Chapter

Life-Threatening Cardiac Arrhythmias during Anesthesia and Surgery

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Abstract

Life-threatening arrhythmias are frequently encountered during anesthesia for cardiac or non-cardiac surgery. They result in a significant cause of morbidity and mortality, particularly in elderly patients. Predisposing factors like electrolytes abnormalities, pre-existing cardiac disease, intubation procedure, anesthetic medications, and various surgical stimulation need to be determined. Early diagnosis and commencement of an appropriate treatment protocol may be lifesaving. Treatment usually involves correction of the underlying causes, cardiac electroversion, and the use of one or more antiarrhythmic agents. Although ventricular tachycardia, ventricular fibrillation, torsade de pointes, and pulseless electrical activity are considered malignant arrhythmias that can lead to cardiac arrest, other types of Brady and tachyarrhythmias are also included in this chapter to enable adopting a more objective approach in the management of arrhythmias intraoperatively, avoiding risks of inappropriate management strategies.

Keywords: arrhythmias, anesthesia, surgery, life-threatening, intraoperatively

1. Introduction

1.1 Definition of cardiac arrhythmias

Accelerated, slowed, or irregular heart rates caused by abnormalities in the electrical impulses of the myocardium.

Cardiac arrhythmias are very common in the general population and are a significant cause of morbidity and mortality of both cardiac and noncardiac surgical procedures during the perioperative period. While the incidence of perioperative arrhythmias is extremely high (the Multicenter Study of General Anesthesia reported a 70.2% incidence of Brady and tachyarrhythmias in 17,201 patients having general anesthesia for a variety of surgical procedures), only 1.6% of these required clinically significant management [1–3]. For cardiac surgery, the patients are more prone to develop arrhythmias with a reported incidence of greater than 90%, while incidence for patients undergoing non-cardiac surgery is lower and varies from 16.3 to 61.7% [4–6]. Patients with pre-existing cardiac disease for cardiac surgery are more prone to develop perioperative rhythm disturbances. It is obvious that arrhythmias that occur during surgery are clinically important as it can evolve to life-threatening malignant arrhythmias with severe hemodynamic instability and

Class	Actions	Drugs (examples)
I	Sodium channel blockade	
IA	~moderate	Quinidine, procainamide
IB	~weak	Lidocaine, mexiletine
IC	~strong	Flecainide, propafenone
II	β blockade	Propranolol, esmolol, sotalol
III	Potassium (K^+) channel blocker	Amiodarone, ibutilide
IV	Calcium (Ca^{2+}) channel blocker	Diltiazem, verapamil

Table 1.
Choice of antiarrhythmic therapies based on Vaughan-Williams classification (classes I–IV).

cardiovascular collapse, necessitating prompt initiation of adequate cardiopulmonary resuscitation (CPR) and defibrillation or electrical cardioversion. Hence, a thorough understanding and prompt diagnosis and intervention are critical for the anesthesiologist in order to reduce severe perioperative adverse outcomes.

An understanding of normal cardiac physiology is essential before rhythm disturbances can be understood. The normal cardiac electrical conduction system is responsible for the contraction of the heart muscle and is represented on the electrocardiogram.

Normal cardiac conduction begins with cardiac impulses coming from the sinoatrial node and travels to both atria. The atria depolarizes and generates the P wave. From here, the impulse propagates to the atrioventricular node, then reaches the his bundle and Purkinje fibers transforming into conduction, causing ventricular contraction and generates the QRS wave [6]. The resting sinus heart rate in adults is usually between 60 and 100 beats/min.

In the heart, electrical stimulation is created by a sequence of ion fluxes through specialized channels in the cardiomyocytes that generate action potential and lead to a coordinated cardiac contraction in systole. Each action potential corresponds to one beat of the heart and the inherent frequency of these cells is essential for maintaining proper rate control. Antiarrhythmic drugs act by modifying this action potential, which results from the alteration of ion channels (**Table 1**).

Five phases of cardiac action potential (as illustrated in **Figure 1**):

- Phase 4: resting potential at –90 mV with minor depolarization from –90 mV to –70 mV; the passive outflow of potassium.
- Phase 0: rapid depolarization from –70 mV to +50 mV; inward voltage-gated sodium channels.
- Phase 1: minor repolarization; outward voltage-gated potassium channels.
- Phase 2: plateau at +50 mV; outward voltage-gated potassium channels and inward voltage-gated calcium channels.
- Phase 3: repolarization from +50 mV to –90 mV; outward voltage-gated potassium channels.

Damage to the normal conduction system of the heart can lead to rhythm disturbances which can be either benign or more serious in nature depending on the hemodynamic consequence of the arrhythmia and the possibility of evolving into a lethal arrhythmia.

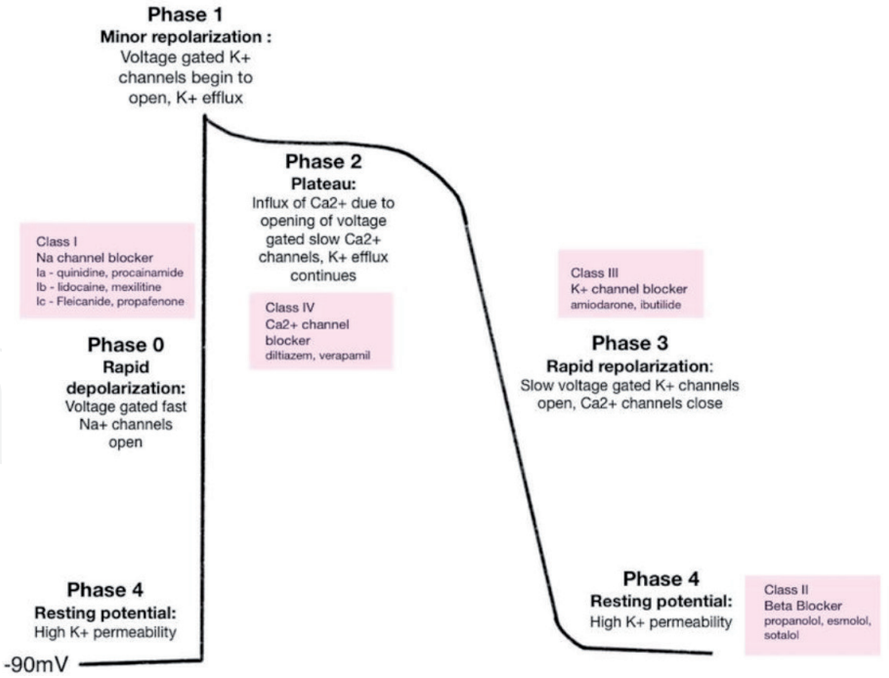

Figure 1.
Cardiac action potential.

Bradyarrhythmias result from decreased intrinsic pacemaker function or blocks in conduction, principally within the AV-node or the His-Purkinje system. Most tachyarrhythmias are caused by re-entry, some result from enhanced normal automaticity or from abnormal mechanisms of automaticity.

2. Mechanism of intraoperative arrhythmias

The principal mechanisms of Brady and tachyarrhythmias which are observed in clinical practice are as follows [5]:

1. Injury to the cardiac conduction system.

2. Re-entry: a mechanism that may precipitate a wide variety of supraventricular and ventricular arrhythmias, implying the presence of a pathologic circuit of an electrical impulse around a functional or anatomic loop.

3. Automaticity: Abnormal depolarization of atrial or ventricular muscle cells during periods of the action potential can lead to arrhythmias.

4. Mutations in ion channels.

5. Ectopic foci.

3. Contributing factors and causes of arrhythmias during anesthesia for surgery

Although the incidence of intraoperative arrhythmias is extremely high, the majority of such arrhythmias are benign and self-limiting. They require no emergency treatment and respond well to pharmacologic interventions or both.

However, in certain patients, some arrhythmias may pose an immediate threat to life by causing profound hemodynamic instability and require urgent clinical attention.

There are several factors likely to contribute to the generation of intraoperative arrhythmias, which can be classified according to patient, anesthesia, and procedures (**Table 2**) [5, 7]. Identifying the causes mainly responsible for intraoperative arrhythmias is prudent before instituting specific therapy or intervention.

Patients with pre-existing heart disease (e.g., myocardial ischemia) have a much higher incidence of arrhythmias intraoperatively. Intracranial pathology such as subarachnoid hemorrhage and raised intracranial pressure can result in ECG abnormalities because of stimulation of the autonomic nervous system.

Airway manipulation most often associated with hemodynamic disturbances is a well-described cause of intraoperative arrhythmias [4, 7, 8]. During anesthesia, arrhythmias can be produced in the presence of a variety of triggering agents and clinical situations such as light plane of anesthesia with hypertension and tachycardia, hypoxemia, and hypercarbia.

Vital organs, such as the brain, heart, and kidneys, must be perfused adequately during general anesthesia and surgery. Most of the anesthetic agents have direct myocardial depressant effects which result in reduced cardiac contractility and sympathetic stimulation of the peripheral vasculature. The net effect is a fall in cardiac output with a fall in perfusing pressure of vital organs secondary to vascular vasodilation. Conversely, tachycardia can have detrimental effects in patients susceptible to ischemia due to reduced myocardial filling time. Even relatively minor fluctuations in cardiovascular and hemodynamic parameters due to arrhythmias can have a significant incidence of various complications, including

Patient-related	**Anesthesia related**	**Surgical related**
Pre-existing cardiac disease, thyrotoxicosis, central nervous system disease (e.g., subarachnoid hemorrhage)	Direct laryngoscopy and intubation	Cardiac surgery (intracardiac surgical manipulation)
	Insufficient level of anesthesia	Surgical manipulation during non-cardiac surgery (e.g., traction to the intestine, oculocardiac reflex). Neurosurgical causes, dental surgery, and laparoscopic surgery
Elderly	Local anesthesia (central neuraxial blockade is associated with pharmacological sympathectomy)	
	Mechanical irritation (e.g., central venous lines, pulmonary artery catheter, chest tube)	
	4Hs 4Ts Hypovolemia, hypoxemia, hyper/hypokalemia (electrolyte disorders) and metabolic disorders (acidosis), hypothermia/hyperthermia Tension pneumothorax, tamponade, toxins/drugs, thromboembolism (pulmonary/cardiac)	

Table 2.
Contributing factors and causes of intraoperative arrhythmias.

cardiovascular events, renal failure, infection, and cerebral infarction, particularly among the elderly co-morbid patients undergoing elective and emergency surgery. The use of halothane during induction in children as well as maintenance of anesthesia has been largely superseded by sevoflurane, which is safer. Electrolyte imbalances, abnormal blood gases, and direct cardiac stimulation via catheters influence the occurrence of arrhythmia and conduction abnormalities. The anesthesiologists should be aware of all the drugs used and able to manage the consequences accordingly [8, 9].

Surgical manipulation and cardiopulmonary bypass during cardiac surgery may precipitate arrhythmias. Vagal stimulation in surgical procedures such as during carotid surgery and peritoneal traction produces bradycardia, conduction block, or even asystole. Dental surgery causes profound stimulation of the autonomic nervous system. Thoracic surgery is associated with an incidence of atrial fibrillation.

4. Anesthetic agents and adjuvants related to arrhythmias

Prolonged cardiac repolarization (represented as QT interval on ECG) induced by various anesthetic agents and adjuvant drugs may trigger the appearance of torsade de pointes (TdP), which in some patients degenerate towards malignant ventricular arrhythmias and sudden cardiac arrest [6, 7, 10]. The duration of QT interval, QT corrected for heart rate (QTc), JT interval, QT dispersion (QTd), QT variability index, and transmular dispersion of repolarization (TDR) are the commonly used ECG markers to check for the possibility of various degrees of TdP under different conditions [11]. All volatile anesthetics, especially isoflurane and desflurane cause QT_C prolongation, while sevoflurane demonstrated no effects on TDR. Propofol is generally considered to be non-torsadogenic. The sympathomimetic properties of ketamine may promote the incidents of TdP [11]. Most opioids have no effect on QTc when used at clinically relevant doses. Succinylcholine has been shown to increase QTc, especially when used in conjunction with thiopental while most nondepolarizing muscle relaxants have no effect on the QT interval. Sugammadex at therapeutic doses has no effect on QTc whereas anticholinesterase-anticholinergic antagonism of neuromuscular blockade with neostigmine and glycopyrrolate or atropine causes clinically significant QTc prolongation. The commonly used local anesthetic agents are relatively safe; nevertheless, extensive central neuraxial blocks may increase the duration of QTc. Several antiemetic drugs, such as droperidol, domperidone, and most 5-HT3 antagonists, produce a significant prolongation of QT. The FDA's black box warning of fatal arrhythmias associated with the administration of droperidol leads to a decrease in the use of this medication in recent years. Midazolam seems to have no effect on QTc and TDR. Although dexmedetomidine may cause mild prolongation of QT interval, it is unlikely to cause TdP. It should also be prudent to use dexmedetomidine with caution, especially in patients with bradyarrhythmias tendencies where the risk of QT prolongation is increased [12–15].

5. Diagnostic evaluation

During surgery, it is not always possible to get 12-lead ECG done. The anesthesiologists would have to make the diagnosis by looking at the continuous ECG monitor in the operating room. Changing the sweep speed on the ECG monitor (from 50 to 25 mm/s) may help with the identification of arrhythmias and their

management. Lead II and V5 are superior for arrhythmia detection and diagnosis. All available leads are displayed on the intraoperative monitor if arrhythmia develops and cannot be readily diagnosed. For non-cardiac surgery, 12-lead ECG can be obtained as soon as feasible [9, 16].

The blood pressure, arterial oxygen saturation, and temperature also need to be monitored. More advanced monitoring such as invasive arterial pressure, pulmonary artery catheterization, and transoesophageal echocardiography can provide additional clues when assessing the patient for causes of cardiovascular collapse. End-tidal carbon dioxide may help with the effectiveness of chest compressions during cardiopulmonary resuscitation. Estimation of serum electrolytes for verification of renal function is important in patients on medications for arrhythmias.

Adequate precautions should be taken during surgery to prevent the development of intraoperative arrhythmias:

- Surgical manipulations which can precipitate arrhythmias should be kept to a minimum.
- Adequate depth of anesthesia may prevent or control intraoperative arrhythmias.
- Hypoxia, hypotension, hypovolemia, hypothermia should be prevented during surgery.

Whenever an arrhythmia develops in the intraoperative period, the anesthesiologists should first be able to eliminate the possible causes of arrhythmia before instituting specific interventions. Attempts to correct them should be made while continuing to evaluate the arrhythmia.

6. Specific intra-operative arrhythmias

6.1 Antiarrhythmic drugs

Patients requiring oral antiarrhythmic should continue the medication until the time of surgery. Specific cardiologist consultation is advised for patients who require pacing-cardioverter devices to suppress or terminate tachyarrhythmias. With life-threatening circulatory compromise, prompt pacing or electroversion is required. Obvious electrolyte imbalance should be corrected, and management provided for underlying heart disease. Specific antiarrhythmic agents are used to suppress arrhythmias and prevent recurrences (**Table 3**).

The administration of antiarrhythmic drugs may paradoxically aggravate the arrhythmias that are being treated or cause new rhythm disorders. This is known as proarrhythmia generally occurs when the dosage of drugs does not exceed the therapeutic range [17]. Proarrhythmia is now considered omnipresent with all antiarrhythmic medications. Care should be taken when using antiarrhythmic drugs in patients with structural heart disease, as they are at higher risk of proarrhythmia with antiarrhythmic medications. These patients, such as heart failure or cardiomyopathy are not candidates for Class IC or Class III antiarrhythmics other than amiodarone or sotalol.

Antiarrhythmic agents, in general, have a narrow therapeutic index. As a result, they are often susceptible to drug interactions with anesthetic agents and can cause significant adverse effects (**Table 4**).

Drug	Action	Dose
Adenosine	AV nodal blockade	6–12 mg
Amiodarone	Class III antiarrhythmic	Bolus 150 mg over 10 min; repeat, if necessary, maintenance of 1 mg/min for 6 hours, then 0.5 mg/min Total dose over 24 h ≤2.4 g
Digoxin	Indirect vagomimetic and slows conduction through AV node	0.5–1.0 mg loading (dose 1: 50%; 25% at 4 hourly intervals; then 0.125–0.25 mg daily) Digoxin levels must be monitored for toxicity (therapeutic blood level: 0.8–2.0 ng/mL; if >3 ng/ml is indicative of toxicity)
Diltiazem	Ca channel antagonist	20 mg × 1 min (0.25 mg/kg) 0.125 mg/kg
Esmolol	Ultrashort-acting β-blocker	150–500 μg/kg × 1 min. 50–200 μg/kg/min
Isoproterenol	β-agonist	2–20 μg/min (0.02–0.15 μg/kg/min)
Lidocaine	Na channel action decreasing duration of action potential	1.0–1.5 mg/kg; 1–4 mg/min (20–50 μg/kg/min)
Propranolol	β-blocker	0.5–3 mg (10–30 μg/kg) q 2 min to max 6–10 mg
verapamil	Ca channel antagonist	5–10 mg. Repeat bolus if needed (maximum dose 30 mg)
Procainamide	Class Ia antiarrhythmic	20–50 mg/min or 100 mg every 5 min until arrhythmia is controlled, QRS prolonged by 50% of original width, hypotension occurs, or total cumulative dose of 17 mg/kg
Sotalol	Class III antiarrhythmic	75 mg over 5 h

Table 3.
The main intravenous agents useful in management of intraoperative arrhythmias.

Antiarrhythmic drugs	Interaction with anesthetic agents
adenosine	• Vasodilation with isoflurane and central neuraxial block • Bronchoconstriction with neostigmine • Asystole with neostigmine, dexmedetomidine, and opioids • Antagonism with aminophylline
digoxin	• Bradycardia is potentiated by halothane and succinylcholine • Caution when using calcium and diuretics (digoxin toxicity)
β blocker	• Myocardial depression with halothane • Bronchoconstriction with neostigmine and atracurium
quinidine	• Prolongs the effects of neuromuscular blocking agents
procainamide	• Antagonizes neostigmine
calcium channel blocker	• Bradycardia and myocardial depression with halogenated agents and dantrolene • Potentiates neuromuscular blockers
lidocaine	• Potentiates the sympathetic blockade of opioids

Table 4.
Antiarrhythmics and its interaction with anesthetics agents.

6.2 Pacing and cardiac electroversion

Both cardiac pacing and electroversion provide a more prompt therapeutic effect and easier dose titration (i.e., pacing mode, rate, current) than with drugs

[8, 18]. They have advantages over drugs for the management of intraoperative arrhythmias.

Pacing is considered in patients with symptomatic bradycardia when a pulse is present but not responded to atropine or second-line drugs (e.g., adrenaline and dopamine) [19]. Temporary cardiac pacing is used in cardiac surgery to increase heart rate, suppress bradycardia dependent tachycardia, overdrive escape rhythms, suppress atrial or ventricular extrasystoles, and terminate re-entrant SVT or atrial flutter. Transcutaneous pacing is used if invasive pacing is not feasible or is impractical.

Cardiac electroversion includes cardioversion (synchronized shocks) or defibrillation (nonsynchronized shocks) of hemodynamically unstable patients, which use high-energy capacitor discharges to simultaneously depolarize all excitable myocardium to terminate arrhythmias. It is highly effective and avoids the potential complications of drug therapy [20]. Defibrillation or unsynchronized cardioversion is indicated in any patients with pulseless ventricular tachycardia or ventricular fibrillation whereas synchronized cardioversion is utilized for the treatment of persistently unstable tachyarrhythmias in patients without loss of pulse. In synchronized cardioversion, the direct current electrical discharge is synchronized with the R or S wave of the QRS complex, avoiding the energy delivery near the apex of T wave, which coincides with a vulnerable period of induction of ventricular fibrillation. The recent use of biphasic cardioversion has shown that less energy is required to convert an arrhythmia to a sinus rhythm. It results in fewer delivered shocks to the patient, less cumulative energy delivered, and less myocardial tissue damage than is found with higher voltage shocks.

6.3 Management of arrhythmias during anesthesia and surgery

Cardiac arrhythmias may not always require treatment. However, the distinction between benign and malignant arrhythmias which carry the risk of sudden death is fundamental [21]. **Figure 2** provides an algorithm for the evaluation and management of rhythm disturbances.

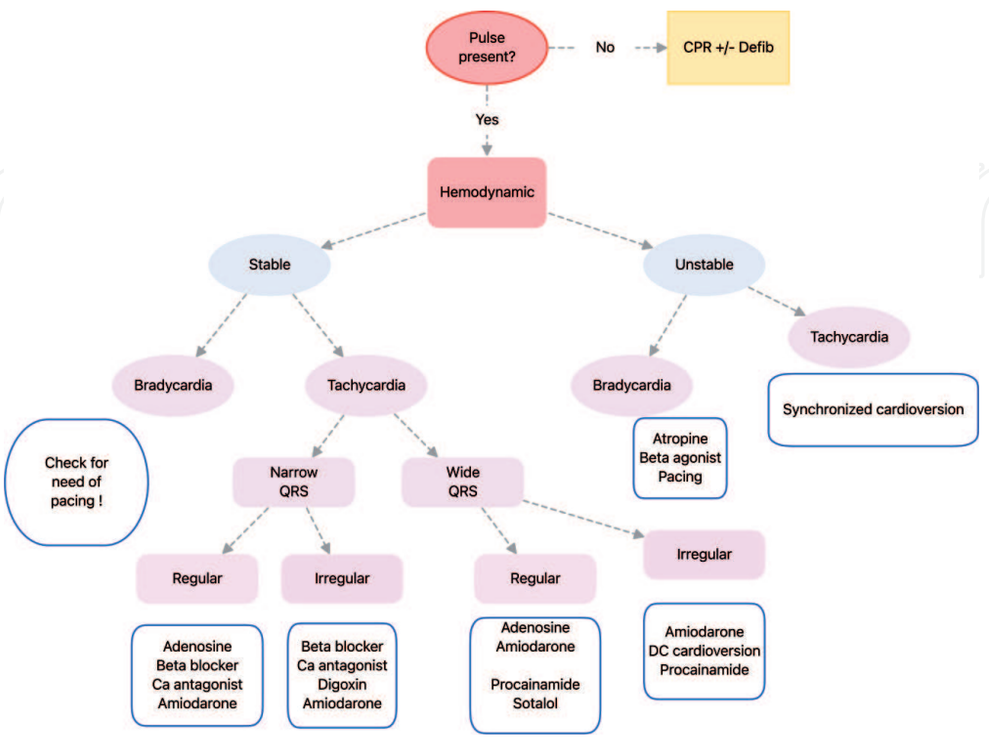

Figure 2.
Management of arrhythmias developed during anesthesia and surgery.

Bradyarrhythmia		**Tachyarrhythmia**		
Sinus arrhythmia	**Conduction defects**	**Sinus arrhythmia**	**Supraventricular arrhythmias**	**Ventricular arrhythmias**
Sinus bradycardia	AV-blocks 1. First degree AV-block 2. Second degree AV-block 3. Third degree AV-block Intraventricular blocks 1. Right bundle branch block (RBBB) or Left bundle branch block (LBBB) 2. Fascicular block. • Left anterior hemi-block (LAHB) • Left posterior hemi-block (LPHB) 3. Bifascicular block 4. Trifascicular block	Sinus tachycardia	1. Premature atrial contraction 2. Paroxysmal supraventricular tachycardia 3. Atrial flutter 4. Atrial fibrillation	1. Premature ventricular contractions (PVCs) 2. Ventricular tachycardia (VT) 3. Ventricular fibrillation 4. Torsade de pointes

Table 5.
Classification of bradyarrhythmia and tachyarrhythmia.

Arrhythmias are broadly classified as bradyarrhythmia and tachyarrhythmia (**Table 5**).

Strategies for clinical care of a patient with bradyarrhythmia (**Figure 3A**) and tachyarrhythmia (**Figure 3B**).

6.4 Bradyarrhythmia

6.4.1 Conduction defects

AV conduction block can occur in the settings of intrinsic cardiac disease, acute myocardial ischemia, general anesthetics, electrolyte abnormalities, and excessive vagal tone. In cardiac surgery, high-grade AV block is not so uncommon complication and thus, a temporary epicardial pacing system is necessary. AV block is classified as first, second, and third-degree (complete). First-degree AV block is generally benign, often needs no treatment apart from careful observation for progression to a higher degree of the block that requires prompt treatment. The second-degree AV block is divided into Mobitz type I and II. In Mobitz type I, the block is often transient and asymptomatic. In Mobitz type II, the block is often symptomatic and has a less favorable prognosis because there is a potential risk of progression to third-degree heart block. Pacing is required if there is severe bradycardia with hemodynamic insufficiency. Third-degree AV block is characterized by electrical instability and may evolve towards asystole. There is no apparent relationship exists between the P waves and QRS complexes. Pacing is typically required because no conduction to ventricles occurs with atrial activity more rapid than ventricular activity (approximately 20–40 beats/min). These bradyarrhythmia (Mobitz type II and third-degree heart block) are not likely to be responsive to atropine and should be treated with transcutaneous pacing or isoproterenol infusion acting as a "chemical pacemaker" while the patient is prepared for transvenous pacing.

Intraventricular conduction defects are generally classified as LBBB, RBBB, or Hemiblock. Intraventricular blocks may be of a His bundle branch block pattern, a fascicular block pattern, or both and result from significant slowing or interruption

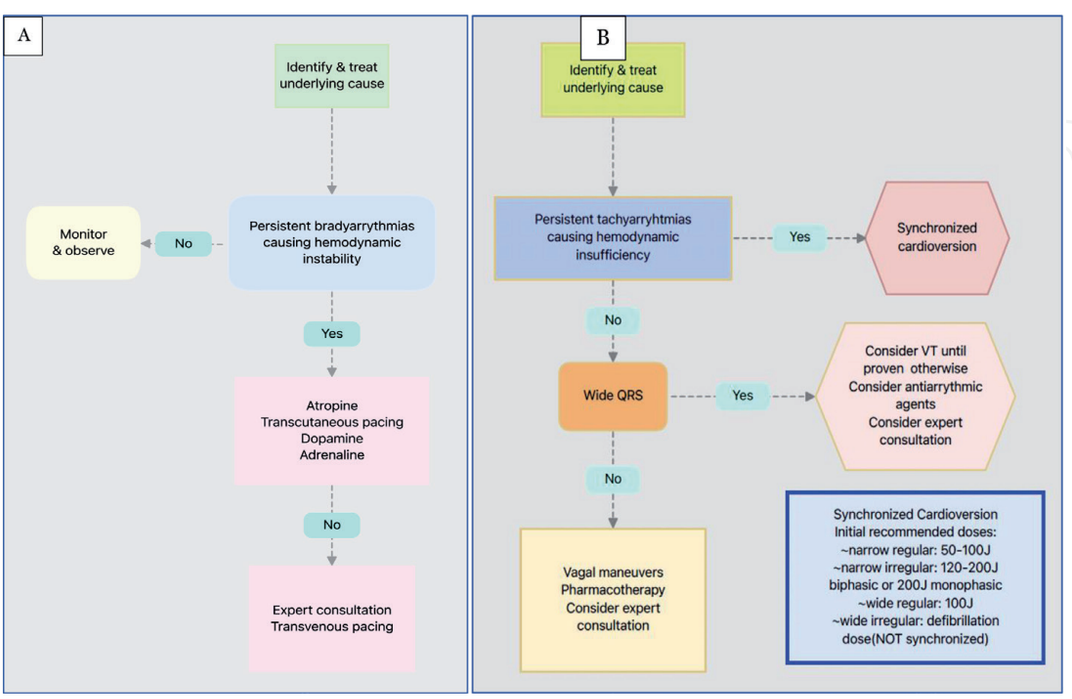

Figure 3.
Algorithm for (A) bradyarrhythmia. Algorithm for (B) tachyarrhythmias. VT: ventricular tachycardia.

of conduction. They are frequently seen in those with and without cardiac disease. In the vast majority of cases, RBBB may be a normal variant with little or no impact on cardiovascular prognosis. LBBB is a more serious conduction disturbance and is always associated with significant heart disease. In the presence of LBBB, acute myocardial infarction is difficult to diagnose. When the LBBB occurs in myocardial infarction, a complete heart block may develop. The anatomical or functional block in a fascicle causes a fascicular block. The left anterior hemiblock is common, while the left posterior hemiblock is uncommon. The combination of RBBB with left anterior or posterior hemiblock is called bifascicular block. Trifascicular block refers to a block of both the left and right bundles or to first- or second-degree AV block with additional bifascicular block. Patients with bifascicular and trifascicular blocks are at risk of a slow progression to an advanced or complete AV block.

6.5 Tachyarrhythmia

Tachyarrhythmias are classified into two categories (narrow complex supraventricular tachycardia and wide complex tachycardia), based on the appearance of QRS complex, heart rate, and regularity.

6.5.1 Supraventricular arrhythmias

6.5.1.1 Premature atrial contractions (PACs)

PACs are a common kind of arrhythmia characterized by early (premature) ectopic beats originating in the atria, which may be seen in patients with heart and chronic lung diseases, sepsis, shock, use of volatile agents, sympathetic stimulation, and excessive alcohol, nicotine, or caffeine. PAC is usually hemodynamically insignificant and self-limiting. But when they are in excess or compromise the cardiovascular function, β-blockers, digitalis or calcium channel blockers can be used after excluding the underlying causes.

6.5.1.2 Paroxysmal supraventricular tachycardia (PSVT)

PSVT is due to the rapid electrical discharge from an ectopic atrial focus, causing regular and consecutive atrial extrasystoles or may be caused by reentry in the AV node by the accessory pathway (AVNRT). It occurs most commonly in normal individuals, who may show no clinical evidence of heart disease. Less common causes of PSVT are rheumatic valvular heart disease, pulmonary embolism, cardiac surgery, thyrotoxicosis, and coronary artery disease. PSVT is characterized by rapid regular atrial rhythm at a rate of 160–220 beats/min, usually with a narrow QRS complex and lacking the P wave. It is typically rapid in onset and conclusion. The majority of patients who develop intraoperative PSVT maintain hemodynamic stability and do not require electrical direct current (DC) cardioversion. For this reason, heart rate control is the mainstay of the therapy that does not require immediate cardioversion [19]. PSVT can be confused with sinus tachycardia, atrial fibrillation, atrial flutter, and ventricular tachycardia. Carotid sinus massage can abruptly terminate the arrhythmia by activation of baroreceptors in the carotid sinus, resulting in increased vagal activity and transient AV nodal conduction block. This aids differentiation between PSVT, atrial flutter, and fibrillation. The Valsalva maneuver can also be used. If these vagal maneuvers are unsuccessful, then rapid intravenous adenosine in a dose of 6–12 mg is the drug of choice for terminating the re-entrant variety of PSVT arrhythmia. Adenosine slows the sinoatrial and AV nodal conduction and prolongs refractoriness, which is very effective in terminating PSVT. Its

ultrashort duration of action (10 s) and very rapid onset of action (15–30 s) make it desirable over other intravenous drugs. However, atrial flutter and atrial fibrillation do not respond to adenosine. Other intravenous drugs that are useful for terminating PSVT are verapamil, diltiazem, and β-blockers. Intravenous digoxin is not clinically useful in the acute control of PSVT because of its delayed peak effect and a narrow therapeutic index. DC cardioversion is indicated for PSVT unresponsive to drug therapy or PSVT associated hemodynamic deterioration. Radiofrequency ablation is the preferred approach for patients with persistent symptomatic re-entry PSVT.

6.5.1.3 Atrial flutter

Atrial flutter is due to electrical impulse re-entry into the atria, often giving an atrial rate of 250–350 beats/min with a ventricular rate of 150 beats/min. It is usually associated with varying degrees of AV block, manifesting 2:1–4:1 AV conduction. The rapid P waves create a classic saw tooth appearance on ECG (best seen in leads II, III, aVF, and V_1) and are called flutter waves (F waves). Normal T waves are lost in F waves. Atrial flutter often occurs in association with other arrhythmias such as AF. It usually signifies the presence of underlying severe heart disease and exacerbation of a chronic condition such as pulmonary disease, thyrotoxicosis, or after cardiac surgery. In many instances, treatment of the underlying disease process restores sinus rhythm. Intraoperative management of atrial flutter depends on the hemodynamic stability of the patient. Synchronized cardioversion using a low energy current (50–100 J) is the treatment of choice if the hemodynamic deterioration is present. If vital signs are stable, intravenous amiodarone, diltiazem or verapamil may convert the flutter to normal sinus rhythm.

6.5.1.4 Atrial fibrillation (AF)

AF is much commoner than an atrial flutter, and is one of the most common of all arrhythmias, especially in the elderly population [20, 22]. It accounts for more than 90% of supraventricular arrhythmia in the perioperative setting. AF has an irregularly irregular rhythm. The absence of P waves and variable QRS complexes on ECG is diagnostic of AF. AF is due to excessively rapid and disorganized atrial electrical activation without effective atrial contraction at a higher ventricular rate. The loss of atrial contraction may lead to a decrease in cardiac output and blood pressure that is often hemodynamically clinically significant. Other complications of AF include heart failure, pulmonary and systemic thromboembolism, and a significant risk of cerebrovascular events. Patients with ischemic heart disease, rheumatic heart disease, hypertension, thyrotoxicosis, and pneumonia are more prone to develop AF. The immediate intraoperative management of AF should begin with an assessment of hemodynamic status and correction of precipitating factors. The onset of AF or faster rates of chronic AF during the intraoperative period may be precipitated by acid-base and electrolyte abnormalities, hypovolemia, myocardial ischemia, sepsis, and surgical manipulation in the thoracic cavity. The goal of management is directed towards the control of ventricular response rate with pharmacological agents that slow AV node conduction. IV β-blockers or calcium channel blockers produce rapid control of rate. In the acute setting, the usefulness of digoxin is limited due to slow onset and low efficacy in high adrenergic states such as surgery. Amiodarone is a good choice for rate and rhythm control in patients with AF in the operating room. This agent also suppresses atrial ectopy and thus, recurrent AF and improves the success rate of electrical cardioversion. In cardiovascular compromised patients, synchronized DC cardioversion at 100–200 J

(biphasic) is the most reliable method of converting AF to sinus rhythm. However, it should not be used in AF of more than 48-h duration without at least 3 weeks of anticoagulation, attempts to restore sinus rhythm may increase the risk of atrial blood clot formation and systemic thromboembolism.

6.5.2 Ventricular arrhythmias

Ventricular arrhythmias during anesthesia are more common in patients with underlying cardiac disease. Their occurrence must be considered life-threatening.

6.5.2.1 Ventricular extrasystole or premature ventricular contractions (PVCs)

PVCs are commonly seen during anesthesia and can be caused by multiple factors such as electrolyte and acid-base disorders, hypoxia, hypercarbia, hypothermia, anesthetic agents, sympathomimetic drugs, and very commonly direct laryngoscopy and tracheal intubation. They are also frequently observed during cardiac and thoracic surgical procedures. PVCs are ectopic beats arising from below the AV node and give rise to a wide and bizarre QRS complex. PVCs can be unifocal, multifocal, or they can alternate with sinus beats in every second (bigeminy) or every third (trigeminy) beat pattern. The management of PVCs should focus on the correction of underlying problems. Asymptomatic or healthy patients generally do not require any treatment. Frequent PVCs, multifocal PVCs, and PVCs occurring on the T wave should be considered a potentially serious event as they can precede runs of life-threatening ventricular tachycardia or fibrillation and require prompt treatment. The immediate availability of a defibrillator is paramount in the event of a deterioration in the rhythm. Lidocaine is the drug of choice. Amiodarone is also helpful. Propanolol, procainamide, and quinidine are other drugs that can be given to abolish PVCs. However, these anti-arrhythmic drugs (classes I and III) may have proarrhythmic effects, particularly in patients with underlying heart disease [23]. Early and continuous vigilance is necessary throughout therapy. It is important to ensure that serum electrolytes (especially potassium) are kept well within the normal range. Cardiac function should be optimized and cardiac ischemia should be managed aggressively. The drugs should be prescribed only if the overall effect is clearly beneficial. Furthermore, the Cardiac Arrhythmia Suppression Trial (CAST) shows that proarrhythmic death can occur even when PVCs are apparently eliminated. Occasionally, PVCs are induced when there is severe bradycardia. Atropine, isoproterenol, or pacing may be effective to abolish the PVCs by speeding up the SA node.

6.5.2.2 Ventricular tachycardia (VT)

VT is a severe, potentially life-threatening arrhythmia as the rhythm can degenerate into ventricular fibrillation, requiring emergent treatment. The ECG shows a rapid ventricular rhythm with broad abnormal QRS complexes. The ratio of P and QRS has no fixed relationship because of atrioventricular dissociation. Like other forms of arrhythmias, the correction of precipitating factors assumes great importance. It can be categorized into non-sustained and sustained ventricular tachycardia [24].

Non-sustained VT (NSVT) is defined as 3 or more PVCs that occur at a rate of more than 120 beats/min and lasting less than 30s without hemodynamic compromise. These arrhythmias are routinely seen in the absence of cardiac disease and may not require drug therapy. However, NSVT should be monitored carefully, as it can generate into a non-perfusing rhythm.

Sustained VT presents with a broad QRS complex that may be monomorphic or polymorphic. Timely termination of VT is desirable even if it is well-tolerated. Amiodarone is the first-line recommended therapy for patients with VT. The alternative pharmacological therapy includes lidocaine and procainamide. Patients may show signs of inadequate perfusion with or without a pulse. Pulseless VT should be treated immediately with defibrillation and initiation of cardiopulmonary resuscitation according to Advanced Cardiac Life Support (ACLS) algorithm, whereas VT with a pulse should be treated with synchronized cardioversion.

6.5.2.3 Ventricular fibrillation (VF)

This arrhythmia is characterized by very rapid, chaotic, grossly irregular, and disorganized broad complexes on the ECG with no mechanical effect, resulting either from rapid discharges of impulses from one or more ventricular foci or from multiple wandering re-enters circuits in the ventricles. On the ECG, the QRS is absent. It is a serious, life-threatening rhythm due to lack or no cardiac output during the arrhythmia. Clinically, pulses will be impalpable and there will be an acute drop in oxygen saturation on pulse oximetry.

VF during anesthesia and surgery is a critical event. The common causes are myocardial ischemia, hypoxia, hypothermia, metabolic electrolyte imbalance, and drug effects. Management includes prompt initiation of cardiopulmonary resuscitation. External defibrillation is the only effective method to convert VF to a viable rhythm. The most important factor affecting survival in patients experiencing VF is time to defibrillation. Survival is best if defibrillation occurs within 3–5 min of cardiac arrest.

As with any pulseless arrest, contributing factors must be investigated and addressed. When VF is refractory to electrical treatment, IV administration of adrenaline 1 mg or amiodarone 150–300 mg may improve the response to electrical defibrillation. Adjunctive therapy with amiodarone, lidocaine, or magnesium may be indicated. A precordial thump is occasionally effective in the termination of VF but should only be attempted if a defibrillator is not immediately available [25]. Standard ACLS algorithms should be followed for electrical, pharmacological, and adjunct therapy.

6.5.2.4 Torsades de pointes (TdP)

It is an atypical polymorphic form of VT characterized by a constantly changing/twisting QRS axis around the baseline. A non-uniform delay in repolarization is the underlying electrophysiological derangement, manifested as prolonged QT interval on ECG. Tdp is usually short in duration and spontaneously reverts to sinus rhythm. However, rapidly recurring episodes may degenerate into VF and cardiac arrest [26]. The management of Tdp depends on hemodynamic stability and is initially aimed at correction of the precipitating factors and use of intravenous magnesium as cellular membrane stabilizer:

1. Single episode and hemodynamically stable: intravenous magnesium sulfate is the first-line therapy and helps to prevent recurrent arrhythmias.

2. Multiple self-terminating episodes and hemodynamically stable: intravenous magnesium and consider temporary transvenous overdrive atrial pacing and/or intravenous isoproterenol infusion, to reduce the RR interval and repolarization time.

3. Hemodynamic instability: prompt synchronized electrical cardioversion, and intravenous magnesium.

4. Pulseless arrhythmia: Follow VF treatment approach. Intravenous magnesium should be administered. Avoid amiodarone since it has a proarrhythmic effect because of the additional prolongation of the QTc interval but administer intravenous lidocaine instead.

Lidocaine is the preferred antiarrhythmic drug for TdP, though there is a lack of evidence to support its use. Other antiarrhythmic drugs such as amiodarone, procainamide, beta-blockers further prolong the QT interval and therefore worsen the condition. β -blockers will slow down the heart rate, increasing the risk of TdP.

6.5.2.5 Pulseless electrical activity (PEA)

PEA, previously known as electromechanical dissociation, is a life-threatening, non-shockable cardiac rhythm. It occurs when the electrical activity of the heart persists but does not usually follow sufficient ventricular response to produce a sufficient cardiac output to generate a pulse and supply blood to the organs in the body. While the absence of a pulse confirms a clinical diagnosis of cardiac arrest, PEA can only be differentiated from other causes of cardiac arrest by ECG. This means that PEA includes any pulseless waveform except for VF, VT, or asystole. PEA is often caused by a profound cardiovascular insult which weakens the cardiac contraction and is usually exacerbated by worsening acidosis, hypoxia, and increasing vagal tone (**Table 6**). Further compromise of the inotropic state of the cardiac muscle leads to inadequate mechanical activity, despite the presence of electrical activity and ultimately causing degeneration of the rhythm and death of the patient. Overall, the prognosis of PEA patients is poor and still shows a high mortality rate despite optimum CPR.

Prompt and good quality CPR according to ACLS guidelines to maintain cardiac output until the PEA can be corrected is the first step in the management of PEA, while potential underlying causes are identified and addressed [27]. Once a diagnosis is made, specific therapy to treat the cause should be commenced immediately. This process may involve the decompression of pneumothorax, pericardial drain for tamponade, fluids infusion for hypovolemia, correction of body temperature for hypothermia, administration of thrombolytics pulmonary embolism, and early coronary angiography with percutaneous coronary intervention (PCI) in patients with myocardial infarction. Where it is not possible to determine and/or reverse the underlying cause of PEA, the treatment of PEA is similar to that of asystole. The mainstay of drug therapy for PEA arrest is intravenous adrenaline 1 mg every 3–5 min. The routine use of sodium bicarbonate is not recommended, except in special situations (e.g., severe metabolic acidosis or hyperkalemia). Atropine is generally no longer recommended for PEA as it has not been shown to have a therapeutic benefit. Defibrillators cannot be used to correct this rhythm, as the problem lies in the response of the myocardial tissue to electrical impulses. Although PEA

4Hs	**4Ts**
Hypoxia	Toxicity
Hypovolemia	Tamponade (cardiac)
Hypothermia/hyperthermia	Tension pneumothorax
Hypokalemia/hyperkalemia (electrolyte disorders) and hydrogen ions (acidosis)	Thromboembolism (coronary or pulmonary)

Table 6.
Factors contributing to the etiology of PEA that is widely thought as 4Hs and 4Ts include as following.

and asystole are often considered fatal arrhythmias, PEA has a slightly better outcome than asystole. Previous data by the National Registry of Cardiopulmonary Resuscitation in 2003 revealed that 10% of hospital patients whose initial rhythm is PEA survive with good neurological outcomes [28].

7. Post resuscitation care

Resuscitation of an intraoperative cardiac arrest victim does not end with ROSC and must be tailored to the needs of the individual patient.

Following ROSC after cardiac arrest, many patients suffer from post-cardiac arrest syndrome, which is a high inflammatory state characterized by brain injury, myocardial dysfunction, systemic ischemia and reperfusion injury, and persistent precipitating pathology [29]. The severity of this syndrome varies according to the duration and cause of cardiac arrest. Management of these patients is challenging and requires a structured approach including restoration of adequate hemodynamics and organ perfusion, optimizing ventilation, treatment of electrolyte abnormalities, glycemic control targeted temperature management, and multimodal prognostication to improve outcomes. Specific therapy is determined by the etiology of arrest and initiating treatment to prevent recurrence [30].

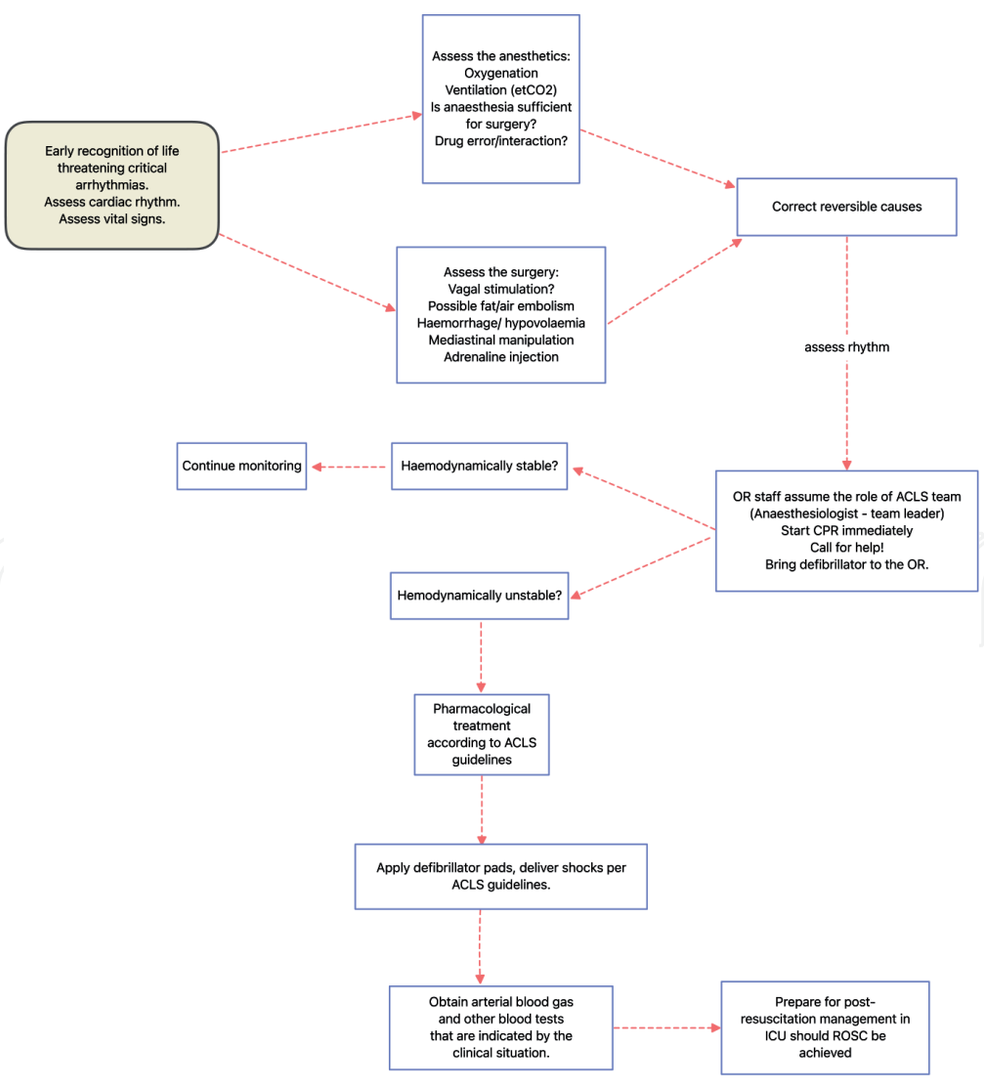

Figure 4.
Summary of management of critical arrhythmias at any time in the operating room [26, 29]. CPR: cardiopulmonary resuscitation; OR: operating room; ROSC: return of spontaneous circulation.

The flow diagram is designed as a step-by-step guide to critical arrhythmias management in the operating room, as shown in **Figure 4** [26, 31].

8. Conclusions

Cardiac arrhythmias can occur in all stages of anesthesia and surgical procedures. They are relatively frequent and continue to be an important source of morbidity and mortality among surgical patients. Most arrhythmias are benign, but some can progress to malignant arrhythmias and necessitate an urgent response. A thorough understanding of the arrhythmias, timely diagnosis as well as performing an early intervention with appropriate therapy enables a proactive approach to patient management and is life-saving for patients.

Conflict of interest

The authors declare no conflict of interest.

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