

*Current Concepts***ATRIAL ARRHYTHMIAS AFTER
CARDIOTHORACIC SURGERY**STEVE R. OMMEN, M.D., JOHN A. ODELL, M.D.,
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ATRIAL tachyarrhythmias early in the recovery period after cardiothoracic surgery are common; they develop in 11 to 40 percent of patients after coronary-artery bypass grafting¹⁻³ and in over 50 percent of patients after valvular surgery.² Technical advances in surgery and anesthesia, as well as changing methods of myocardial protection, have not decreased the incidence of postoperative atrial tachyarrhythmias.^{1,3,4} Although such arrhythmias have been thought of as transient and benign, they may have important consequences. Atrial fibrillation, flutter, and tachycardia are related forms of atrial tachyarrhythmia that may coexist in patients after cardiothoracic surgery and have similar clinical and therapeutic implications.

ETIOLOGY

The underlying mechanism of recurrent atrial fibrillation in patients who are not undergoing surgery appears to be multiple, simultaneously propagating reentrant electrical impulses, or wavefronts, that circulate inside the atria.⁵⁻⁸ Less is known about the cause of postoperative atrial fibrillation, for which other arrhythmogenic factors exist. Frequently, pericardial inflammation or effusion is present after cardiac surgery before atrial fibrillation develops.^{9,10} Excessive production of catecholamines and autonomic imbalance during the postoperative period may also be involved in the arrhythmias. The role of the autonomic nervous system in atrial fibrillation unrelated to surgery has been well established,^{11,12} and epinephrine has been found to cause arrhythmogenic changes in human atrial cells.¹³ The

interstitial mobilization of fluid with resultant changes in volume and pressure can affect the neurohumoral environment and electrical properties of the atria.¹⁴

Electrophysiologic studies of cells of the right atrial appendage in patients who undergo cardiac surgery have led to the identification of two distinct action potentials: normal electrical activity and electrical activity characterized by resting membrane depolarization, slowed upstroke, reduced amplitude, and prolonged refractoriness.¹³ This latter type of action potential leads to slow conduction and regions of blockage, both of which can in turn lead to arrhythmias. In one study, only 1 of 10 patients with normal action potentials had postoperative atrial fibrillation, as compared with 8 of 10 patients with the abnormal action potential.¹³ Although the study examined the electrical correlates of postoperative atrial arrhythmias, it did not explain why these rhythms develop.

RISK FACTORS**Preoperative Factors**

Patients with a history of atrial fibrillation are at increased risk for postoperative atrial arrhythmia.³ It is more difficult to predict the occurrence of these tachyarrhythmias in patients without such a history. As in atrial fibrillation unrelated to surgery, age is consistently the independent factor most strongly associated with postoperative atrial fibrillation.¹⁻³ As an estimate, less than 5 percent of surgical patients under 40 years of age, but more than one third of those who are 70 or older, have postoperative atrial tachyarrhythmias, a near doubling of the risk in each successive decade of life. Age-associated changes in the atria such as dilatation, muscle atrophy, and decreased conduction¹⁵ may explain the strong association.

Concomitant valvular heart disease is also associated with postoperative atrial tachyarrhythmias.² It is unclear whether this is because of the additional complexity of the required surgical procedure or the valvular disease itself. Neither the degree of ischemia nor the extent of coronary artery disease is a consistent predictor of postoperative atrial tachyarrhythmias.¹⁻³ Electrocardiographic studies have found the duration of the signal-averaged P wave to be significantly longer in patients who subsequently had postoperative atrial tachyarrhythmias (negative predictive accuracy, 82 to 100 percent; positive predictive accuracy, 37 to 65 percent).^{16,17} Characteristics that have not been identified consistently as inde-

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pendent risk factors include hypertension, left ventricular dysfunction, angina pectoris, and noncardiac illnesses.¹⁻³

Intraoperative Factors

Cardiopulmonary bypass deprives the heart of blood flow and thus could result in atrial injury and postoperative arrhythmias. Although some data suggest that extremely long surgeries increase the likelihood of atrial fibrillation, the duration of aortic cross-clamping or cardiopulmonary bypass is not a strong predictor of postoperative arrhythmias.

Myocardial protection is designed to reduce the metabolic demand of the ventricular myocardium during the operation. Traditional hypothermic cardioplegia does not adequately cool the atria or ensure complete electrical arrest in the atria,^{18,19} and the use of warm cardioplegia is becoming more common. However, studies of myocardial protection have not found different rates of postoperative atrial tachyarrhythmias to be associated with the various techniques.²⁰⁻²²

Routine testing for repolarization and conduction abnormalities is not technically feasible, but the value of attempting to induce atrial fibrillation in the operating room has been studied in 50 patients who underwent coronary bypass surgery.²³ The intraoperative induction of atrial fibrillation had a sensitivity of 94 percent and a specificity of 41 percent (negative predictive value, 93 percent) for the occurrence of postoperative atrial tachyarrhythmias. At present, there are no practical intraoperative screening techniques for risk stratification.

CONSEQUENCES OF POSTOPERATIVE ARRHYTHMIAS

Atrial arrhythmias are most frequent in the first two to three days after cardiothoracic surgery, but they can occur at any point in the recovery period.³ Although postoperative atrial tachyarrhythmias are often seen as a temporary problem related to surgery, neither the long-term maintenance of sinus rhythm nor the long-term outcome of these arrhythmias has been studied rigorously. The arrhythmias are often a transient phenomenon, but they recur in a significant proportion of patients.

Atrial arrhythmias can lead to discomfort and hemodynamic instability. Though perhaps not causally related to complications, the occurrence of postoperative atrial tachyarrhythmias is associated with a need for prolonged inotropic support, the use of intraaortic balloon pumps, and reoperation for bleeding.³ Patients with postoperative tachyarrhythmias spend nearly twice as many days in the intensive care unit as patients without this complication, and their total period of hospitalization is extended by three to four days,² with accompanying increased costs.²⁴

The thromboembolic complications of postoperative atrial tachyarrhythmias, particularly atrial fibrillation, can be devastating. Stroke is estimated to occur after coronary-artery bypass grafting in 1 to 6 percent of patients.^{2,25} Risk factors for postoperative stroke include a history of neurologic deficit (relative risk, 6.0), a history of congestive heart failure (5.3), mitral regurgitation (4.3), carotid-artery bruits (3.9), and postoperative atrial tachyarrhythmias (3.0).²⁵

The thromboembolic potential develops early after the onset of atrial fibrillation. In 317 patients with nonsurgical acute-onset atrial fibrillation (less than three days' duration), transesophageal echocardiography showed a 14 percent prevalence of left atrial thrombus and a 39 percent prevalence of spontaneous echo contrast.²⁶ Spontaneous echo contrast occurs in states of low blood flow; it is thought to be due to the formation of rouleaux and the presence of fibrinogen (or its products) and is a strong predictor of subsequent thromboembolism.²⁷

PREVENTION OF ATRIAL ARRHYTHMIAS

Digoxin is frequently given to patients as prophylaxis against atrial arrhythmias, although data do not support the practice.²⁸ In fact, because digoxin shortens atrial refractoriness, it may theoretically increase the likelihood of atrial fibrillation.

Two meta-analyses of beta-blockers in the prevention of postoperative atrial tachyarrhythmias found the drugs to have a protective effect.^{29,30} In addition to the general finding, in nearly every trial studied there was at least some evidence of a favorable effect.²⁸ However, caution must be exercised in interpreting the results of these studies. The methods of detecting arrhythmia varied, and in several of the trials, some patients randomly assigned to placebo were withdrawn from beta-blocker therapy they had been given preoperatively. Patients whose beta-blocker therapy is discontinued postoperatively have a higher incidence of postoperative atrial tachyarrhythmias.^{31,32} Patients who are receiving beta-blockers before surgery should continue to receive them, if possible, afterward.

Verapamil helps control the heart rate during atrial arrhythmias, but it does not consistently prevent arrhythmias.²⁸ Studies comparing intravenous diltiazem and placebo have found both better control of the heart rate and a significantly lower incidence of postoperative atrial tachyarrhythmias (5 percent vs. 18 percent) among patients given diltiazem, without evidence of hemodynamic compromise.^{33,34}

There are few data to support the use of membrane-active antiarrhythmic agents in the prevention of postoperative atrial arrhythmias. Small trials of quinidine,³⁵ procainamide,³⁶ amiodarone,³⁷ propafenone,³⁸ and sotalol³⁹ have not found these agents to

be of significant benefit. Larger studies found sotalol to be effective in the prevention of supraventricular arrhythmias after coronary-artery bypass surgery (and to result in shorter hospital stays), though associated hypotension was an important problem for patients.^{40,41} It is unclear which of the effects of sotalol — membrane activity or beta-blockade — was responsible for the benefit.

Overall, the only firm recommendation that can be made is to avoid the withdrawal of beta-blockers. Prophylactic strategies are more likely to be efficacious if they are applied only to patients at high risk.

PREVENTION OF THROMBOEMBOLISM

Because a potential for thromboembolism develops soon after the onset of atrial fibrillation, prompt anticoagulant therapy should be strongly considered if the arrhythmia persists for 24 hours or more. However, no studies have addressed the specific timing of thrombus formation or the risk-benefit ratio of anticoagulant therapy in postoperative atrial fibrillation. Data from a large series of patients with atrial fibrillation unrelated to surgery found no difference between the rate of thromboembolism in patients with chronic arrhythmias and the rate in those with paroxysmal arrhythmias.⁴² Whether this pattern is also found in postoperative arrhythmias is unknown. Patients with paroxysmal atrial fibrillation, as well as those with persistent episodes, should be considered at risk for stroke. The risk of postoperative bleeding must be factored into the decision of whether, and when, to give anticoagulant therapy to a patient with postoperative atrial tachyarrhythmia.

Atrial thrombi appear much less likely to form in patients with atrial flutter or atrial tachycardia than in patients with fibrillation. The prophylactic use of aspirin in postoperative atrial fibrillation has not been studied, but in patients with atrial fibrillation unrelated to surgery, a dose of 325 mg per day decreased the number of thromboembolic events, as compared with placebo,⁴³ but 75 mg per day did not.⁴⁴ For patients with contraindications to long-term anticoagulant therapy, the potential benefits of the removal of the left atrial appendage are still under investigation.^{45,46}

TREATMENT

Pharmacologic Therapy

The principles of treatment for postoperative atrial tachyarrhythmias are similar to those for atrial arrhythmias in other circumstances, except that inflammation and increased sympathetic tone have an important etiologic role in postoperative arrhythmias. The control of the ventricular rate, anticoagulation, and conversion to sinus rhythm are the goals of therapy. However, it is often difficult to

maintain sinus rhythm postoperatively, particularly in the first 14 days after surgery. Digoxin is commonly used to control the heart rate in atrial fibrillation, though its benefits may be limited in the presence of excess catecholamines.^{11,12} Digoxin has two effects on the atrioventricular node. At lower concentrations, conduction is slowed by a vagotonic effect that is easily reversed by enhanced sympathetic tone. Higher concentrations of digoxin are required to have a direct effect on atrioventricular conduction. However, postoperative myocardial sensitization may reduce the difference between a therapeutic and a toxic plasma concentration of digoxin. If digoxin is used, there must be adequate body stores before an effect can be expected.⁴⁷ Although it may not be as effective as other atrioventricular nodal blocking drugs, the positive inotropic effect of digoxin makes it the drug of choice in patients with left ventricular dysfunction and postoperative atrial tachyarrhythmias.

Diltiazem is effective, and its short-term administration is tolerated by patients with ventricular dysfunction.⁴⁸ Verapamil and esmolol are also frequently given after surgery to control the ventricular response. Although verapamil and esmolol lower the heart rate, they often also cause hypotension.^{49,50}

Reports on the use of various antiarrhythmic drugs to induce a return to normal rhythm in patients with postoperative atrial tachyarrhythmias describe a wide variation in results, probably due to differences among patient populations, dosing regimens, and study designs and to the fact that spontaneous reversions to sinus rhythm are possible.⁵¹⁻⁵⁶ In the United States, procainamide, quinidine, and amiodarone are available for intravenous administration for this purpose. Although both procainamide and quinidine can cause hypotension, this problem is more common with quinidine.

Intravenous amiodarone is not more effective than digoxin in inducing cardioversion, and it is unclear whether either drug is better than placebo.⁵⁵ Ibutilide, an intravenous antiarrhythmic agent with class III properties, was recently approved by the Food and Drug Administration for pharmacologic cardioversion in patients with atrial fibrillation or flutter. No data have been published on the postoperative use of ibutilide. Because the drug prolongs repolarization, torsade de pointes is a potential adverse reaction and continuous electrocardiographic monitoring for at least four hours after administration is critical even if sinus rhythm is restored. Flecainide,⁵³ propafenone,⁵⁴ and sotalol⁵⁶ are available for intravenous use in some countries. On the basis of the increased mortality seen in the Cardiac Arrhythmia Suppression Trial (CAST),⁵⁷ flecainide should not be the first choice for long-term oral therapy in patients with postoperative atrial arrhythmias. Therapy with propafenone (like flecainide, a class IC an-

TABLE 1. DOSAGE OF DRUGS USED TO TREAT POSTOPERATIVE ATRIAL ARRHYTHMIAS.*

DRUG	IV BOLUS	IV INFUSION	ORAL DOSE	COMMENTS
Anticoagulants				
Heparin	5000 units	Sufficient to produce aPTT 2–3 times control value		
Warfarin			Sufficient to produce INR of 2.0–3.0	
Drugs to control heart rate				
Beta-adrenergic blockers				
Esmolol	500 µg/kg	50–200 µg/kg/min		May cause bradycardia, hypotension, or bronchospasm
Propranolol	1 mg every 5 min (maximum, 5 mg)	2–3 mg/hr	10–80 mg every 6–8 hr	
Metoprolol	5 mg every 5 min (maximum, 15 mg)		25–100 mg twice a day	
Atenolol	5 mg every 10 min (maximum, 10 mg)		50–200 mg/day	Adjust dose in the case of renal failure
Calcium-channel blockers				
Diltiazem	0.25 mg/kg	5–15 mg/hr	180–360 mg/day	May cause bradycardia or hypotension
Verapamil	0.15 mg/kg	5 mg/hr	120–480 mg/day	
Antiarrhythmic drugs				
Class IA				
Procainamide	10–15 mg/kg	1–6 mg/min	500–2000 mg twice a day†	May cause hypotension; adjust dose in the case of renal failure
Quinidine	200–400 mg		300–600 mg three times a day†	May cause GI upset or hypotension; adjust dose in the case of renal failure
Disopyramide			150–300 mg twice a day†	May cause CHF; adjust dose in the case of renal failure
Class IC				
Flecainide	2 mg/kg		50–150 mg twice a day	May cause bronchospasm
Propafenone	1–2.5 mg/kg	2 mg/min	150–300 mg three times a day	
Class III				
Amiodarone	150 mg	1000 mg/day	200–400 mg/day	May cause pulmonary fibrosis, cirrhosis, or thyroid abnormalities
Sotalol	0.2–1.5 mg/kg	0.15 mg/kg/hr	80–240 mg twice a day	May cause hypotension; adjust dose in the case of renal failure
Ibutilide	1 mg (may repeat once)			

*IV denotes intravenous, aPTT activated partial-thromboplastin time, INR international normalized ratio, kg kilogram of body weight, GI gastrointestinal, and CHF congestive heart failure.

†This dosage is for the long-acting preparation.

tiarrhythmic drug) should also be cautioned against, according to some experts, though it was not studied in CAST. Table 1 lists recommended doses of medications used to treat postoperative atrial arrhythmias.

Electrical Cardioversion

Early electrical cardioversion may be necessary in hemodynamically compromised patients. Unfortunately, there are drawbacks to countershock therapy. The conditions (pericardial inflammation, autonomic imbalance, and so forth) that contributed to the arrhythmia persist after cardioversion and may lead to relapse. There is also evidence that atrial stunning is present after cardioversion. The consequent lack of mechanical activity may lead to new, spontaneous echocardiographic contrast formation and the potential development of thrombi.⁵⁸

GENERAL THERAPEUTIC RECOMMENDATIONS

To treat a postoperative atrial arrhythmia, we recommend prompt anticoagulant therapy with heparin and the administration of an atrioventricular nodal blocking agent to control the heart rate (Fig. 1). Unless contraindicated, beta-adrenergic-blocking drugs should be strongly considered to control the heart rate because of their sympatholytic activity. If beta-blockers are contraindicated, the agent of choice is diltiazem. Digoxin may be useful for patients with left ventricular dysfunction. If there is no spontaneous reversion to sinus rhythm within 24 hours, electrical cardioversion should be considered. If atrial fibrillation recurs, treatment with a membrane-active antiarrhythmic drug should be started. Postoperative atrial tachyarrhythmias may result from the pericardial inflammation and autonomic imbalance of

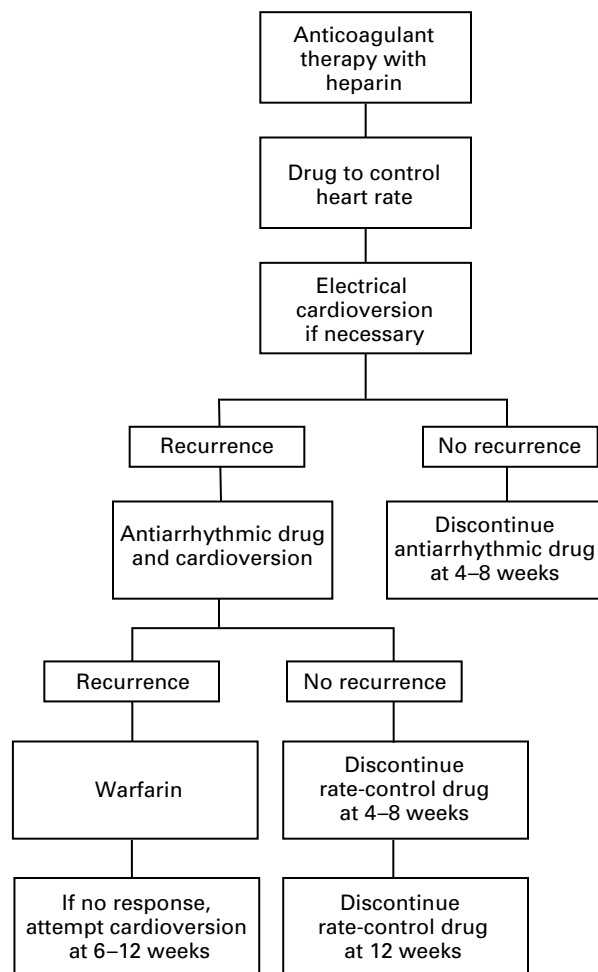


Figure 1. Recommended Treatment of Postoperative Atrial Tachyarrhythmias.

the early postoperative period; they are therefore often transient, and an attempt should be made to discontinue the antiarrhythmic medications after the patient leaves the hospital.

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