When and How Do I Have to Treat the Arrhythmias After Thoracic Surgery?

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15.1 Introduction

Cardiac arrhythmias are a common phenomenon affecting millions of people worldwide. In more than 60 % of healthy adults, atrial and ventricular premature beats can be detected on 24-h Holter monitoring [1]. In Europe, atrial fibrillation is present in about 2–3 % of the population, and its prevalence is likely to increase owing to widespread population aging [2]. Symptomatic bradycardia is a frequent reason for permanent pacemaker implantation. Ventricular extrasystoles are the most common type of arrhythmia that occurs after myocardial infarction. Arrhythmias may occur at any age but are more common among older people.

Since arrhythmias are common in the general population, it is not surprising that they are also frequently observed in patients undergoing surgery, particularly postoperatively. Some patients have a history of arrhythmias; in others, they occur for the first time. New arrhythmias are a well-known complication after surgery and may impact perioperative morbidity and mortality. This paper summarizes the pathophysiology, risk factors, and the management of arrhythmias in patients undergoing noncardiac thoracic surgery. Table 15.1 lists the negative implications these arrhythmias have.

Table 15.1 Negative implications of postoperative atrial fibrillation

Increased mortality
Increased pulmonary complications
Hemodynamic deterioration and instability
Induction or exacerbation of heart failure
Increased mean lengths of intensive care unit and hospital stay
Increased mean hospital charges

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Table 15.2 Risk factors for perioperative arrhythmias, particularly postoperative atrial fibrillation

Patient-related risk factors
Increasing age
Male sex
Structural heart disease (coronary artery disease, valve disease, left ventricular hypertrophy, systolic and diastolic left ventricular dysfunction)
Extracardiac risk factors (obesity, previous stroke, and concomitant lung disease)
Surgery-related risk factors
Surgical trauma (type of procedure/operation, magnitude of lung resection, dissection around the atria, mechanical factors such as instrumentation)
Hemodynamic stress (volume overload or depletion, hypertension, endogenous catecholamines)
Metabolic changes (hypoxemia, hypercarbia, acid-base imbalances)
Electrolyte disturbances (particularly hypokalemia)
Drug effects (beta-blocker withdrawal, digoxin, exogenous catecholamines, phosphodiesterase inhibitors (milrinone), levosimendan)

15.2 Pathophysiology

The clinical manifestation of arrhythmias requires both the presence of a vulnerable cardiac substrate and a trigger that initiates the arrhythmia. Changes in myocardial structure and electrical function constitute the substrate for arrhythmias. Examples for typical arrhythmia substrates are atrial fibroses (favoring atrial fibrillation) and a post-myocardial infarction scar (promoting ventricular tachycardia). The substrate is patient specific but may be modified by the below discussed risk factors. The arrhythmia trigger is defined as a single incident that may set off an arrhythmia. The trigger often takes the form of a premature beat, but may also consist of acceleration or slowing of the heart beat or myocardial stretch [3].

Many perioperative factors can be considered to affect both the arrhythmia substrate and trigger, thereby increasing atrial and ventricular susceptibility to arrhythmias. Risk factors can be classified into patient and surgery related (Table 15.2).

15.2.1 Patient-Related Risk Factors

Various patient-related clinical and nonclinical risk factors for postoperative arrhythmias have been described. One of the most relevant patient-specific risk factor is age. Increasing age has been demonstrated to be correlated with the development of arrhythmias in the general population as well as in the postoperative setting. Age-related structural and/or electrophysiological changes appear to lower the threshold for atrial and ventricular arrhythmias in the elderly. Since patients undergoing thoracic surgery present with a mean age of 67 years, the risk for the development of arrhythmias is inherently increased [4]. Arrhythmias are most likely to occur in patients with structural heart disease. Patients undergoing noncardiac thoracic

surgery often have the substrate of atrial enlargement or elevation in atrial pressures, which predispose to atrial tachyarrhythmias. A history of arrhythmias predisposes to postoperative events. Reported extracardiac risk factors for postoperative atrial tachyarrhythmias include obesity, previous stroke, and history of chronic obstructive pulmonary disease [5]. These risk factors are identical to those known to increase the propensity to the development of atrial fibrillation in the nonsurgical setting.

15.2.2 Surgery-Related Risk Factors

Postoperative arrhythmias are a well-known problem during and after cardiothoracic surgery; however they may also complicate major abdominal surgery. The prevalence depends on the type of operation and the extent of cardiac monitoring after surgery. The prevalence of postoperative arrhythmias may range from 4 % of patients undergoing major general surgery, vascular, and orthopedic surgery to 20 % in patients having elective colorectal surgery [6].

The trauma associated with surgical procedures predisposes patients to atrial and ventricular arrhythmias. Inflammatory mechanisms have been proposed in the development of postoperative arrhythmias since their incidence peaks at 2 to3 days after surgery [5]. Hemodynamic stress favoring arrhythmias may result from surgical trauma, volume overload or depletion, hypertension, and increased levels of endogenous catecholamines. Hypoxemia, hypercarbia, acid-base imbalances, as well as mechanical factors such as instrumentation often predispose to electrophysiological changes favoring the occurrence of arrhythmias. Hypokalemia may provoke postoperative atrial and ventricular arrhythmias [7].

Beta-blocker withdrawal has been associated with an increased rate of postoperative supraventricular tachyarrhythmias. A state of heightened catecholamine effect occurs because chronic beta-blocker use leads to a higher density of beta-adrenergic receptors. Digoxin use has been described as a risk factor for paroxysms of atrial fibrillation after surgery. The intravenous administration of catecholamines and phosphodiesterase inhibitors such as milrinone or enoximone and levosimendan has been reported to cause ventricular premature beats, short runs of ventricular tachycardia, and atrial fibrillation [5].

It is worth noting that the pathogenesis of postoperatively occurring atrial and ventricular arrhythmias is often multifactorial; it involves some or all of the mentioned mechanisms.

15.3 Atrial Fibrillation and Other Supraventricular Arrhythmias

Isolated atrial premature beats are very common after thoracic surgery and are often related to electrolyte or other metabolic imbalances. Atrial premature beats are usually readily identified by surface ECG or continuous telemetric monitoring. Paroxysmal supraventricular tachycardia develops in about 3 % of patients

undergoing general surgery. The most frequent sustained arrhythmia is atrial fibrillation. The incidence varies widely (from 12 to 44 %) depending on the type of surgery and patient characteristics. In an analysis of 2588 patients undergoing noncardiac thoracic surgery, the incidence of postoperative atrial fibrillation was 12.3 % [8]. In a multivariate analysis, significant risk factors for the occurrence of atrial fibrillation were male sex (relative risk (RR) 1.72), advanced age (RR in patients with age 70 or grater 5.3), a history of congestive heart failure (RR 2.51), a history of arrhythmias (RR 1.92), a history of peripheral vascular disease (RR 1.65), resection of mediastinal tumor or thymectomy (RR 2.36), lobectomy (8.91), bilobectomy (7.16), pneumonectomy (8.91), esophagoectomy (2.95), and intraoperative transfusions (1.39) [9].

Patients with atrial fibrillation have longer mean intensive care unit and hospital stays. Mean hospital charges are more than 30 % higher when compared with patients without atrial fibrillation. Importantly, an increased mortality in patients with postoperative atrial fibrillation has been demonstrated [10]. However, since many patients with postoperative atrial fibrillation have complex comorbidities, it is not clear to what extent the arrhythmia itself contributes to this increase in mortality [4].

With the aim to facilitate preoperative risk stratification, thoracic surgical procedures were recently divided into low- (<5 %), moderate- (5–10 %), and high- (>15 %) risk groups based on their expected incidence of postoperative atrial fibrillation (Table 15.3). In moderate- and high-risk patients, extended ECG monitoring is recommended (e.g., postoperative telemetry for 48–72 h) [11, 12].

Table 15.3 Risk stratification of thoracic surgery procedures for their risk of postoperative atrial fibrillation

Low-risk procedures (<5 % incidence)	Intermediate-risk procedures (5–15 % incidence)	High-risk procedures (>15 % incidence)
Flexible bronchoscopy with and without biopsy Photodynamic therapy Tracheal stenting Placement of thoracostomy tube or PleurX catheter (CareFusion Corporation, San Diego, California) Pleuroscopy, pleurodesis, decortication Tracheostomy Rigid bronchoscopy Mediastinoscopy Thoracoscopic wedge resection Bronchoscopic laser surgery Esophagoscopy/PEG/esophageal dilation and/or stenting	Thoracoscopic sympathectomy Segmentectomy Laparoscopic Nissen fundoplication/myotomy Zenker diverticulectomy	Resection of anterior mediastinal mass Thoracoscopic lobectomy Open thoracotomy for lobectomy Tracheal resection and reconstruction/carinal resection Pneumonectomy Pleurectomy Volume reduction/ bullectomy Bronchopleural fistula repair Clagett window Lung transplantation Esophagectomy Pericardial window

Atrial premature beats usually do not need specific treatment. Paroxysmal supraventricular tachycardia occurs from time to time and treatment is often simple. If vagal maneuvers are not successful, adenosine can be used in increasing doses (Table 15.4). Success rates exceed 95 %. Electrical cardioversion is rarely needed. The management of atrial fibrillation is much more complex.

15.3.1 Treatment of Atrial Fibrillation

Given the often transient nature of new-onset postoperative atrial fibrillation (the arrhythmia frequently resolves within 4–6 weeks), the control of the ventricular response rate is usually the initial therapy. Conventionally, nondihydropyridine calcium channel antagonists (verapamil and diltiazem) and digitalis have been used for treating postoperative atrial fibrillation (Table 15.4) [11, 12]. However, since calcium antagonists may be associated with hypotension and are contraindicated in patients with heart failure, they may not be the ideal drugs in patients with compromised heart function. The same is true for digitalis, which acts primarily by increasing vagal tone.

Table 15.4 Drugs used for postoperative arrhythmias

Drug	Dosing	Indication	Side effects
Adenosine	6 or 12 mg, the 18 mg as an iv bolus	Paroxysmal SVT	Transient heart block, flushing, chest pain, induction of AF (rare)
Atropine	0.4–1 mg iv	Bradycardia or AV block	Excessive tachycardia
Verapamil	5–10 mg iv	Rate control of AF, paroxysmal SVT	Hypotension, exacerbation of CHF, AV block
Diltiazem	10–20 mg iv bolus, then infusion at 5–15 mg/h	Rate control of AF, paroxysmal SVT	Hypotension, exacerbation of CHF, AV block
Esmolol	0.5 mg/kg bolus and infusion at 0.05 mg/kg/h; increase by 0.05 mg/kg/h every 5 min	Rate control of AF	Hypotension, bronchospasm, exacerbation of CHF
Metoprolol	5 mg iv every 5 min × 3	Rate control of AF	Hypotension
Digoxin	0.25 mg iv every 4–6 h up to 1 mg	Rate control of persisting AF	Delayed onset, nausea, vomiting
Amiodarone	Prophylaxis, 300 mg iv, then 600 mg orally for 3–5 days; treatment, 150 mg iv over 10 min, then 1 mg/min × 6 h, then 0.5 mg/min	Rate control and conversion of AF, frequent non-sustained/sustained VT, VF	Hypotension, bradycardia abnormal QTc prolongation with torsade de pointes (rare), acute respiratory distress syndrome (rare, after supra-therapeutic doses)

AF atrial fibrillation, *CHF* congestive heart failure, *SVT* supraventricular tachycardia, *VT* ventricular tachycardia, *VF* ventricular fibrillation

The effects of digitalis are attenuated postoperatively when sympathetic tome is markedly increased. Beta-blockers have been shown to be effective when atrial fibrillation occurs after surgery. Patients taking beta-blockers before surgery should have beta-blockade continued; abrupt withdrawal is associated with an increased risk of complications and should be avoided. Amiodarone iv should be preferred in patients with known severe systolic dysfunction. The drug also exerts antiarrhythmic effects, which may lead to the termination of the arrhythmias. A prospective, randomized, controlled, double-blinded study included 254 patients undergoing thoracic surgery for lung cancer. The patients received either 300 mg of amiodarone or placebo intravenously after surgery and an oral dose of 600 mg or placebo twice a day for 5 postoperative days. Amiodarone significantly decreased the prevalence of atrial fibrillation (38 patients (placebo group) vs. 11 patients (amiodarone group)). A number needed to treat of 4.4 (3.1–7.8) was calculated and adverse events occurred equally in both study arms (total of ten patients) [13].

Acute pulmonary toxicity has been reported with amiodarone in patients undergoing lung resection. Amiodarone or at least high amiodarone doses (>1000 mg/day) should be avoided in these patients. Preexisting pulmonary disease is associated with an increased risk of amiodarone pulmonary toxicity [14].

Class I antiarrhythmic drugs (sodium channel blockers like flecainide and propafenone) may be used in patients without structural heart disease. However, even in those patients, these agents may exert proarrhythmic effects (e.g., convert well-tolerated atrial fibrillation compromising atrial flutter).

Immediate electrical cardioversion is indicated in patients who demonstrate severe hemodynamic deterioration in response to new-onset atrial fibrillation. It is highly effective (>90 % conversion rate). However, early recurrences are frequent. Cardioversion from well-tolerated postoperative atrial fibrillation is usually not necessary because of a frequent self-limited course. New atrial fibrillation after thoracic surgery often resolves within 4–6 weeks, regardless of treatment.

Patients who develop atrial fibrillation after surgery are at risk of thromboembolic events, including stroke. In the individual postsurgical patient with an embolic event, the cause may be unclear, as underlying comorbidities are often responsible for such strokes, rather than the arrhythmia itself. However, based on evidence that anticoagulant therapy prevents episodes of systemic embolization in the broad population of patients with atrial fibrillation, anticoagulation seems reasonable in patients with postoperative AF who have stroke risk factors (age > 65, female gender, prior stroke, hypertension, congestive heart failure diabetes). However, a reduction of events with anticoagulant therapy in this population has never been well studied.

15.3.2 Prevention of Atrial Fibrillation

Several strategies to prevent postoperative atrial fibrillation have been studied. In daily practice, the most widely used prophylactic therapy seems to be the administration of beta-blockers. Prophylactic beta-blocker administration reduces the incidence of postoperative atrial fibrillation by about 50 % [15]. The greatest benefits

are seen when beta-blockers are initiated some time prior to surgery. Contraindications need to be carefully considered. Amiodarone significantly lowers the incidence of postoperative atrial fibrillation. The prophylactic potency seems to be comparable with that of beta-blockers. It is worth reminding the rare complication of intravenous amiodarone, the onset of acute respiratory distress syndrome in the postoperative period in patients undergoing lung resection. In one randomized study, magnesium iv was effective in reducing the incidence of postoperative atrial fibrillation. However, these results have never been confirmed by other studies.

Thoracic epidural analgesia (TEA) with bupivacaine has been shown to decrease the prevalence of atrial fibrillation after lung resection surgery [16]. However, a retrospective matched pair analysis could not confirm these results. A cohort of 1,236 patients undergoing resections was included into the study: 937 received a combination of general anesthesia and TEA (TEA) and 299 received general anesthesia only (non-TEA). After matching 311 TEA patients and 132 non-TEA patients, no differences on the occurrence of postoperative atrial arrhythmia could be demonstrated [17]. Thus, the role of central neuraxial analgesia for the prevention of postoperative atrial fibrillation is unclear.

To the knowledge of the authors, no systematic data are available that have evaluated how different institutions use prophylactic drug administration for the prevention of postoperative atrial fibrillation. The own experience suggests that atrial fibrillation prophylaxis is not a routine. Most institution try to optimize all aspects of perioperative care thereby minimizing the arrhythmia risk [18].

15.4 Ventricular Arrhythmia

Isolated ventricular premature betas documented postoperatively do not indicate an increased risk for the development of malignant ventricular tachyarrhythmias (i.e., sustained ventricular tachycardia, ventricular fibrillation), and, therefore, there is no need for treatment. Non-sustained and sustained ventricular tachyarrhythmias are rare. Reported incidences after surgery range from 0.5 to 1.5 %. Ten most patients developing theses arrhythmias have severe heart disease with depressed left ventricular function or suffer from severe acute postoperative complications (e.g., hemodynamic instability, myocardial ischemia, septic shock, major surgical complications).

When frequent and complex premature beats and non-sustained ventricular tachycardia occur, the correction of any reversible cause of arrhythmias (see above) should be pursued. Antiarrhythmic drugs may be indicated when longer repeated episodes of non-sustained or sustained ventricular tachycardia develop. The preferred antiarrhythmic drug is amiodarone administered intravenously [14]. Class I antiarrhythmic drugs have also been used successfully (e.g., lidocaine); however, in this setting, they are also associated with an increased risk for ventricular proarrhythmia. In the case of hemodynamic deterioration due to sustained ventricular tachyarrhythmias, either R-wave triggered DC cardioversion (in the case of hemodynamically well-tolerated ventricular tachycardia) or, after hemodynamic collapse

due to unstable ventricular tachycardia or ventricular fibrillation, immediate defibrillation and cardiopulmonary resuscitation may become necessary [10].

Most patients with known, previously documented ventricular tachyarrhythmias do have an implanted cardioverter/defibrillator. These devices are effective in terminating spontaneous arrhythmia, even in the postoperative setting. All devices should be thoroughly evaluated before and after surgery to make sure that its function has not been damaged or changed. If electrocautery is to be used, pacemakers should be placed in a triggered or asynchronous mode; implantable cardioverters should have arrhythmia detection suspended before surgery.

15.5 Bradyarrhythmias

Bradyarrhythmias are common after cardiac surgery (particularly after valve surgery), but are relatively rare after noncardiac thoracic surgery [19]. In the majority of cases, they consist of transitory episodes of low ventricular heart rate resulting from (usually preexisting) sick sinus syndrome or various degrees of atrioventricular blocks. They often result from increased vagal tone caused by an intervention, such as spinal or epidural anesthesia, laryngoscopy, or surgical intervention. Bradyarrhythmias may gain hemodynamic relevance because of a decrease in cardiac output [19]. Atropine can reverse symptomatic bradycardia. It is prudent to stop all unnecessary medications that can cause increased AV block like beta-blockers or calcium channel blockers. Temporary electrical pacing may be required in symptomatic bradycardias not responding to atropine. In some cases, when the conduction defect does not revert, permanent pacing may be necessary.

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