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Research Article

The outcome of anaesthesia related cardiac arrest in a Sub-Saharan tertiary hospital

O.O. Adekola [a,*], G.K. Asiyanbi [b], I. Desalu [a], J.O. Olatosi [a], O.T. Kushimo [a]

[a] *Department of Anaesthesia and Intensive Care Unit, College of Medicine, University of Lagos, Lagos University Teaching Hospital, P.M.B 12003, Lagos, Nigeria*
[b] *Department of Anaesthesia and Intensive Care Unit, Lagos University Teaching Hospital, P.M.B 12003, Lagos, Nigeria*

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KEYWORDS

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Abstract *Background:* Anaesthesia related cardiac arrest is undesirable, and different attempts have been made to reduce the mortality associated with it through continuous specialist training, and provision of state of art equipment, combined with rigorous research.

Patients and methods: We determined the outcome of all cardiac arrests that occurred within 24 h of a surgical procedure and anaesthesia from January 2013 to May 2014.

Results: There were nine anaesthesia related cardiac arrest in 4229 cases, (incidence of 21.28 per 10,000), with a mortality of 7/4229; (16.55 per 10,000). There were 60 perioperative cardiac arrests (incidence of 141.88 per 10,000), with a mortality of 55/4229 (130.05 per 10,000). There was return of spontaneous circulation in 34 (56.67%) cases, among them only 7 (20.59%) survived to hospital discharge. The independent determinant of perioperative mortality was the duration of cardiac arrest ⩾ 5 min (RR 10.50, 95% CI 2.721–40.519, $p < 0.001$), cardiac arrest in the absence of a witness (RR 9.56, 95% CI 2.486–36.752, $p < 0.001$), nonstandard time of cardiac arrest (RR 3.2, 95% CI 1.792–5.714, $p < 0.001$), ASA physical status ⩾ III (RR 2.017, 95% CI 1.190–3.417, $p = 0.017$), and emergency surger (RR 2.17, 95% CI 1.151–4.049, $p = 0.011$).

Conclusion: Anaesthesia related cardiac arrest and mortality were linked to cardiovascular depression from halothane overdose in our institution. The burden can be reduced by improving on establishing standard monitoring in the perioperative period, and a team approach to patients care.

1. Introduction

Unexpected death sometimes occurs in the course of a surgical procedure or administration of an anaesthetic. This has been attributed to multiple confounding factors such as human error, anaesthesia and surgical techniques, the emergency nature of the procedure, the American Society of Anesthesiologists (ASA) physical status classification III and higher, presence of underlying medical disease, and extremes of age [1–3]. Attempts have been made to reduce the mortality during

* Corresponding author at: Department of Anaesthesia and Intensive Care Unit, College of Medicine, University of Lagos, Lagos University Teaching Hospital, P.M.B 12003, Lagos, Nigeria. Tel.: +234 8033242924.
E-mail address: oyebolaadekola@yahoo.com (O.O. Adekola).
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anaesthesia and surgery through continuous cardiopulmonary resuscitation and specialist training, improvement in surgical and anaesthetics techniques, the use of perioperative monitoring, and specific guidelines for critical incident [1–3].

The mortality related to anaesthesia has been described as death under, as a result of, or, within 24 h of an anaesthetic [2,4]. This has declined during non-cardiac surgery in recent time in the developed countries to a range from 0.5 to 1 per 10,000 in adults, and 1.4 to 4.6 per 10,000 in children.[1] The mortality following anesthesia related cardiac arrest however, is still high in developing nations with a range from 3.14 to 29.6:10,000 in a surgical population [4,5]. The high rate has been attributed to poor infrastructure, lack of adequate monitoring facilities, delay in presentation to tertiary or specialist facilities, and shortage of blood products [4–6].

The factors contributing to anaesthesia related cardiac arrest include airway or ventilation problems, medication accidents, poor preoperative evaluation, poor supervision of trainees, and infusion/transfusion mishaps [4,7].

There is a need for continuous evaluation of events which might contribute to perioperative cardiac arrest and mortality. This might give an insight into the quality of anaesthesia and surgical care, allow preventive measures to be instituted, and provide direction for future research. We investigated the outcome of anaesthesia related cardiac arrest and mortality during the 24 h period of non-cardiac surgical procedures.

1.1. Patient and methods

This was an audit of anaesthesia related cardiac arrest from January 2013 to May 2014. Institutional Human and Research Ethics Committee approval was obtained. A standard preoperative assessment was done by the attending anaesthetist the day before elective procedures, and immediately before an emergency or urgent surgical procedure.

1.2. Surgical and anaesthetic technique

All procedures were performed using standard anaesthetic and surgical techniques adapted to the individual procedures, and at the discretion of the attending physician. The monitoring instituted included a noninvasive blood pressure, pulse oximetry, electrocardiogram, capnography, anaesthetic vapour concentration, oxygen concentration, and ventilation parameters according to standard of care and the patients need.

The primary outcome of the study determined the anaesthesia related cardiac arrest and mortality. The secondary outcome determined the crude (total perioperative) cardiac arrest and mortality.

The potential predictors of mortality following cardiac arrest such as age, sex, ASA status, the urgency of surgery as defined by the ASA physical status designation of "emergency" by anaesthesia providers, witnessed cardiac arrest, and the duration of cardiac arrest were studied.

Surgical procedures were grouped according to the unit performing the operation: general surgical procedures, neurosurgical, thoracic, gynaecological, and maxillofacial procedures, as well as paediatric, Ear/Nose/Throat (ENT), trauma, orthopaedic and urological procedures.

The primary electrocardiogram rhythm during the cardiac arrest was recorded (asystole, ventricular fibrillation, ventricular tachycardia or pulseless electrical activity). If several rhythms were present, only the first one recorded was used.

The type of anaesthesia was recorded as the primary type of anaesthetic in use at the time of arrest: general, regional (peripheral neuraxial or central neuraxial) anaesthesia, or monitored anaesthesia care (MAC). If the patient received a combined general and regional technique, it was considered that the arrest occurred during general anaesthesia.

The cadre of the primary anaesthesia provider at the time of cardiac arrest was documented (junior registrar, senior registrar or consultant).

The probable cause of cardiac arrest was determined after the event by one of the researchers by reviewing the anaesthetic data. Other factors analysed included the patient's haemodynamic stability before arrest (defined as the need for infusion of vasopressors before arrest), the occurrence of more than a 10 min period of tachycardia (> 120 beats/min), hypotension (systolic blood pressure < 80 mmHg), or hypertension (systolic blood pressure > 160 mmHg) that preceded the arrest [3].

Cardiac arrest was classified as occurring during induction, maintenance, or emergence or recovery from anaesthesia [3]. The time of arrest was documented as standard time occurred during regular working hours (07:00–16:00, Monday through Friday), or non-standard time during the night (16:01–07:59, Monday through Friday) and weekends (Friday 16:01 through Monday 06:59). This timing is based on the work schedule of the anaesthetist in our institution.

1.3. Definition of variables

Basic safety monitoring in the operating room was described during regional anaesthesia and monitored anaesthesia care to include a non-invasive blood pressure, pulse oximetry and continuous electrocardiogram display, and during general anaesthesia to include additional monitoring: capnography, delivered anaesthetic vapour concentration, oxygen concentration, and ventilation parameters [3].

The primary comorbid conditions were defined by the Hosking diagnostic criteria [8]. These criteria included cardiovascular disease, hypertension, end-stage organ failure, systemic disease, infectious disease or sepsis, cerebrovascular disease, and diabetes mellitus [3,8].

Cardiac arrest was described as an event that required cardiopulmonary resuscitation (CPR). Only cardiac arrest that occurred after anaesthesia has been initiated was included. This included cardiac arrest, which occurred during anaesthesia or surgery, transport to the recovery room or intensive care unit, and during the recovery room stay, ward transfer, or, up to a period of 24 h after an anaesthetic [3]. The crude mortality was described as combined surgical and anaesthetic mortality associated with the administration of anaesthesia within 24 h [2].

[1] The incidence is higher in infants than older children and in children with ASA physical status 3–5 than those with ASA physical status 1–2. Anesthesia related cardiac arrest is linked with respiratory impairment in children. It has been suggested that a reduction in anesthesia related cardiac arrest in pediatric patients can be achieved by improving supervision of junior doctors, encourage additional training, the use of practice guidelines, efficient blood bank services, equipment maintenance, and quality assurance monitoring [2,4].

The probable causes of cardiac arrest were grouped into five categories [3].

Primary haemorrhage was described as intraoperative haemorrhage regardless of aetiology, such as that which resulted from coagulopathy, trauma, surgical, or ruptured vessels.

Primary cardiac arrest was described as causes which included myocardial infarction, high-degree blocks, or dysrhythmias due to any aetiology, such as electrolyte abnormalities, placement of pacemaker wires, and medication related asystole.

Respiratory was described as causes which included hypoxia secondary to primary pulmonary pathology, or respiratory depression and upper airway obstruction.

Anaesthesia related death included the following.[3]

Any cardiac arrest that occurred after an anaesthetic drug (narcotic, muscle relaxant, induction agent) was administered to a stable patient who had either immediate arrest or depression of ventilation leading to hypoxemic cardiac arrest.

Airway management was described as inability to intubate the trachea, unrecognized accidental extubation, postextubation hypoxia and loss of airway during tracheostomy.

Other causes were described as pulmonary embolic event such as thromboembolism, air, fat, or carbon dioxide embolism, anaphylactic or other drug reactions.

The outcome of cardiac arrest included the following.

The return of spontaneous circulation (ROSC) was described as survival for at least 1 h after initial resuscitation.

Hospital survival was described as survival to discharge from the hospital.

Dead was described as the cessation of spontaneous circulation and breathing after a cardiac arrest despite cardiopulmonary resuscitation.

1.4. Statistical analysis

The incidence of anaesthesia related cardiac arrest and mortality, crude (total perioperative) cardiac arrest and mortality was determined during the study period. Continuous variables were analysed using mean ± SD, mean and interquartile range (IQR), while categorical variables were analysed with frequency, and/or percentile. The association between independent predictors of perioperative mortality was determined using Chi square or Fischer's Exact as appropriate.

All analyses were performed with the use of Statistical Package for the Social Sciences (SPSS) for Windows Computer Software Version 20 Programme. A p value less than 0.05 was considered statistically significant.

2. Results

There were nine anaesthesia related cardiac arrests in 4229 cases (incidence of 21.28 per 10,000), with a mortality of 7/4229 (16.55 per 10,000) Table 1.

There were 60 perioperative cardiac arrests (incidence of 141.88 per 10,000), with a mortality of 55/4229 (130.05 per 10,000). Adults were 3242 (76.66%) and females 2325 (54.97%).

There was the return of spontaneous circulation (ROSC) for at least one hour in 34(56.67%), of whom only 7

Table 1 The characteristics of anaesthesia related cardiac arrests ($n = 9$).

No	Characteristic of surgery			Characteristic of cardiac arrest					
	Age	ASA	Type	Time of arrest	Event leading to arrest	ECG rhythm	Period	Outcome	Anaesthesia
1	2 months	II	General	Standard	Loss of airway/unable to ventilate		Induction	Hospital survival	GA
2	2 months	I	Urological	Standard	Adverse drug event: overdose of halothane		Induction	Hospital survival	GA
3	2 years	II^E	ENT	Nonstandard	Adverse drug event: overdose of halothane		Induction	ROSC	GA
4	3 years	III	General	Nonstandard	Adverse drug event: overdose of halothane	Asystole	Induction	Dead	GA
5	4 months	IV^E	ENT	Nonstandard	Loss of airway/unable to ventilate		Induction	Dead	Monitored anaesthesia care
6	43 years	IV^E	ENT	Nonstandard	Loss of airway/unable to ventilate		Induction	ROSC	Monitored anaesthesia care
7	54 years	III	General	Nonstandard	Adverse drug event: overdose of isoflurane	Asystole	Maintenance	ROSC	GA
8	68 years	III	General	Standard	Adverse drug event: overdose of isoflurane	VT	Maintenance	ROSC	GA
9	75 years	III^E	General	Nonstandard	Adverse drug event: overdose of isoflurane	VF	Induction	ROSC	GA

ASA – American Society of Anesthesiology physical status.
ENT – Ear/Nose/Throat.
VT – ventricular tachycardia.
VF – ventricular fibrillation.
ROSC – return of spontaneous circulation.
GA – general anaesthesia.

Table 2 The distribution of perioperative cardiac arrest and hospital survival in a surgical population.

Variables	Cardiac arrest (n = 60)		Hospital survival (n = 7)	
	Frequency (f)	Per cent (%)	Frequency (f)	Per cent (%)
Age (years)				
1–18	14	23.33	3	21.43
>18	46	76.67	4	8.69
Sex				
Female	33	55	3	9.09
Male	27	45	4	14.81
ASA				
I	2	3.33	2	100
II	14	23.33	2	14.29
III	19	31.67	2	10.53
IV	19	31.67	1	5.26
V	6	10	0	0
E	35	58.33	3	8.57
Co-existing disease				
None	49	81.67	5	10.20
Hypertension[a]	8	13.33	1	12.5
Diabetes mellitus[a]	5	8.33	0	0
Sickle cell disease	1	1.67	1	100
Location				
Recovery	33	55	0	0
Operation room	24	40	7	29.17
Ward	2	3.33	0	0
In transit	1	1.67	0	0

ASA – American Society of Anesthesiology physical status.
[a] Some patients had more than one co-existing medical condition.

(20.59%) cases survived to hospital discharge. The median duration of cardiac arrest was 24.58 ± 16.51 min.

The frequency of cardiac arrest was higher during non-standard working hours 40(66.67%), and in the recovery room 34(56.67%) Table 2. Basic monitoring facility was available in 12(20%). A high proportion of the anaesthesia was administered by trainee anaesthetists 47(78.33%).

Cardiac arrest occurred more among patients scheduled for general surgical procedures 18(30%), followed by neurosurgical 10(16.67%) and gynaecological procedures 9(15%) Table 3.

Primary haemorrhage was the most frequent cause of perioperative cardiac arrest in 28(46.67%), followed by non-haemorrhagic hypovolaemia in 9(15%) Table 4.

The independent determinant of perioperative mortality was the duration of cardiac arrest $\geqslant$ 5 min (RR 10.50, 95% CI 2.721–40.519, $p < 0.001$), cardiac arrest in the absence of a witness (RR 9.56, 95% CI 2.486–36.752, $p < 0.001$), non-standard time of cardiac arrest (RR 3.2, 95% CI 1.792–5.714, $p < 0.001$), ASA physical status $\geqslant$ III (RR 2.017, 95% CI 1.190–3.417, $p = 0.017$), and emergency surgery (RR 2.17, 95% CI 1.151–4.049, $p = 0.011$) (Table 5).

Cardiac compression was instituted in all episodes of cardiac arrest; 52(86.67%) received adrenaline at a median dosage of 2.5 (IQR 2-4 mls), and a median interval of 5.62 min.

Three of the patients with halothane induced cardiac arrest receive atropine at a median dosage of 1.2 mg. Electrocardiogram rhythm during the arrest was asystole in 2, pulseless electrical activity (1), ventricular fibrillation (2) and ventricular tachycardia (2). Automated electrical defibrillation was, however, instituted only in one of the patients with a shockable rhythm.

3. Discussion

The anaesthesia related cardiac arrest in our audit was 21.28 per 10,000 cases, with an associated mortality of 16.55 per 10,000 cases. This value is close to a reported prevalence of 25.5:10,000 and 11.5:10,000, respectively in our environment [4]. We attributed the difference in rates to variation in study methods; we conducted a cohort study of perioperative cardiac arrest, while their study was a case control type, in which data may be under, or overestimated, due to the time lag between event, data collation and analysis. In spite of the difference, both studies were conducted in a similar practice environment, where there is poor infrastructure, inadequate monitoring facilities, and a great proportion of anaesthesia and surgical procedures are conducted by trainees, outside the standard working hours [4].

On the contrary, anaesthesia related and crude mortality is on the decline globally [1–3]. This is a result of continuous cardiopulmonary resuscitation and specialist training, improvement in surgical and anaesthetics techniques, the use of perioperative monitoring, and the adoption of specific guidelines for critical incidents in the perioperative period [1–3]. Despite the decline, the principal cause of mortality is still the same, and it includes inadequate supervision of trainees, drug overdose, drug mistakes, airway obstruction, aspiration of gastric contents, insufficient monitoring, and lack of postoperative care [2].

Table 3 The distribution of perioperative cardiac arrest by surgical procedures.

Surgical procedures				
General surgical n = 18(30%)	Neurosurgical n = 10(16.67%)	Gynaecological n = 9(15%)	Orthopaedic n = 7(11.67%)	Cardiothoracic n = 5(8.33%)
Exploratory laparotomy (11)	Craniectomy for tumour excision (4)	Exploratory laparotomy (4)	Open reduction internal fixation (2)	Pericardiotomy (1)
Endoscopic retrograde cholangiopancreatogram [ERCP], (1)	Endoscopic third ventriculostomy (1)	Vaginoplasty (1)	Below knee amputation (1)	Left closed thoracostomy tube drainage for massive bilateral carcinomatous pleural effusion (1)
Mastectomy (3)	Reversion of ventriculo-peritoneal shunt + cleft palate pair (1)	Enterocutaneous fistula with faecal peritonitis (1)	Hip arthroplasty (1)	Pacemaker insertion (1)
Splenectomy (1)	Bore hole (1)	Subtotal hysterectomy (2)	Bilateral below knee amputation (1)	Mediastinal tumour excision (1)
Excision of cancer of colon (1)	Ventriculo-peritoneal shunt for haemorrhagic stroke (1)	Myomectomy (1)	Above knee amputation (1)	Right closed thoracostomy tube drainage for tension pneumothorax (1)
Thyroidectomy (1)	Cervical discectomy (1)		Ray amputation (1)	
	Surgical procedures			
	Paediatric n = 4(6.66%)	ENT n = 5(8.33%)	Trauma n = 1(1.67%)	Maxillofacial n = 1(1.67%)
	Excision of sacral teratoma (1)	Adenotonsillectomy (1)	Above knee amputation (1)	Parotidectomy (1)
	Inguinal herniotomy (1)	Parotidectomy (1)		
	Jejunal resection (1)	Tracheostomy (3)		
	Reversal f colostomy (1)			

Table 4 The causes of perioperative cardiac arrest.

Causes	n = (60) Frequency (f)	Percent (%)
Primary haemorrhage	29	48.33
Non-haemorrhagic hypovolaemic	9	15
Anaesthesia related		
Adverse drug event[a]	7	11.67
Loss of airway/unable to ventilate	2	3.33
Primary cardiac		
Dysrhythmias	2	3.33
Placement of pacemaker wires	1	1.67
Respiratory		
Inadequate oxygenation	1	1.67
Mucous plugged endotracheal tube	1	1.67
Aspiration of gastric content	1	1.67
Others		
Surgical procedure	2	3.33
Co-existing medical condition	5	8.33

[a] Overdose of inhalational agent (halothane or isoflurane).

Table 5 The determinants of perioperative mortality following cardiac arrest.

Variable	Unadjusted RR	95% CI	p value
Age (P)	0.826	0.441–1.546	0.565
Sex (M)	0.955	0.535–1.704	0.875
ASA ⩾ III	2.017	1.190–3.417	0.017
Unwitnessed cardiac arrest	1.208	1.023–1.427	0.016
Comorbidity	1.18	0.67–2.09	0.875
Emergency surgery	2.17	1.151–4.049	0.011
Nonstandard time of cardiac arrest	3.2	1.792–5.714	< 0.001
Duration of cardiac arrest ⩾ 5 min	10.50	2.721–40.519	< 0.001[a]
Hypotension	1.4	0.79–2.481	0.252

RR – relative risk, CI – confidence interval, ASA – American Society of Anesthesiology physical status, P – paediatric, M – male.
[a] Fisher's exact test.

These observations were highlighted in our audit where only 20% of anaesthetized patients had basic monitoring facilities, and a great proportion of anaesthesia and surgery was conducted by trainees. A great proportion of our anaesthesia related cardiac arrest was also due to halothane overdose induced cardiovascular depression, which was common during induction in our paediatric population. This, however, was the pattern of anaesthesia related cardiac arrest before the introduction of sevoflurane for induction in the paediatric population [9]. Halothane is known to cause cardiovascular depression in a dose dependent manner, with depression of the Sino Atrial and Atrioventricular Nodes, and subsequent bradycardia. This may be the reason for the administration of atropine in some of our patients with halothane induced cardiac arrest.

We attributed the over dose of inhalational agent to human error on account of possible fatigue of the attending anaesthetist. This is because a high proportion of the cardiac arrest occurred during non-standard working hours (16–07:59 h), after a full day schedule.

Another contributory factor to anaesthesia related cardiac arrest was loss of airway and inability to ventilate occurred during surgical tracheostomy in two patients with severe airway obstruction. One of them presented with Ludwig's Angina, and the second with orbital teratoma. In such circumstances, an awake fiberoptic intubation would have been an alternative method of securing the airway; however, this facility was unavailable during the audit. Though, there was the return of spontaneous circulation in both patients for 12–24 h, they however, did, not survive to hospital discharge.

The high crude mortality in our study may be related to the late presentation for medical intervention in some of our patients, and the acute shortage of blood products in our institution. As a high proportion of the studied population consisted of those with ASA status of III and above (73.33%), who presented with sepsis, anaemia from massive blood loss, and multiple organ dysfunction require various supportive therapies. The observed mortality in this group of patients was high (93.18%). Other contributory factors to the high mortality after cardiac arrest in our audit included the emergency nature of the surgical procedure, the duration of cardiac arrest greater than five minutes, and the occurrence of the arrest in the absence of a medical personnel.

The prolonged period of arrest in our cohort may also be responsible for the poor hospital survival rate (11.11%), despite the RSOC in 56.67% patients. This may because the chance of survival after cardiac arrest declines by 7–10% for every min of no-flow status [10]. In individuals with cardiac arrest over 12 min, only 2–5% of patients can achieve long-term survival [11,12].

In our audit, hypotension was not a determinant of outcome, and this is similar to observations made in a similar study in our country [4]. However, in an audit conducted in America, haemorrhage and hypotension were significant independent predictors of perioperative arrests and mortality [3].

A high proportion of our studied cohort had general surgical, neurosurgical and gynaecological procedures performed, and this is contrary to observation in a similar study in the country, where they reported a high proportion of neurosurgical and cardiothoracic procedures.

The type of anaesthesia administered was not a determinant of mortality in our study. This is not in agreement with previous documentations of a significant association between general anaesthesia and perioperative cardiac arrest and mortality [2,3].

In our audit, cardiac arrest was observed during MAC, spinal and local anaesthesia. These patients had ASA IV and V physical status, and were in poor clinical conditions. Two of the patients who had arrest during spinal anaesthesia were scheduled for knee amputation on account of poor glycaemic control, sepsis and hypokalemia. The third patient presented with a high preoperative diastolic blood pressure (125 mmHg). All of them did not survive to hospital discharge. In contrast to our reports in previous studies conducted in our country, cardiac arrest was not encountered during local anaesthesia and MAC [4,5]. Sprung et al. [3] however, reported that the frequency of cardiac arrest during general anaesthesia has decreased over time, while that during regional anaesthesia and MAC remained consistent.

Cardiac compression was performed in all our cohort. Some patients, however, did not receive adrenaline. The interval of administering adrenaline was more than that outlined by Advanced Cardiac Life Support guidelines [13,14]. A similar observation regarding adrenaline administration was made by Johansson et al. [15]. Only one of the four patients with shockable rhythm was defibrillated. These observations may suggest that cardiopulmonary resuscitation is not performed according to standard protocol in our institution [13,14]. Nevertheless, the interval of administering adrenaline had reduced compared to an earlier study in our hospital [16,17]. This may be because our hospital developed a locally adapted three day CPR Training Programme for all Doctors and Nurses [15].

In conclusion, anaesthesia related cardiac arrest was associated with cardiovascular depression secondary to halothane overdose. The provision of sevoflurane in the hospital may avert this. There is a need to improve on establishing standard monitoring, promote team work, and audit events after cardiac arrest. This may improve adherence to guidelines and outcome in future studies.

This study is limited by the number of cases, which was insufficient to perform statistical analysis to identify independent factors predicting survival outcome. There is therefore a need for a large scale multicenter study in the country, or a longitudinal study in our institution. This will ensure pooling of a large sample.

Conflict of interest

None declared.

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