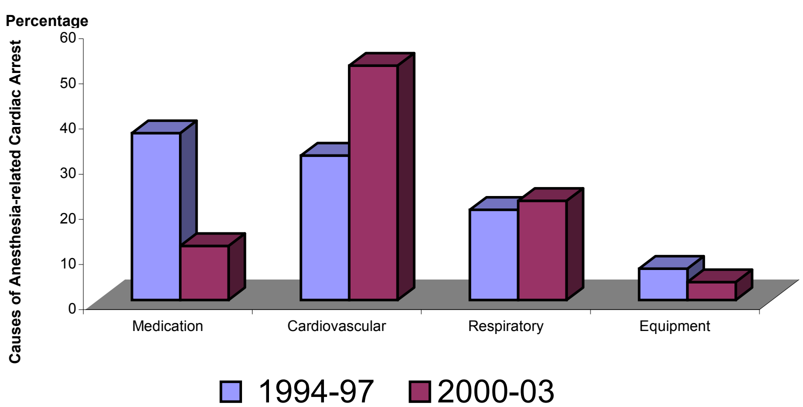
50 arrests occurred in ASA physical status 1 or 2 patients; medications (most commonly halothane alone or in combination with intravenous agents) were deemed responsible in 64% of these arrests (Figure 2).



32% of all arrests were classified as cardiovascular; in more than a third of these, the exact cause of arrest could not be determined. Many of these patients had severe congenital heart lesions and were ASA physical status 3-5. Hemorrhage or hyperkalemia resulting from massive transfusion were the most frequent known causes of arrest in the cardiovascular category.

Since the publication of the initial series of anesthesia-related cardiac arrest, over 200 additional cases have been submitted to the POCA Registry; approximately half of these arrests were related to anesthetic causes. The cause profile of cardiac arrest has changed considerably from 2000 to 2003 (Figure 3). Medication-related causes have declined from 37% to 12% of the total, due primarily to the near disappearance of cases of cardiovascular depression from the inhaled agents. The concomitant increase in the proportion of cardiovascular causes of arrest from 32% to 52% is seen in Figure 3. Hypovolemia (often from hemorrhage) or the metabolic consequences of massive transfusion (usually hyperkalemia) were the most frequent causes of arrest in this category. As in the earlier series, arrests were reported in children with congenital heart lesions; though these arrests were felt to be anesthesia-related, the exact cause of arrest often could not be determined.

Figure 3: Causes of Anesthesia - related cardiac arrest in children in two time periods



The demographic profile of the cases submitted since 1999 has also changed (Table 3). These changes may relate to the decline in the number of arrests being reported to the POCA Registry that are caused by the inhalation agents; these arrests often occurred in ASA physical status 1 or 2 patients who were less than one year of age and were presenting for elective surgery. It is interesting that despite the above-mentioned changes, the mortality rate during the two time periods (26% and 27%) has not changed.

Table 3: Comparison of demographic data from
POCA Registry cases 1994-1997 Versus 2000-2003

	1994-1997	2000-2003
ASA physical Status		
1	15%	4%
2	18%	15%
3	37%	46%
4	27%	22%
5	2%	13%
Age		
<1 Month	15%	13%
1-5 Months	28%	25%
6-11 Months	13%	10%
12 mo-5 yrs	31%	25%
6-18 yrs	13%	27%
Emergency Surgery	21%	30%
Mortality	26%	27%

Optimizing Outcomes: Prevention Of Cardiac Arrest

The treatment of cardiac arrest, once it has occurred, has been reviewed elsewhere.[9] The focus of this discussion is on optimizing outcomes through the prevention of cardiac arrest.

The improvement in cardiac arrest and mortality rates for anesthetized children over the past 50 years reflects many improvements that have been made in pediatric perioperative care. Most recently, the frequency of medication-related cardiac arrests has declined dramatically, presumably as a result of the decreased use of halothane in favor of the newer agents, particularly sevoflurane. The latter agent possesses a number of attractive characteristics. The MAC for sevoflurane remains constant in the first six months of life,[10] while the MAC for halothane is less in the newborn than it is in the six month old;[11] Therefore, anesthetic overdose of the newborn with sevoflurane is less likely. Heart rate, one of the main determinants of cardiac output, is maintained under sevoflurane anesthesia, and decreased with halothane.[10] Sevoflurane, when compared to halothane, is less depressant of myocardial contractility in infants[12] and children.[13] Based on these differences, a switch from halothane to sevoflurane by pediatric anesthesiologists would predictably result in a decline in medication-related cardiac arrests.

The toxicity of bupivicaine when inadvertently injected into the intravascular space is well recognized. Alternative agents with less potential for toxicity include ropivicaine and the L isomer of bupivicaine. Recent reports of cardiac arrest associated with ropivicaine[14, 15] emphasize that compulsive attention to detail is required when local anesthetics of any kind are used. The risk of cardiac arrest from inadvertent intravascular injection is reduced (but not eliminated) when aspiration for blood and a test dose are negative, and when incremental doses rather than full doses are injected.

Cardiac arrests from hypovolemia (usually secondary to hemorrhage) and from the consequences of massive transfusion (usually hyperkalemia) are considered anesthesia-related when the anesthesiologist could possibly have prevented the arrest in some manner. Failure by the anesthesiologist to secure adequate intravenous access preoperatively, and failure to keep up with intraoperative blood loss are the most common reasons why such arrests are deemed, at least in part, anesthesia-related. At least some of these arrests are preventable with adequate anticipation and attention to detail.

Hyperkalemia from massive transfusion is also potentially preventable, through awareness of the problem and a few simple steps to reduce the amount of potassium administered in transfused blood. As blood ages, potassium leaks from the intracellular space into the plasma. (Table 4) This leakage is dramatically accelerated in irradiated blood. The anticoagulant used influences how blood ages. Packed cells, because of the reduced amount of plasma, have a lower potassium load than whole blood. The amount of potassium administered, and thus the risk of a hyperkalemic cardiac arrest, is reduced by the following recommendations:

1. Use the freshest PRBCs available. Avoid using whole blood.
2. Don't irradiate the blood except when absolutely necessary (e.g. a premature baby or immunocompromized child). When irradiation is required, the time between irradiation and blood administration should be minimized.
3. In high risk situations (e.g. newborn or infant requiring >1 blood volume, or with irradiated blood), measure the potassium in the blood to be transfused. If the potassium level is high, consider washing the cells in the cell saver, and resuspending the cells in plasma prior to administration.

Table 4: Potassium leakage from stored blood

	CPD Whole blood		CPD PRBC'S	CPDA-1 PRBC'S	Irradiated CPD PRBC'S
Days Stored	0	21	21	35	1
PH	7.2	6.8	6.8	6.71	6.8
Potassium concentration (meq/L)	4	20	20	78	20
Potassium load (meq/unit)	2	10	5	20	5

Creating The Appropriate Environment For The Care Of Children

Beginning with Beecher and Todd[1], many studies have identified higher anesthetic risk for children than for adults. Such risk can be reduced be creating a specialized environment for children. The recognition of particular high-risk situations aids us as pediatric anesthesiologists in creating practice guidelines and making modifications to the perioperative environment.

Practice Guidelines: Guidelines assist the anesthesiologist as well as our surgical colleagues and other practitioners in establishing safe and consistent approaches to high- risk situations. Prevention of life threatening episodes of apnea in expremature infants is an excellent example. The creation of institutional guidelines for the approach to sedation by anesthesiologists and non-anesthesiologists is another.

The Perioperative Environment: Recently, the American Academy of Pediatrics endorsed the concept of a specialized environment for the provision of anesthetic care to children.[16] The AAP guidelines include a requirement for specialized training, experience and credentialing of personnel. Facility issues include pediatric equipment, drugs, and the necessary support services (e.g. nursing, pharmacy, laboratory, radiology, intensive care, and pain management). The responsibility for creating institutional guidelines rests with the hospital.

Defining the Scope of Practice: In other subspecialties, such as congenital heart surgery, mortality rates are reduced in high-volume centers. The same is likely to be true for anesthesia. Anesthetic outcomes for children, measured by occurrence of cardiac arrest[17] or bradycardia[18] are improved when anesthesiologists trained or experienced in the care of children are involved. Clearly, it may be inappropriate and unnecessary to transfer an otherwise healthy child to the referral center for minor surgery. However, patients defined as high risk based on age or severe underlying disease is best cared for in a referral center, or locally by a pediatric anesthesiologist. Some form of credentialing based on training or annual pediatric caseload could be used to define “pediatric anesthesiologist”.

Programs of Quality Improvement: Quality improvement programs, by themselves, do not necessarily prevent patient injury. In the Pediatric Closed Claims study, however, care was judged substandard in 54% of all claims.[19] therefore; there is the potential for quality improvement programs to reduce patient injury. Bad outcomes may result from random errors, which are difficult to prevent, or from system errors, which should be controllable. Morbidity and Mortality Conferences are a useful part of this process, if only to provide an opportunity for interaction amongst the individuals in a group practice. In addition, Continuous Quality Improvement (CQI) is the process of continuously evaluating anesthesia practice to identify systematic problems (“opportunities”) and implementing strategies for prevention. The purposes are not to blame or identify “bad apples”. A successful CQI program has the following characteristics: 1)It focuses on system improvement rather than individual outliers. 2)If it relies on self-reporting, it is simple for the practitioner to complete in order to maximize compliance. 3)It is context-sensitive, such that patient disease and surgical risks are taken into account in decisions about quality of care. 4) It directly reflects quality of care by including measures of effectiveness and safety of care as well as patient and family satisfaction. 5)It is continuous and self-generating.[20]

References

1. Beecher H and Todd D: A study of the deaths associated with anesthesia and surgery. Ann Surg 140:2-35, 1954.
2. Tiret L, Nivoche Y, Hatton F et al: Complications related to anesthesia in infants and children. Br J Anaesth 61:263-269, 1988.
3. Cohen M, Cameron C, Duncan P: Pediatric anesthetic morbidity and mortality in the perioperative period. Anesth Analg 70:160-167, 1990.
4. Morray J, Geiduschek J, Ramamoorthy C et al: Anesthesia-related cardiac arrest in children: Initial findings of the POCA Registry. Anesthesiology 93:6-14, 2000.
5. Petruscak J, Smith R, Breslin P: Mortality related to ophthalmologic surgery. Surv Anesthesiol 18:87-95, 1974.
6. Romano P: General anesthesia morbidity and mortality in eye surgery at children's hospitals. J Pediatr Ophthalmol Strabismus 18:17-21, 1981.
7. Keenan R, Boyan C: Cardiac arrest due to anesthesia: A study of incidence and causes. JAMA 253:2373-2377, 1985.
8. Cheney F: ASA Closed Claims Project Report: The effect of pulse oximetry and end-tidal CO2 monitoring on adverse respiratory events. ASA Newsletter 56:6-10, 1992.
9. Morray J: Anesthesia-related cardiac arrest in children: An update. In New Concepts and Techniques in Pediatric Anesthesia. Mason L and Kim M Editors. Anesthesiology Clinics of North America 20(1):1-28, 2002.
10. Lerman J, Sikich N, Kleinman S et al: The pharmacology of sevoflurane in infants and children. Anesthesiology 80: 814-824, 1994.
11. Lerman J, Robinson S, Willis M et al: Anesthetic requirement for halothane in young children 0-1 months and 1-6 months of age. Anesthesiology 59:421-424, 1983.
12. Wodey E, Pladys P Copin C et al: Comparative hemodynamic depression of sevoflurane versus halothane in infants. Anesthesiology 87:795-800, 1997.
13. Holzman R, van der Velde M, Kaus S et al: Sevoflurane depresses myocardial contractility less than halothane during induction of anesthesia in children. Anesthesiology 85:1260-1270, 1996.
14. Chazalon P, Tourtier J, Villevielle T et al: Ropivicaine-induced cardiac arrest after peripheral nerve block: Successful resuscitation. Anesthesiology 99:1449-51, 2003.
15. Huet O, Eyrolle L, Mazoit J et al: Cardiac arrest and plasma concentration after injection of ropivicaine for posterior lumbar plexus blockade. Anesthesiology 99:1451-3, 2003.
16. American Academy of Pediatrics: Guidelines for the pediatric perioperative environment. Pediatrics 103:512-515, 1999.
17. Keenan R, Shapiro J, Dawson K: Frequency of anesthetic cardiac arrest in infants: Effect of pediatric anesthesiologists. J Clin Anesth 3:433-437, 1991.
18. Keenan R, Shapiro J, Kane F et al: Bradycardia during anesthesia in infants: An epidemiologic study. Anesthesiology 80:976-982, 1994.
19. Morray J, Geiduschek J, Caplan R et al: A comparison of pediatric and adult anesthesia malpractice claims, Anesthesiology 78:461-467, 1993.
20. Morray J, Haberkern C: Quality improvement in pediatric anesthesia. In: Bissonette B and Dalens B (eds) Principles and Practice of Pediatric Anesthesia, McGraw Hill, 2002, pp1509-1517.