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Chapter 6

Arrhythmias Post Coronary Artery Bypass Surgery

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Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.70423

Abstract

Arrhythmias are common after cardiac surgery such as coronary artery bypass grafting surgery. Although most of these arrhythmias are transient and have a benign course, it may represent a significant source of morbidity and mortality. Postoperative arrhythmias (POAs) include atrial tachyarrhythmias, ventricular arrhythmias, and bradyarrhythmias. The incidence of POAs has not changed despite improvements in anesthetic and surgical techniques. The tachyarrhythmias in the postoperative period include atrial fibrillation, atrial flutter, supraventricular tachycardia, and ventricular tachycardia. The clinical significance of each arrhythmia depends on several factors that include cardiac function, patient's comorbidities, arrhythmia duration, and ventricular response rate. Tachycardia with uncontrolled ventricular rates can cause diastolic and later on systolic dysfunction, reduce cardiac output, and result in hypotension or myocardial ischemia. In the other hand, bradyarrhythmias may have a remarkable influence on patients with systolic or diastolic ventricular dysfunction. Arrhythmia management starts preoperatively with optimizing the patient's condition and controlling patient's risk factors, intraoperatively with careful attention to hemodynamic changes during surgery and uses appropriate anesthesia, and postoperatively with correction of temporary and correctable predisposing factors, as well as specific therapy for the arrhythmia itself. The POAs treatment urgency and management options are determined by the clinical presentation of the arrhythmia.

Keywords: coronary artery bypass surgery, arrhythmias, atrial fibrillation, ventricular arrhythmias, bradyarrhythmias

1. Introduction

Arrhythmias are common after cardiac surgery such as coronary artery bypass grafting (CABG) surgery and represent a significant source of morbidity and mortality. Although most of these arrhythmias are transient and have a benign course, it may prolong intensive care

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and hospital stay, and in rare instances, it may lead to mortality. Postoperative arrhythmias (POAs) include atrial tachyarrhythmias (ATs) and to a lesser extent ventricular arrhythmias (VAs) and bradyarrhythmias [1]. The incidence of POAs has not changed despite improvements in anesthetic and surgical techniques, and evidence suggests that its incidence may be increasing [2].

The clinical significance of each arrhythmia depends on several factors that include underlying cardiac function, patient's comorbidities, arrhythmia duration, and ventricular response rate. So, POAs could be tolerated in some patients and a source of morbidity and mortality in others, depending on the interaction between these factors [1, 3]. Rapid ventricular rates with tachycardia can cause diastolic and later on systolic dysfunction, reduce cardiac output, and result in hypotension or myocardial ischemia [4, 5]. Bradydysrhythmias, particularly with the loss of atrial function, may have a remarkable influence on patients with systolic or diastolic ventricular dysfunction [6].

Arrhythmia management starts preoperatively with optimization of the patient's condition and controlling patient's risk factors. Intraoperatively, it includes careful attention to hemodynamic changes during surgery and uses appropriate anesthesia. Postoperatively, it includes correction of temporary and correctable predisposing factors, as well as specific therapy for the arrhythmia itself [7]. The POAs treatment urgency and management options are determined by the clinical presentation of the arrhythmia [7]. Self-terminating arrhythmias without overt cardiac disease often need no therapy. However, arrhythmias with hemodynamic instability, especially in patients with critical stress conditions like systemic infections or persistent pericardial effusion need urgent intervention to restore a stable clinical status [7].

The aim of this chapter is to review post-CABG arrhythmias pathophysiology and management.

2. Pathophysiology

The primary function of CABG is to reestablish perfusion to ischemic myocardium with utilizing autologous arteries and veins. This may be achieved by using different surgical techniques. The POAs pathophysiology, incidence, and clinical course may vary depending on the surgical techniques used. Initially, cardiac surgeries were performed on a beating heart, but with the development of cardiopulmonary bypass (CPB) machine and cardioplegia, most CABG surgeries were performed on a pump. However, interest in off-pump coronary artery bypass (OPCAB) surgery had revived in the 1990s [8]. Reported potential benefits of OPCAB include lower end-organ damage with less cerebrovascular accidents (CVA), fewer cognitive deficits, renal failure, less psychomotor defects, reduced systemic inflammation, and lower transfusion rates [9]. However, variable outcomes have been reported in studies comparing these strategies [9]. Minimally invasive surgery without use of CPB and through smaller incisions- and robotic-assisted approaches have also been developed [9]. This method is most often used for left internal mammary artery (LIMA) to left anterior descending artery (LAD) grafts. Additional benefits may also include reduced operative time, reduced recovery time,

decreased the need for blood transfusion, less time under anesthesia, reduced duration of ICU stay, less pain, and an estimated 40% savings over conventional CABG [10].

The development of POAs is related to factors that influence the atrial and ventricular myocardium. These primarily include: a previous *anatomic substrate*, caused by degenerative changes typical of age and underlying disease, and *electrical substrate* derived from the perioperative processes that alter the membrane potentials, increase the dispersion of the refractory periods, and decrease the conduction velocity [11]. Inflammation and hyperadrenergic state appear to play a fundamental role in the development of postoperative tachyarrhythmias, favoring automatism [11]. Hypokalemia and hypomagnesemia characteristic of this period alter phase III of the membrane action potential, increasing the automatism, and slowing the conduction speed [11]. Atrial and ventricular ischemia due to hypoxemia is another contributing factor [11].

Several perioperative risk factors have been implicated in atrial and ventricular susceptibility to POAs, but their relative role is still uncertain. Risk factors for POAs may be classified into patient- and surgery-related factors [7].

2.1. Patient-related risk factors

Various patient-related risk factors have been described to cause POAs. These include:

2.1.1. Age

Increasing age is associated with age-related structural and electrophysiological changes that may lead to postoperative atrial tachyarrhythmias in the elderly. Old age has been demonstrated to be correlated with the development of POAs [2, 3, 12–14].

2.1.2. Underlying structural heart disease

Patients with underlying structural heart disease are at higher risk of developing POAs compared to patients with a normal heart. Structural heart disease in the atria and ventricles provides a substrate for arrhythmia via abnormal automaticity, triggered activity, or reentry. Cardiac surgery patients often have the substrate of atrial enlargement and elevation of atrial pressures may function as a substrate for atrial arrhythmias. It is well known that large atrial size and fibrosis supports propagation of atrial reentrant circuits and helps in maintaining atrial fibrillation (AF). Similarly, patients with ventricular dysfunction, ventricular dilation, or fibrosis are at higher risk of having ventricular arrhythmias [4]. Other important risk factors for POAs include previous history of arrhythmias (e.g., AF), cardiac surgery, and POAs. Also, severe right coronary artery stenosis [15], sinus nodal or atrioventricular nodal branch disease [13, 16, 17], and mitral valvular disease (particularly rheumatic mitral stenosis) have been reported as risk factors for POAs. The preoperative brain natriuretic peptide plasma concentration is another predictor of POAs [18].

2.1.3. Other comorbidities

Noncardiac comorbidities have been reported to increase the risk of POAs especially AF. These include obesity [19], previous stroke, and history of chronic obstructive pulmonary disease [20].

2.2. Surgery-related risk factors

Cardiac surgery may lead to POAs via multiple surgery-related mechanisms and risk factors that include:

2.2.1. Trauma and inflammation

Cardiac surgery provokes a vigorous inflammatory response due to a variety of metabolic, endocrine, and immune changes known as the "stress response," which has important clinical implications [21, 22] (**Figure 1**). Surgical trauma, blood loss or transfusion, hypothermia, and CPB are nonspecific activators of the inflammatory response [18, 19]. Surgical trauma may contribute to a higher degree of the inflammatory response compared to CPB [23]. These effects predispose to atrial and ventricular arrhythmias in the early postoperative period. Inflammatory mechanisms have been proposed for the development of postoperative AF (POAF) as its incidence peaks at early postoperative days. Inflammation may be related to the development of clinically aberrant or silent pericarditis. Unfortunately, clinical criteria, such as fever, pleuritic chest pain, pericardial rubs, and electrocardiogram changes correlate poorly with postoperative pericarditis and supraventricular arrhythmias [7]. However, patients with pericardial effusion in one study had a higher incidence of supraventricular arrhythmias (63% compared with 11% in patients without effusions) [24].

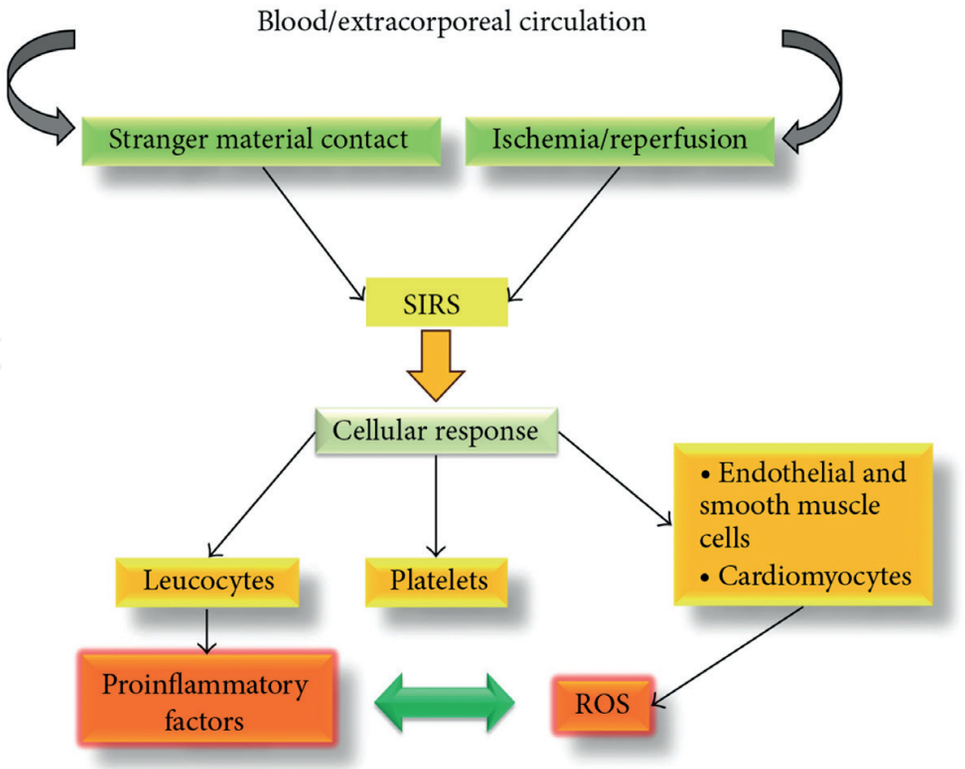


Figure 1. Pathophysiologic changes in response to cardiopulmonary bypass and the extracorporeal circulation. ROS, reactive oxygen species; SIRS, systemic inflammatory response syndrome (from Ref. [22]).

2.2.2. Hemodynamic stress

Atrial and ventricular hemodynamic changes during CABG predispose to POAs. The risk factors for POAs include atrial changes at the time of cardiac surgery, such as acute atrial trauma from cannulation, enlargement, hypertension, and ischemia [7]. Postoperative pulmonary edema and postoperative pleural effusion requiring thoracentesis have also been described as possible risk factors [25]. Hemodynamic changes might trigger focal arrhythmias [7]. It is possible that atrial stretch, hypertension, pressure and volume shifts, and heightened catecholamine states can trigger AF foci from the pulmonary veins [26].

2.2.3. Ischemic injury

The coronary blood flow is interrupted during CABG surgery and CPB, and the heart is put under circulatory arrest. This interruption of coronary blood flow causes ischemia-reperfusion injury that is exacerbated by adverse neutrophil-mediated myocardial inflammation and injury [27–29]. Atrial and ventricular ischemia or infarction triggers POAs [30]. Myocardial focal ischemia may occur due to endogenous or exogenous catecholamines, hypoxemia, hypercarbia, acid-base imbalances, drug effects, and mechanical factors. CPB, cross-clamp times, type of cardioplegia, and CABG surgical technique are also critical in determining ischemic injury. The incidence of AF has been demonstrated to be lower after OPCAB than conventional CABG. OPCAB is also associated with a lesser degree of inflammation [21].

2.2.4. Perioperative drugs

Beta-blocker withdrawal has been associated with an increased rate of postoperative supraventricular arrhythmias [31]. In contrary, some studies showed that preoperative digoxin use is a risk factor for POAs [2, 32], but not in the others [33]. Intravenous inotropic agents may be associated with POAs in some patients. The reported primary arrhythmias are sinus tachycardia (ST) and premature ventricular beats (PVCs), although other supraventricular (SVT) or ventricular arrhythmias (VT) have been reported. Clinically significant proarrhythmic effects with these agents appear to occur rarely. At conventional doses, intravenous inotropic agents are relatively safe concerning proarrhythmic effects. Inotropic agents increase sinoatrial node automaticity and decrease atrioventricular (AV) nodal conduction time [34, 35]. Dobutamine use has been reported to induce ventricular ectopic activity in 3–15% of patients [34]. Dopamine is more likely to be associated with a dose-related ST or AF [34]. Finally, short-term intravenous administration of the phosphodiesterase inhibitors amrinone and milrinone has been reported to cause PVCs and short runs of VT in up to 17% of patients [34]. Amiodarone and sotalol are useful and can be considered appropriate alternatives in high-risk patients [36]. Patients who need urgent CABG may benefit from intravenous and oral amiodarone combination in addition to beta-blockers. Although corticosteroids are associated with risk, it may be considered in selected CABG patients [36].

2.2.5. Electrolytes disorders

Hypokalemia leads to alteration of the electrophysiologic properties of cardiac myocytes with an increase in the action potential duration (increase in phase-3 depolarization), enhanced

automaticity (increased slope of diastolic depolarization), and decreased conduction velocity [37]. These changes may provoke POAs [37]. Preoperative serum potassium levels of <3.5 mmol/L have a significant association with perioperative arrhythmias in patients undergoing elective CABG surgery [37].This association might be particularly evident in the atria, where changes in inward-rectifier potassium currents are supposed to act as profibrillatory mechanisms [38]. However, hypokalemia is more likely to be associated with VAs [38]. Moreover, it is worth noting that arrhythmogenesis is often multifactorial. Catecholamine release increases cellular potassium uptake and thus decreases serum potassium levels [39]. Serum potassium levels greater than 5.5 mmol/L appear to be associated with the development of POAF and atrial flutter (AFL) [37]. The role of magnesium remains controversial. The low serum magnesium levels—which is frequently seen after cardiac surgery—correlate with an increased incidence of POAs [7]. However, magnesium supplementation has produced conflicting results. Magnesium supplementation should be considered in all patients with hypomagnesemia [40–41].

2.2.6. Other factors

The human epicardial fat pads (FPs) contain parasympathetic ganglia [42]. There are two posterior FPs with the first one located in the superior vena caval-atrial junction and contains postganglionic fibers that lead to the sinoatrial (SA) node. The second FP is located at the pulmonary vein-left atrium and contains postganglionic fibers that lead to the atrioventricular (AV) node [43–45]. The anterior epicardial FP located in the aortopulmonary window that is routinely dissected and removed in CABG because it is located where the aortic cross-clamp is typically placed. Preservation of the human anterior epicardial FP during CABG decreases the incidence of POAF in one study [46], but not in another more recent study [47].

3. Postoperative atrial tachyarrhythmias (POATs)

3.1. Postoperative atrial fibrillation (POAF)

3.1.1. Epidemiology

AF is the most common complication seen after CABG surgery. The incidence of POAF is approximately 30% after isolated CABG, 40% after valve replacements or repair, and about 50% after combined CABG and valve surgeries [2, 48–51]. The incidence of POAF increases with older age [2, 52, 53].

3.1.2. Diagnosis

The diagnosis of POAF is confirmed based on the telemetry and 12-lead electrocardiogram (ECG) recordings with an abrupt change in heart rate and rhythm, and loss of P waves [16, 54]. Atrial electrograms obtained from temporary atrial epicardial pacing wires that are often routinely placed at the time of cardiac surgery can be helpful in confirming the diagnosis of AF, AFL, and other forms of supraventricular tachycardia (SVTs) [54].

3.1.3. Clinical course

POAF usually occurs within 2–4 days after cardiac surgery, with a peak incidence on the second postoperative day [12, 55]. In POAF patients without a prior history of atrial arrhythmias, AF is usually self-limited. About 15–30% of POAF convert to sinus rhythm within 2 h and up to 80% within 24 h [56, 57]. The mean duration of AF in one report was 11–12 h [57], and >90% of the patients were in sinus rhythm 6–8 weeks after surgery [57, 58]. In another study, only 2 out of 112 patients who had paroxysmal AF after CABG were still in AF at 6 weeks [59].

3.1.4. Prognosis

Although POAF is often self-limiting, its clinical effects depend on ventricular rate, ventricular function, arrhythmia duration, symptoms, hemodynamic stability, and risk of thromboembolism. [60]. POAF is associated with increased postoperative thromboembolic risk and stroke [25, 60–62]. In a series of 4507 patients, the incidence of stroke was significantly higher in those who developed POAF (3.3 versus 1.4%) [2]. Patient's underlying comorbidities, such as older age, previous cerebrovascular disease (CVA), the presence of a carotid bruit, peripheral vascular disease (PVD), and CPB time, have an important role in the development of in-hospital stroke [63–65]. In a review of 2972 patients undergoing CABG and/or valve surgery, POAF was associated with late onset stroke only if accompanied by a low cardiac output syndrome (3.9 versus 1.9%) [66]. Besides, POAF development is associated with a prolonged length of hospitalization [2, 25, 54]. The POAF is associated with an additional 2–4 days hospital stay after CABG surgery with an additional cost [54]. However, this effect seems to be less prominent with current cardiac surgical care [67]. Additionally, POAF may result in hemodynamic compromise [68], ventricular dysrhythmias [2], and iatrogenic complications associated with therapeutic interventions [53]. POAF may result in increased in-hospital and long-term mortality in a subset of patients [3, 60]. In a retrospective study of 6475 patients undergoing CABG at a single institution: 994 patients (15%) developed POAF. Higher in-hospital (7.4 versus 3.4%) and 4-year mortality (26 versus 13%) was noted in POAF patients but also with more comorbidities (i.e., older age, hypertension, and left ventricular hypertrophy) [60].

3.1.5. POAF management

The management of POAF should include the strategy for prevention and treatment of POAF when it develops. PAOF management starts with the optimization of medical comorbidities, if possible (e.g., hypoxia), and the correction of underlying electrolyte disturbances (e.g., potassium and magnesium abnormalities) [53]. POAF is treated similarly to AF in nonsurgical patients by rhythm control via pharmacological or electrical approach or heart rate control, and appropriate antithrombotic therapy.

3.1.5.1. Rhythm control versus heart rate control

In the atrial fibrillation follow-up investigation of rhythm management (AFFIRM) trial, the rate control versus rhythm control in nonsurgical patients with AF was studied and found that the use of rhythm control had no survival advantage, and it was associated with more frequent hospitalizations and adverse drug effects [69]. However, some studies involving

patients with AF after cardiac surgery have suggested that rhythm control may offer advantages over rate control. This is still controversial and the evidence is inconclusive [11, 70–72].

Treatment strategies of POAF aim to reduce symptoms, limit adverse hemodynamic effects, decrease the length of hospital stay, prevent readmissions, and improve survival [73]. The rhythm control strategy has the advantage of a rapid conversion to sinus rhythm, which restores atrial activity, functional capacity, and might reduce thromboembolic. The rate control strategy has the advantage of avoiding the potential adverse effects of antiarrhythmic drugs and complications associated with cardioversion [73]. In a recent trial, there was no difference in hospital admissions during a 60-day follow-up, with randomizing POAF patients to either rhythm control therapy with amiodarone or rate control [73]. As a result, the main aim of rhythm control therapy in POAF patients should be to improve AF-related symptoms. In asymptomatic patients and those with acceptable symptoms, rate control or deferred cardioversion preceded by anticoagulation is a reasonable approach [73].

In the following paragraph, rate control and rhythm control options will be discussed briefly.

(A) Rate control strategy: the rate control may be achieved by using beta-blockers, nondihydropyridine calcium channel blockers, digoxin, or a combination of these medications. Beta-blocker agents are the drug of choice, particularly for ischemic heart disease patients, because of the increased adrenergic stress in the postoperative period [53, 73, 74]. However, beta-blockers might be poorly tolerated or relatively contraindicated in patients with known bronchial asthma or bronchospastic lung diseases, active congestive heart failure, or AV conduction block [53]. The nondihydropyridine calcium channel blocker agents represent an alternative AV nodal blocking agent. Digoxin is less effective when the adrenergic tone is as high as in the postoperative period, but it may be used in patients with congestive heart failure. Amiodarone is another agent that can be used as it has been reported to be effective in controlling heart rate. Also, intravenous amiodarone administration has been associated with improved hemodynamic status [75, 76]. For further information about drugs used in AF rate control see **Table 1**.

(B) Rhythm control: the rhythm control could be archived by using a direct current cardioversion (electrical cardioversion) or antiarrhythmic drugs (pharmacological cardioversion). Electrical cardioversion is indicated on an urgent basis in hemodynamically unstable patients, acute heart failure, or myocardial ischemia. Also, it may be used electively to restore sinus rhythm when a pharmacologic attempt has failed to resume a sinus rhythm [53]. Rhythm control with antiarrhythmic medications is preferred in symptomatic patients despite rate control trial, or when the control of ventricular response is hard to achieve. Amiodarone [77–79] or vernakalant [79, 80] have been efficient in converting POAF to sinus rhythm. Other antiarrhythmic medications that may be used include procainamide [80], ibutilide [81], and sotalol [82]. With ibutilide use, electrolyte imbalance should be corrected to avoid polymorphic ventricular tachycardia [82]. For further information about drugs used in AF rhythm control see **Table 2**.

3.1.5.2. Anticoagulation

POAF is associated with poor short- and long-term outcomes, including high rates of early and late stroke, and late mortality as mentioned earlier. However, the indication and timing of anticoagulation in POAF patients should take into consideration the risk of postoperative bleeding.

Drug	Route of admintration and doses	Side effects sects	Remarks
Beta-blockers			
Atenolol	Oral 25–100 mg QD	Bradycardia, hypotension, fatigue, depression, negative inotropy, bronchospasm, AVB	Decrease dose if CrCl <35
Bisoprolol	Oral 2.5–10 mg QD	As above	Good choice for HF patients
Carvedilol	Oral 3.125–25 mg BID	As above	Good choice for HF patients
Esmolol	I.V. 500 mcg/kg bolus over 1 min, then 50–300 mcg/kg/min	As above	Only IV Higher rate of hypotension
Metoprolol tartrate	IV 2.5–5.0 mg bolus over 2 min; up to 3 doses Oral 25–100 mg BID	As above	
Metoprolol XL (succinate)	Oral 50–400 mg QD	As above	Good choice for HF patients
Nadolol	Oral 10 (usual initial adult dose 40 mg)–240 mg QD	As above	Dosage adjustments based on CrCl
Propranolol	IV 1 mg over 1 min, up to 3 doses at 2-min intervals Oral 10–40 mg up to 160–320 mg/day divided in BID to QID doses 80–160 to 320 mg QD (ER)	As above	
Calcium channel blockers			
Diltiazem	IV 0.25 mg/kg bolus over 5 min followed by 0.05–0.15 mg/kg/h continuous infusion Oral 30 mg TID/QID up to 480 mg/day 120–360 to 480 mg Q D (ER)	Bradycardia, hypotension, ankle swelling, exacerbation of HF, AVB	Do not use in HF Drug interaction via CYP3A4 including digoxin and warfarin
Verapamil	IV 5–10 mg (0.075–0.15 mg/kg) over bolus at least 2 min; may give an additional 10 mg (0.15 mg/kg) after 30 min if no response, then 0.005 mg/kg/min infusion Oral 80–120 mg TID up to 480 mg/day 180–480 mg QD or 240 BID (ER)	Bradycardia, AVB, hypotension, constipation, exacerbation of HF	As diltiazem
Others			
Digoxin	IV 0.25 mg IV with repeat dosing to a maximum of 1.5 mg over 24 h Oral 0.125–0.25 mg QD	Bradycardia, AVB, nausea, vomiting, visual disturbance	Narrow therapeutic window Adjust for renal failure Drug interactions via p-glycoprotein
Amiodarone	IV 150 mg over 10 min, followed by 1 mg/min continuous IV infusion for 6 h, then 0.5 mg/min continuous infusion for 18 h Oral 400–800 mg/day PO in divided doses for 2–4 weeks to a total load of up to 10 g, then 100–200 mg QD	Bradycardia, hypotension, AV block, QTc prolongation, phlebitis on chronic use: Ocular, pulmonary, hepatic, hematological, neurological complications	Monitor thyroid, liver and lung functions

AVB, atrioventricular block; CrCl, creatinine clearance; BID, twice daily; h, hour; ER, extended release; HF, heart failure; IV, intravenous; mg milligram; min, minute; QD, once daily; QID, 4 times a day; QTc, correct QT interval; TID, 3 times a day.

Table 1. Medications commonly used for atrial fibrillation **rate control** with its dosage and common possible side effects.

Drug	Usual doses	Side effects sects	Additional information
Vaughan Williams class IA			
Disopyramide	**Oral** IR 100–200 mg q 6 h ER 200–400 mg q 12 h	HF Prolonged QT interval Prostatism Glaucoma	Metabolized by CYP3A4: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., phenytoin, phenobarbital, rifampin) Avoid other QT interval prolonging drugs
Procainamide	**IV** 15–17 mg/kg infused at a rate of 20–30 mg/min or alternatively100 mg IV every 5 min, max. 1 g MD 1–4 mg/min	HF Prolonged QT interval May cause hypotension, myopathies, blood dyscrasias, and SLE-like syndrome	Drug of choice for WPW with AF Avoid other QT interval prolonging drugs Adjust for renal failure
Quinidine	**Oral** IR 200–300 mg q 6–8 h up to 600 mg q 6 h ER 324 mg–648 mg q 8–12 h	Prolonged QT interval Diarrhea Bradycardia, AV block, bundle-branch block, digitalis toxicity	Inhibits CYP2D6: ⇧ concentrations of metoprolol, tricyclic antidepressants, antipsychotics; ⇩ efficacy of codeine Inhibits P-glycoprotein:⇧ digoxin concentration
Vaughan Williams class IC			
Flecainide	**IV** 1.5–3 mg/kg over 10-20 min **Oral** LD 200 (wt < 70 kg)–300 mg (wt > 70 kg), MD 50–200 mg BID max. 400 mg/day	Sinus or AV node dysfunction HF CAD Atrial flutter Brugada syndrome Renal or liver disease May cause blurred vision	Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7–10% of population) and renal excretion (dual impairment can ⇧⇧ plasma concentration) Decrease dose if CrCl < 35
Propafenone	**IV** 1.5–2 mg/kg over 10–20 min **Oral** IR: 150–300 mg q 8 h ER: 225–425 mg q 12 h (Oral LD 450 mg (wt < 70 kg)–600 mg (wt > 70 kg), MD 450–900 mg/d divided into q 8 h (IR), or 12 h (ER))	Sinus or AV node dysfunction or Infranodal conduction disease HF CAD Atrial flutter Brugada syndrome Liver disease Asthma may cause dysgeusia	Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7–10% of population)—poor metabolizers have ⇧beta blockade Inhibits P-glycoprotein: ⇧digoxin concentration Inhibits CYP2C9: ⇧warfarin concentration (⇧INR 25%) Decrease dose in hepatic failure
Vaughan Williams class III			
Amiodarone	**IV** LD 150 mg over 10 min; followed by 1 mg/min for 6 h; then 0.5 mg/min for 18 h or change to oral dosing; after 24 h, consider decreasing dose to 0.25 mg/min **Oral** 400–600 mg daily in divided doses for 2–4 wk; maintenance typically 100–200 mg QD	Sinus or AV node dysfunction Infranodal conduction disease Lung disease Prolonged QT interval	Inhibits most CYPs to cause drug interaction: ⇧concentrations of warfarin (⇧INR between 0–200%), statins, many other drugs Inhibits P-glycoprotein: ⇧digoxin concentration

Drug	Usual doses	Side effects sects	Additional information
Dronedarone	**Oral** 400 mg BID	Bradycardia HF Liver disease Thyriod disease pulmonary fibrosis Prolonged QT interval	Metabolized by CYP3A: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., phenytoin, phenobarbital, rifampin) Inhibits CYP3A, CYP2D6, P-glycoprotein: ⇧concentrations of some statins, digoxin, beta blockers, sirolimus, tacrolimus Avoid in long-standing persistent or permanent AF and HF
Dofetilide	**Oral** 125–500 mcg BID if CrCl >60, 250 mcg if CrCl 40–60, 125 mcg if CrCl 20–40 Decrease MD if QTc increased by >15% of >500 ms 2–3 h after dose or consider discontinuing it	Prolonged QT interval and torsades de pointes Renal disease Hypokalemia hypomagnesaemia AV block, bradycardia, sick sinus syndrome	Adjust dose for renal function, body size, and age (avoid if CrCl < 20) Drug interactions via CYP3A4: CI to use with verapamil, cimetidine, ketoconazole, trimethoprim, prochlorperazine, HCTZ, and megestrol Discontinue amiodarone at least 3 m before initiation Avoid other QT interval prolonging drugs
Ibutilide	**IV** 1 mg over 10 min; may repeat 1 mg once if necessary (if weight <60 kg, use 0.01 mg/kg)	Prolonged QT interval and torsade de pointes hypotension CAD HF	Mointor K and mg level
Sotalol	**Oral** 40–160 mg q12 h **IV** 75–150 mg QD or BID over 5 h (only if patient cannot take oral)	Prolonged QT interval Sinus or AV nodal dysfunction HF	Renal excretion: CI if Cr Cl <40 decrease dose if CrCl 40–60 Risk of torsade de pointes (do not initiate sotalol therapy if the baseline QTc is longer than 450 ms. If the QT interval prolongs to 500 ms or greater, the dose must be reduced, the duration of the infusion prolonged or the drug discontinued) Avoid other QT interval prolonging drugs correct hypokalemia/hypomagnesemia

ACC AF [74]; JACC [53]. AF, atrial fibrillation; AV, atrioventricular; BID, twice daily; CAD, coronary artery disease; CI, contraindicated; CrCl, creatinine clearance; ER, extended release; h, hour; HCTZ, hydrochlorothiazide; HF, heart failure; IL, immediate release; IV, intravenous; LD, loading dose; INR, international normalized ratio; MD, maintenance dose; min, minute; max, maximum; SLE, systemic lupus erythematosus; Q, every; QD, once daily; wt, weight. http://www.pdr.net/

Table 2. Medications commonly used for atrial fibrillation Rythm Control with its dosage and major pharmacokinetic and drug interactions.

Oral anticoagulation at discharge has been associated with a reduced long-term mortality in patients with POAF [83] but without evidence from controlled trials [75]. POAF that persists for longer than 48 h should be anticoagulated with warfarin or nonvitamin K antagonist oral anticoagulants (NOACs). The NOACs are available for the treatment of nonvalvular AF. NOACs have been found to be as efficacious or even superior to warfarin in the prevention of stroke in nonvalvular AF patients with high risk of thromboembolism, with similar to lower rates of major bleeding, and also lower rates of intracranial hemorrhage [84].

3.1.6. Prevention of POAF

Beta-blockers are effective in reducing POAF and SVTs. Propranolol uses reduced POAF incidence from 31.7% in the control group to 16.3% in the treatment group [85]. In the majority of beta-blocker studies, it is administered postoperatively [86]. Amiodarone reduced the incidence of POAF and hospital stay compared to beta-blocker therapy in several meta-analyses [86–89]. Prophylactic administration of sotalol may be considered for patients at risk of developing AF after cardiac surgery [76, 90, 91]. Also, administration of colchicine postoperatively may reduce POAF [75, 92]. Statin use preoperatively did not prevent POAF in a prospective controlled trial [93], despite that initial reports from meta-analyses were encouraging [94–96].

Other therapies for the prevention of POAF have been studied in small trials, but have not demonstrated clear beneficial effects [76]. These include angiotensin converting enzyme inhibitors (ACEIs) [97], magnesium [85, 98, 99], n-3 polyunsaturated fatty acids [100–108], corticosteroids [109–111], and posterior pericardiectomy [112]. Conflicting results have also been reported for acetylcysteine [113], and sodium nitroprusside [114].

Nonpharmacologic therapy with atrial pacing has been tested in various studies [7]. One meta-analysis showed a significant reduction in POAF with atrial pacing (OR 0.57, 95% CI 0.38–0.84) [67], and most [115–117] but not all [118, 119] published studies showed benefit with this therapy. Besides, there are conflicting findings as to the relative value of the different types of atrial pacing [115, 116].

3.2. Postoperative atrial flutter (POAFL)

Unlike POAF, POAFL after CABG is not well studied. In a single-center study with 80 consecutive patients who underwent CABG with no previous history of AFL, 16 patients (20%) had documented POAFL. Ten of these patients showed temporary AFLs that were curable without radiofrequency catheter ablation (RFCA), and 37.5% of the patients with POAFL (i.e., 7.5% of the patients after CABG) showed sustained or repeated AFL with subjective symptoms [120]. In another study that looked at ATs late after open heart surgery, it was found that cavotricuspid isthmus (CTI)-dependent AFL was the most common. Atypical AFL becomes progressively more widespread with more extensive atriotomy [121]. AFL and ATs that developed late after cardiac surgery are believed to be due to scars created by incisions applied to the right and/or left atrium either for establishing extracorporeal circulation or access to intracardiac structures (coronary sinus, interatrial or interventricular septum, atrioventricular valves, etc.) [122]. The scars created by these incisions play a significant role in the development of ATs, months or years after surgery [123, 124].

AFL in the early postoperative period is managed as POAF with rate control or rhythm control and anticoagulation based on arrhythmia duration and patient risk factors. On long-term catheter ablation of AFL is an effective, safe, and potentially curative procedure.

3.3. Supraventricular tachycardia (SVT)

3.3.1. Epidemiology

Sinus tachycardia (ST) represents an appropriate autonomic response to a physiological stress. The upper limit of normal rate for sinus tachycardia is calculated from the formula

(220 bpm minus age) [125]. Inappropriate ST may be seen in some patients, especially with young age, but it is rare and should be considered a diagnosis of exclusion [125]. The term 'SVT' refers to paroxysmal tachyarrhythmias that require atrial or AV nodal tissue, or both, for their initiation and maintenance [126]. It is typical of a sudden or paroxysmal onset and includes AV nodal reentrant tachycardias (AVNRT), AV reentrant tachycardias (AVRT), and atrial tachycardias. The overall incidence of perioperative arrhythmias in noncardiac surgery varies from 16 to 62% with intermittent ECG monitoring and up to 89% with continuous Holter monitoring [127]. It is more likely to be supraventricular than ventricular in origin [127]. In small study, the incidence of persistent SVT in noncardiac surgery patients was 2% during surgery and 6% in the postoperative period [128].

3.3.2. Diagnosis

12-lead ECG and rhythm strips during tachycardia are diagnostic and may give an impression about the most likely diagnosis. Although ST is usually easy to diagnose on 12-lead ECG, the presence of first-degree AV block, which is not uncommon after cardiac surgery, may give ECG appearance that mimics SVT due to P wave merge with T wave (P wave hidden within T wave). ECG features of ATs including SVTs are shown in **Table 3**.

3.3.3. Clinical course

ATs occur most frequently 2–3 days postsurgery and are likely related to sympathetic stimulation associated with an inflammatory response [129]. Patients with known SVT may have an exacerbation of their tachycardia in the perioperative period. However, SVT may be diagnosed for the first time in the perioperative period [2, 7, 130, 131]. SVT is often associated with a high sympathetic tone, but other precipitants may contribute to its occurrence. The clinical symptoms, time of onset, and natural course of ATs are identical in patients with cardiac, thoracic, or other surgery.

3.3.4. Prognosis

The prognosis of perioperative SVTs is good, but it may be associated with increased hospital stay [128].

3.3.5. Management

The SVT management, in general, depends on the hemodynamic status of the patient. If the patient with SVT is hemodynamically unstable, synchronized cardioversion is recommended for acute termination of the tachycardia when vagal maneuvers or adenosine is ineffective or not feasible [132]. Before initiating specific drug therapy for acute SVT in hemodynamically stable patients, it is important to assess and correct possible precipitating factors such as respiratory failure or electrolyte imbalance. SVT may respond to vagal maneuver if the patient can do it. Adenosine might be used if there is no contraindication. SVT also responds to rate control drugs such as beta-blockers (e.g., esmolol, metoprolol, bisoprolol) or nondihydropyridine calcium channel antagonists (e.g., diltiazem, verapamil). Intravenous (IV) digoxin, IV amiodarone, adenosine, IV or oral beta-blockers, diltiazem, and

verapamil are potentially harmful in acute treatment in patients with pre-excited AF (AF in patients with Wolff-Parkinson-White (WPW) syndrome) [133]. Of note, atrial tachycardia unifocal or multifocal usually respond to rate control drugs but are not amenable to direct current cardioversion. Cardiac electrophysiology study with catheter ablation is an effective long-term management for recurrent SVT.

4. Postoperative ventricular tachyarrhythmias (POVTAs)

The postoperative ventricular tachyarrhythmias (POVTAs) range from isolated PVC to VT or ventricular fibrillation (VF).

4.1. Premature ventricular complexes (PVCs)

4.1.1. Epidemiology

Isolated PVCs including nonsustained ventricular tachycardia (NSVT) are seen in about 50% of patients during and after cardiac surgery [134]. PVCs can be related to electrolyte or other metabolic imbalances [7].

4.1.2. Diagnosis

PVCs may be seen on continuous telemetric monitoring and 12-lead ECG, however, careful evaluation of the ECG tracing is needed to be distinguished from atrial ectopy with aberrant ventricular conduction [7].

4.1.3. Clinical course

Patients with postoperative PVCs may be asymptomatic or may have palpitations with a skipped beat, or dizziness. It is rarely associated with hemodynamic instability.

4.1.4. Prognosis

Patients with isolated and noncomplicated PVCs postoperatively do not exhibit increased risk of malignant VAs [135, 136]. On the contrary, frequent PVCs (>30 per hour) may reduce ventricular function and therefore have an adverse impact on the short-term outcome. There was no significant difference in mortality in patients with versus patients without frequent postoperative PVCs and NSVT (8 versus 5%), at an average follow-up of 3 years, in a study including 185 postoperative patients [137]. However, in another study of 126 patients with postoperative PVCs, it was shown that patients with left ventricular ejection fraction (LVEF) of <40% had a 75% mortality rate and 33% incidence of sudden death at an average follow-up of 15 months, whereas none of the patients with preserved left ventricular function had sudden death [135]. Thus, PVCs are not related to mortality with good LV function, and long-term outcome after cardiac surgery seems to be closely related to the left ventricular function.

4.1.5. Management

Correction of any reversible cause of ventricular arrhythmias should be performed. Hemo dynamically stable and asymptomatic PVCs do not usually need treatment with antiarrhythmic therapy on short or long-term. Lidocaine has been used with a successful result in reducing hemodynamically significant or symptomatic PVCs, but without improving mortality. Empirical use of class I antiarrhythmic drugs for suppression of frequent and/or complex PVCs had no beneficial effects on mortality rate and may be harmful as shown in several studies in another setting [138, 139]. Additionally, overdrive pacing, using either atrial or atrioventricular sequential pacing, has been used without significant results [138, 139]. Patients with asymptomatic NSVT after cardiac surgery and preserved LVEF generally have a favorable long-term prognosis and do not require invasive workup with an electrophysiology study. The use of implantable cardioverter defibrillators (ICDs) has shown no benefits in improving prognosis in this population [140].

4.2. Ventricular tachyarrhythmias

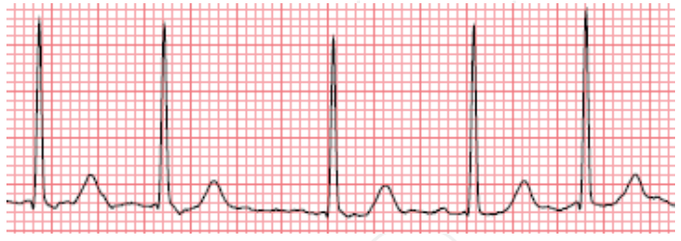
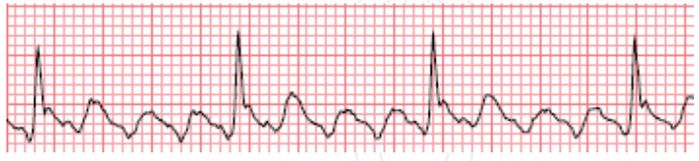
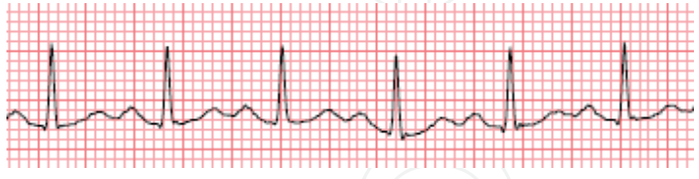
4.2.1. Epidemiology

Sustained VT and VF rarely occur after cardiac surgery with an incidence of 0.4–1.7% in most of the studies [138, 141], but an incidence of 3.1% has been reported [142]. Furthermore, it is life threatening and affects outcome [134, 143].

4.2.2. Pathophysiology and risk factors

Pathophysiology of POAs, in general, was disused in item 2. Coronary artery disease (CAD) leads to a broad spectrum of changes and may trigger arrhythmia mechanisms via enhanced automaticity, triggered activity, and reentry. While myocardial infarction (MI) related scar constitutes the clinical model of reentry [144], focal activation due to abnormal automaticity is the primary mechanism involved in the VT during acute ischemia [145]. Early and delayed after depolarization result from focal discharge by calcium overload and triggered activity is another likely mechanism of VT initiation in ischemia, but this needs to be proven experimentally thus far [146, 147]. Acute ischemia activates the adenosine triphosphate-sensitive potassium (K-ATP) channels, causing an increase in extracellular potassium along with acidosis and hypoxia in the cardiac muscle. As a result of the minor increases in extracellular potassium depolarize the myocardiocyte's resting membrane potential, which can increase tissue excitability in early phases of ischemia [145]. The mechanism underlying the VT associated with healed or healing MI is reentry in more than 95% of cases [144].

Complex ventricular arrhythmias (VAs) are associated with multiple risk factors [7]. Based on clinical studies, the conditions associated with VAs after cardiac surgery may include: increased age, female gender, presence of unstable angina, congestive cardiac failure, hemodynamic instability, preoperative use of inotropes, preoperative use of IABP, emergency surgery, electrolyte disturbances, hypoxia, hypovolemia, myocardial ischemia/infarction, acute graft closure, reperfusion after cessation of CPB, and inotropes antiarrhythmic drugs use, on-pump surgery, and PVD [134, 135, 141, 143, 148].

	Heart rate (bpm)	Regularity	Onset	Atrial activity	Response to adenosine	Rhythm strip
AF	A: 350-500 V: 100- 220	Irregular	Sudden	No discrete P-waves	Transient slowing of ventricular rate	
AFL	A: 250-350 V: 150 (slower or faster depends on AV conduction)	Regular (irregular with variable AV conduction)	Sudden	Flutter waves "saw-tooth" pattern)	Transient slowing of ventricular rate	
ST	220-age	Regular	Gradual	Normal P-waves	Transient slowing	
AT	150-250	Regular	Sudden	Abnormal P-waves morphology	-Transient slowing of ventricular rate -May terminate tachycardia in 70% of the cases	
AVNRT	150-250	Regular	Sudden	No obvious P-waves as P- wave is in or just after QRS (Pseudo r′ in V1 or S wave in inferior leads)	Termination of tachycardia	

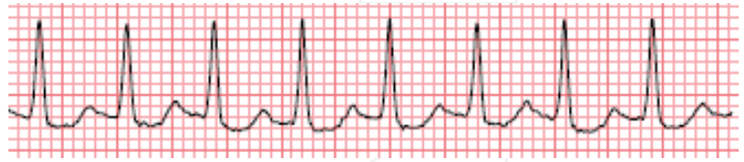
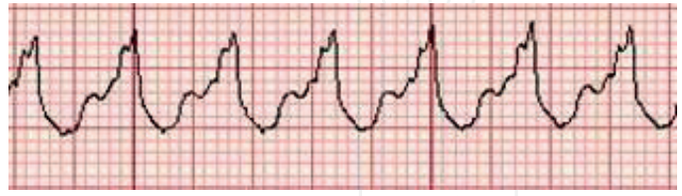
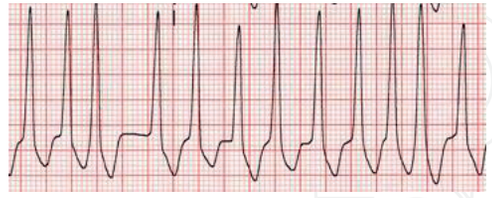
	Heart rate (bpm)	Regularity	Onset	Atrial activity	Response to adenosine	Rhythm strip
AVRT (WPW)	150-250	Regular	Sudden	**ORT:** P-waves may be buried in QRS complex, or retrograde P wave may be seen at the end of the QRS complex or in the early part of the ST segment	Termination of tachycardia	
				ART: P wave normal with short PR and delta wave (wide QRS complexes due to abnormal ventricular depolarization via accessory pathway)	Termination of tachycardia	
				AF: no P-wave, irregular rhythm, and wide QRS complex	Should not be used	

Table 3. Differential diagnosis of atrial tachyarrhythmias.

4.2.3. Diagnosis

In addition to a clinical history and a physical exam, the general evaluation of a patient with CAD and suspected or documented VAs includes performing a 12-lead ECG and an echocardiogram. Telemetry monitoring with careful evaluation of VAs initiation and termination is very helpful. Based on ECG criteria, wide complex tachycardias (WCT) may be either ventricular or SVT with aberrancy. However, in patients with structural heart disease like prior infarction, the diagnosis is mostly VT. If feasible, a 12-lead ECG and atrial electrograms through temporary epicardial wires placed at the time of cardiac surgery should be obtained. The presence of AV dissociation strongly suggests VT [138]. Although the ECG diagnosis of a WCT is challenging, it is important to remember that VT is the cause in at least 80% of cases [149].

4.2.4. Clinical course

Clinical presentation of patients post cardiac surgery with VTs is variable. The hemodynamic state of these patients depends mainly on the rate of the tachyarrhythmia and the left ventricular function. Therefore, some patients may be asymptomatic. Other patients with VT may complain of palpitations, dyspnea, or chest discomfort as their main symptoms. VTs may present with syncope and sudden cardiac death as a result of hemodynamic compromise. Incessant VT, even if it is hemodynamically stable, can lead to hemodynamic deterioration and heart failure [150, 151].

4.2.5. Prognosis

The prognosis is correlated with the type of arrhythmia and the type and degree of structural heart disease [7]. As mentioned earlier, PVCs and NSVT generally have no impact on the outcome. However, patients with sustained VAs have poorer short- and long-term prognosis.

POVAs predicts higher in-hospital mortality (21.7–31.5%) compared with (1.4–2.9%) in control [134, 143, 148]. In one study, POVAs was associated with increased long-term mortality over a mean follow-up of 3.5 years. Patients with POVAs had a high risk of death in the POVAs group during the first 6 postsurgical months (6-month survival of POVAs 59.8 versus 93.8% for POVAs free group). This difference in survival persisted over time [148].

4.2.6. Management of POVAs

Asymptomatic PVCs and hemodynamically stable short runs of NSVT do not need specific intervention, and the correction of any reversible cause of VAs is generally sufficient. Postoperative sustained VAs treatment follows the ICD indications used in other clinical settings [150, 152]. However, the postoperative patients require close attention to the identification and treatment of reversible causes of arrhythmia like electrolyte or other metabolic disturbances, myocardial ischemia, or mechanical complications of surgery [7]. Sustained VAs should be promptly cardioverted either by drugs infusion or electrically based on hemodynamic stability. Hemodynamically stable sustained VTs may be initially treated with antiarrhythmic drugs infusion.

4.2.6.1. Pharmacological treatment

It includes antiarrhythmic medication use and standard medical therapy. The most commonly used antiarrhythmic agents include:

- Amiodarone: IV amiodarone is frequently used as a first-line treatment for VAs as it is better tolerated in patients with low ejection fraction than the other antiarrhythmic drugs. The recommended starting dose of Cordarone I.V. is 1000 mg over the first 24 h of therapy. It is usually delivered by bolus infusion of 150 mg over 10–15 min, followed by 1 mg/min for 6 h, then 0.5 mg/min infusion for 18 h. The alternative dose would be 300 mg over 1 h then infusion at 50 mg/h. Additional 150 mg blouses may be given but frequent boluses during the first 24 h should be limited due to the risk of hepatic toxicity [153].

- Lidocaine: it is generally a good choice if ischemia is suspected. Lidocaine is administered as a bolus of 0.75–1.5 mg/kg, followed by an IV infusion of 1–4 mg/min (the maximal dose is 3 mg/kg/h). In elderly patient and patients with congestive heart failure or hepatic dysfunction, the lidocaine dose should be reduced [153].

- Procainamide: it is often a second line drug, and it is given as loading dose of 15–18 mg/kg administered as a slow infusion over 25–30 min or 100 mg/dose. The infusion rate should not exceed 50 mg/min. The loading dose may be repeated every 5 min as needed to a total dose of 1 g. However, it should be stopped if hypotension occurs, or QRS complex widens by 50% of its original width. This is followed by a maintenance dose of 1–4 mg/min by continuous infusion. The procainamide maintenance infusion should be reduced by 1/3 in patients with moderate renal or cardiac impairment and by 2/3 in patients with severe renal or cardiac impairment [153].

Standard medical therapy includes beta-blockers, and ACEIs drugs have been demonstrated to improve long-term survival particularly in patients with left ventricular dysfunction.

4.2.6.2. Nonpharmacological management

- Overdrive pacing: in patients with slower VTs who have ventricular epicardial wires, overdrive pacing may be performed. Electrical cardioversion/defibrillation should be easily available because of the possibility of acceleration of the VT or degeneration to VF [154].

- Electrical cardioversion/defibrillation: in patients with cardiac arrest, basic life support (BLS) and advanced cardiovascular life support (ACLS) should be followed. Electrical defibrillation should be performed for VF, pulseless VT, and hemodynamically unstable VT. Electrical cardioversion may be used for stable sustained VT as the first choice or for those who do not respond to antiarrhythmic medications. The recommended energy with current biphasic defibrillators ranges from 150 to 200 Joules. Sedation with short-acting agents should precede cardioversion in awake patients [154].

- Emergency mechanical support: in postoperative patients who are not responding to standard resuscitation maneuvers, initiation of emergency CPB in the intensive care unit may be considered. In one study, CPB use in a postoperative cardiac arrest was associated with a 56% long-term survival rate with a 22% incidence of soft tissue infections and no mediastinitis [154].

- Implantable cardioverter-defibrillator (ICD) therapy:

In the absence of a reversible cause of sustained VT or cardiac arrest after CABG, long-term management may include electrophysiology study and eventually an ICD implantation. Patients with NSVT, prior MI, and left ventricular dysfunction (LVEF <40%) may be considered for electrophysiology testing and implantation of an ICD if a sustained ventricular arrhythmia is induced [152, 155]. Multicenter automatic defibrillator implantation trial (MADIT) study [152] excluded subjects within 2 months after CABG and 3 months after percutaneous transluminal coronary angioplasty (PTCA), and MADIT-II study [156] excluded subjects within 3 months after revascularization. Conversely, early revascularization was permitted in MUSTT (Multicenter Unsustained Tachycardia Trial) study [155], which enrolled subjects at least 4 days after revascularization, and sudden cardiac death in heart failure trial (SCD-HeFT) study [157] made no specific exclusion on the timing of revascularization. However, in SCD-HeFT, the median time from CABG to enrollment was 3.1 years, and from PCI to enrollment was 2.3 years. Therefore, ICD implantation within 90 days of coronary revascularization for patients who otherwise meet ICD implant criteria for primary prevention of sudden cardiac death (SCD) is not addressed in the published device-based therapy guidelines. Revascularization has significant time-dependent benefits. In fact, MADIT-II study showed that the efficacy of ICD therapy in patients with ischemic left ventricular dysfunction is time dependent, with a significant life-saving benefit in patients receiving device implantation more than 6 months after coronary revascularization (CR). The lack of ICD benefit early after CR may be related to a relatively small risk of SCD during this period [158]. Although, sudden cardiac arrest (SCA) has a higher incidence in patients with reduced LVEF in the months after acute MI and/or following revascularization [159, 160]. The two randomized controlled trials, defibrillator in acute myocardial infarction (DINAMIT) and immediate risk stratification improves survival (IRIS), showed that early ICD implantation does not reduce mortality [161, 162]. In both of those trials, there was a reduction in arrhythmic death, which was counteracted by a concomitant increase in death due to other causes [163]. Similarly, the coronary artery bypass graft (CABG)-patch trial [164] examined ICD implantation at the time of elective CABG surgery showed a small decrease in arrhythmic death, but no benefit for overall mortality in patients with preoperative LVEF ≤35%. However, one should keep in mind that the epicardial ICDs tested in this trial differed significantly from the current transvenous endocardial ICD systems. A retrospective study evaluating ICD implantation within 3 months of cardiac surgery suggested the benefit of ICD implantation for secondary prevention. In this study, 164 patients were with ICD implantation within 3 months of cardiac surgery; 93 of these patients had an ICD for sustained pre or postoperative VT or VF requiring resuscitation. During the mean follow-up of 49 months; the primary endpoint was total mortality (TM) and/or appropriate ICD therapy (ICD-T), and secondary endpoints are the TM and ICD-T, and individual end points of TM and ICD-T were observed in 52 (56%), 35 (38%), and 28 (30%) patients, respectively, with 55% of TM, and 23% of ICD-T occurring within 2 years of implant [165].

Overall, ICD for *primary prevention* of SCD *can be useful* in patients who are within 90 days of revascularization and who are not within 40 days of an acute MI if:

- they are previously qualified for primary prevention of SCD or

- revascularization is unlikely to result in an improvement in LVEF to level >35% [166].

ICD for *secondary prevention* (i.e., in patients resuscitated from cardiac arrest due to VT/VF) *is recommended* for patients within 90 days of revascularization who have:

- previously satisfied criteria for ICD implantation if they have abnormal left ventricular function or

- SCD is unlikely related to myocardial ischemia/injury and have normal left ventricular function [166].

ICD implantation *can be useful* in patients who are within 90 days of revascularization if SCD was not related to acute myocardial ischemia/injury and who were subsequently found to have coronary artery disease that is revascularized with normal left ventricular function, or SCD was not related to acute myocardial ischemia/injury and who were subsequently found to have coronary artery disease that is revascularized with normal left ventricular function [166]. ICD is *not recommended* in patients within 90 days of revascularization who were resuscitated from cardiac arrest due to VT that was related to acute MI/injury, with normal left ventricular function, and who undergo complete coronary revascularization [166]. ICD with appropriately selected pacing capabilities *is recommended* in patients within 90 days of revascularization who require nonelective permanent pacing, who would also meet primary prevention criteria for implantation of an ICD, and in whom recovery of left ventricular function is uncertain or not expected [166].

An alternative approach for primary prevention of SCD in patients with ischemic cardiomyopathy and low LVEF undergoing revascularization would be the use the wearable cardioverter-defibrillator vest during the 3 months waiting period after revascularization until LVEF is reassessed and design made about permanent ICD implantation [163].

- Ventricular tachycardia ablation:

There are no studies of VT ablation in POVAs situation. In patients with extensive structural abnormalities, especially those with prior MI, multiple morphologies of VT might develop. Therefore, VT ablation does not eliminate the need for ICD and/or antiarrhythmic therapy. VT episodes might occur in up 0–60% of patients who have received an ICD for secondary prevention and in 2.5–12% of patients with ICD implanted for primary prevention [167]. Because antiarrhythmic drugs do not eliminate the risk of VAs, VT catheter ablation may be needed to reduce the frequency of VT episodes, especially patients with incessant VT or frequent ICD therapy [149]. Ablation is usually indicated in cases of recurrent, monomorphic VT arising from a specific substrate that can be targeted by mapping techniques.

5. Postoperative bradyarrhythmias (POBAs)

5.1. Epidemiology

Bradyarrhythmias (BAs) are common after cardiac surgery, but it mostly consists of transient episodes of low ventricular heart rate. The conduction defects post cardiac surgery

include sinus node dysfunction, partial and complete bundle branch blocks, and various degrees of atrioventricular (AV) block. The right bundle branch block (RBBB) was the most frequently noted abnormality [168]. Bradyarrhythmias may decrease cardiac output in patients with relatively fixed stroke volumes. The risk of developing conduction disturbances after CABG or valvular surgery leading to permanent pacemaker (PPM) implantation is about 0.4–1.1% of patients after isolated CABG and 3–6% after heart valve operations [169–171]. It seems that in the current surgical era that the incidence of postoperative PPM implantation has decreased due to improvements in surgical techniques, technological innovations and enhanced understanding of the mechanisms of injury [172]. However, some studies have shown an increased incidence of PPM implantation after cardiac surgery after the year 2000 [173].

5.2. Pathophysiology and risk factors

Conduction disorders after cardiac surgery are explained by one of the following two mechanisms: (1) direct trauma to the conduction system in operative procedures in proximity to the sinoatrial or AV nodes or the His bundle; or (2) ischemic injury to the conduction system due to extensive coronary artery disease might compromise myocardial protection during intraoperative cardioplegic arrest [174].

The risk factors for POBAs may be classified as preoperative, operative, and postoperative factors. Preoperative risk factors include age >75 years, the use of rate lowering cardiac medications (e.g., beta-blockers, calcium channel blockers, digoxin, and antiarrhythmic drugs), the presence of conduction system disease preoperatively, right bundle branch block (RBBB) or left bundle branch block (LBBB), first-degree AV block or left anterior fascicular block (LAFB) [169–171, 175–178].

Operative risk factors include myocardial ischemia, inadequate cardiac protection during surgery, and direct surgical injury to conduction system, prolonged CPB time and cross-clamp time, and reoperation [171, 172, 174, 179, 180].

Postoperative risk factors include postoperative conduction disturbances and high-grade AV block [174, 175, 181].

5.3. Clinical course and management

Temporary electrical pacing may be required in symptomatic bradycardias. It is common practice nowadays to place temporary epicardial atrial and ventricular pacing wires placed at the time of surgery to facilitate temporary pacing when needed. In some cases, as mentioned above, the conduction defect does not revert, and permanent pacing may be necessary. Chronotropic medications, such as theophylline or aminophylline, have been used for sinus bradycardia after transplantation to improve SSS [182] or high-grade AVB [183] and may decrease the need for permanent pacing but its long-term effect is not encouraging.

The challenge with POBAs is often to determine when to implant the PPM as the sinus node function or AV conduction may recover in some patients. Recovery of conduction system is

common with long-term follow-up. Only 30–40% of patients with a permanent pacemaker due to sinus node disease remain pacemaker dependent. However, the rate of recovery is less in patients with postoperative AVB, as 65–100% of patients with complete heart block, remain pacemaker dependent. Currently, the usual practice is to implant a PPM if postoperative symptomatic complete AVB or severe sinus node dysfunction persists longer than 5–7 days [184]. PPM implantation may be considered earlier if the underlying intrinsic rhythm is absent or temporary pacing leads fail.

6. Conclusion

Arrhythmias are common after CABG. Although tachyarrhythmias are frequent, they are usually transient and have a benign course. POAF represents the most frequently observed ATs. VAs are less common but have an adverse impact on the short and long-term outcome. POTAs management includes optimization of the patient's condition, controlling patient's risk factors, and careful attention to hemodynamic changes during surgery with using appropriate anesthesia. Postoperatively, it is important to correct reversible arrhythmia predisposing factors, followed by specific therapy based on the arrhythmia type and its hemodynamic effect.

On the other hand, bradyarrhythmias are also frequently observed after cardiac surgery. However, most of the conduction disturbances are transient and recovered spontaneously. PPM implantation may be required in patients with persistent symptomatic bradycardia due to sick sinus syndrome or second degree type 2, third degree, or high-grade AV block.

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