Noncardiac surgery: Postoperative arrhythmias

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Postoperative arrhythmias are common and represent a major source of morbidity after both cardiac and noncardiac surgical procedures. Postoperative dysrhythmias are most likely to occur in patients with structural heart disease. The initiating factor for an arrhythmia in a given patient after surgery is usually a transient insult, such as hypoxemia, cardiac ischemia, catecholamine excess, or electrolyte abnormality. Management includes correction of these imbalances and medical therapy directed at the arrhythmia itself. The physiologic impact of arrhythmias depends on arrhythmia duration, ventricular response rate, and underlying cardiac function. Similarly, urgency and type of treatment is determined by the physiologic impact of the arrhythmia, as well as by underlying clinical status. The purpose of this review is to provide current concepts of diagnosis and acute management of arrhythmias after noncardiac surgery. A systematic approach to arrhythmia diagnosis and evaluation of predisposing factors is presented, followed by consideration of specific bradyarrhythmias and tachyarrhythmias in the postoperative setting. (Crit Care Med 2000; 28[Suppl.]:N145–N150)

KEY WORDS: arrhythmia; postoperative; cardioversion; atrial fibrillation

P ostoperative arrhythmias are common and represent a major source of morbidity. The patient's underlying cardiac status is the key to management. Although this manuscript is focused on noncardiac surgery, findings from studies conducted after cardiac surgery can be extrapolated (albeit carefully) to these perioperative arrhythmias.

Postoperative dysrhythmias are most likely to occur in patients with structural heart disease. The initiating factor in a given patient, however, is usually a transient imbalance, often related to hypoxia, ischemia, catecholamines, electrolyte abnormalities, or other factors. Management includes correction of these imbalances, as well as specific therapy for the arrhythmia itself.

The physiologic impact of a given arrhythmia depends on its duration, ventricular response rate, and the underlying cardiac function. Bradyarrhythmias may decrease cardiac output in patients with relatively fixed stroke volumes. Loss of the atrial "kick" may cause a dramatic increase in pulmonary pressures in patients with diastolic dysfunction. Similarly, tachyarrhythmias may decrease diastolic filling and reduce cardiac output, resulting in hypotension, and increase myocardial oxygen consumption, produc-

ing myocardial ischemia. Clearly, the impact of an arrhythmia in a particular situation depends on the patient's cardiac physiology and function. The urgency for and type of treatment required is determined by the physiologic impact of the arrhythmia, as well as by clinical status.

The purpose of this review is to provide an updated approach to current concepts of diagnosis and acute management of arrhythmias after noncardiac surgery. A systematic approach to diagnosis and evaluation of predisposing factors will be presented, followed by consideration of specific arrhythmias.

DIAGNOSIS OF POSTOPERATIVE ARRHYTHMIAS

Basic Principles. The first principle in managing arrhythmias is to treat the patient rather than the electrocardiogram. Accordingly, one must first decide whether the problem is an arrhythmia or an artifact and whether the cardiac rhythm is sufficient to account for the patient's problem.

The next step is to establish the urgency of treatment. Clinical assessment includes evaluation of pulse, blood pressure, peripheral perfusion, and the presence of myocardial ischemia and congestive heart failure. If the patient loses consciousness or becomes hemodynamically unstable in the presence of a tachyarrhythmia other than sinus tachycardia, prompt electrical cardioversion is indi-

cated. If the patient is stable, there is more time to establish the diagnosis and decide on the most appropriate course of treatment. Bradyarrhythmias produce less diagnostic challenge and treatment options are relatively straightforward.

The goals of antiarrhythmic therapy depend on the type of rhythm disturbance. The initial goal must always be to establish hemodynamic stability. In the critical care unit, the first treatment of tachyarrhythmias is to slow the ventricular response. In the case of problematic bradyarrhythmias, ventricular rate must be increased. The next goal is to restore sinus rhythm, if possible. If restoration of sinus rhythm cannot be achieved, prevention of complications becomes an issue.

Classification of Tachyarrhythmias. Tachyarrhythmias are usually classified according to anatomical origin, as supraventricular or ventricular. Supraventricular arrhythmias include sinus tachycardia, atrial flutter and fibrillation, ectopic atrial tachycardia, multifocal atrial tachycardia, junctional tachycardia, atrioventricular (A-V) nodal reentrant tachycardias, and accessory pathway reciprocating tachycardias. Ventricular arrhythmias consist of premature ventricular beats, ventricular tachycardia, and ventricular fibrillation.

It is useful to consider tachyarrhythmias from a treatment standpoint, dividing them into those that traverse the A-V node and those that do not. Tachyar-

rhythmias traversing the A-V node include atrial flutter and fibrillation, automatic (ectopic) atrial tachycardia, multifocal atrial tachycardia, A-V nodal reentry, and A-V reciprocating tachycardias. Tachyarrhythmias that do not traverse the A-V node include preexcited atrial arrhythmias (with antegrade conduction through a bypass tract), ventricular tachycardia, and ventricular fibrillation. The clinical import of this classification is that the ventricular response of the tachycardias which traverse the A-V node can be controlled by pharmacologically altering A-V nodal conduction. When the arrhythmias do not use the A-V node, drugs that slow A-V nodal conduction can be dangerous.

Tachycardia Diagnosis. A comprehensive description of arrhythmia diagnosis is beyond the scope of this discussion. However, a basic approach to systematic arrhythmia diagnosis will be presented. A 12-lead electrocardiogram with a long rhythm strip and a previously obtained 12-lead electrocardiogram for comparison are ideal; barring that, a long rhythm strip of a lead in which P waves are visible is preferred. The approach can be outlined by using the following four steps (Table 1) (1).

Locate the P Wave

Is a P Wave Visible? If no P waves are present and the QRS complexes are irregular, the rhythm is most likely atrial fibrillation. A regular narrow QRS complex tachycardia without discernible P waves is most likely caused by A-V nodal reentry. Recordings obtained from esophageal or intracardiac leads can provide useful information about atrial activity.

How Fast Are the P waves? In adults, sinus tachycardia usually occurs at a rate between 100 and 180 beats/min. Atrial tachycardias and A-V nodal reentrant tachycardias usually present with rates from 140 to 220 beats/min. Rates between 260 and 320 beats/min are most likely to represent atrial flutter (Table 2).

What Is the Morphology of the P Waves? Normal P waves are upright in

leads I, II, aVF, and V_4–V_6. Automatic atrial tachycardia often presents with negative P waves in one or more of these leads.

Establish the Relationship Between the P Wave and QRS Complex

If there are more P waves than QRS complexes, then A-V block is present. If there are more QRS complexes than P waves, the rhythm is junctional or ventricular in origin. If the relationship is 1:1, then measurement of the PR interval (or RP interval) can provide useful diagnostic clues.

Examine the QRS Morphology

A narrow QRS complex (<0.12 msec) indicates a supraventricular arrhythmia. A wide QRS complex can be present with either ventricular tachycardia or supraventricular tachycardia with preexisting bundle-branch block, aberrant ventricular conduction, or antegrade conduction via an accessory A-V connection.

Search for Other Clues

The clues to look for depend on the situation. Carotid sinus massage (and other vagal maneuvers) increase A-V block and can either break a supraventricular tachycardia or bring out previously undetected flutter waves. Any patient with a ventricular rate of exactly 150 beats/min should be suspected of having atrial flutter with 2:1 A-V block. A rate >200 beats/min in an adult should raise the suspicion of an accessory pathway. In the presence of a wide complex tachycardia, A-V dissociation is diagnostic of ventricular tachycardia. Capture beats (supraventricular conduction with a narrow QRS complex) make aberration unlikely and favor a ventricular origin of wide QRS complexes. The presence of fusion beats (which result from simultaneous ventricular activation via ventricular and supraventricular sources) likewise suggests ventricular tachycardia. Certain QRS morphologies favor ventricular tachycardia over aberration (2).

PREDISPOSING FACTORS FOR POSTOPERATIVE ARRHYTHMIAS

It is clearly recognized that postoperative stress predisposes patients to develop-

opment of arrhythmias. Specific factors include hypoxemia, hypercarbia, myocardial ischemia, endogenous or exogenous catecholamines, electrolyte or acid-base imbalances, and drug effects, as well as mechanical factors, such as instrumentation (3). Atrial fibrillation after cardiac surgery is the best-studied postoperative arrhythmia. Proposed mechanisms for postoperative atrial fibrillation include acute atrial distention, atrial inflammation from surgical trauma or pericarditis, ischemic injury caused by cardioplegia, and electrolyte and volume shifts during bypass that can alter atrial repolarization (4). It should be recognized, however, that arrhythmogenesis may be multifactorial; attribution of an arrhythmia to a single predisposing factor may oversimplify a complex situation. For example, hypokalemia predisposes to the development of perioperative ventricular arrhythmias. Catecholamine release, however, increases cellular potassium uptake and thus, decreases serum potassium levels (5). In this context, it may not be clear whether a given arrhythmia is related to hypokalemia, is catecholamine-mediated with hypokalemia as an epiphenomenon, or results from a combination of both factors.

Regardless of their complexity, it is clear that identification and correction of potential predisposing factors is essential for prevention and management of postoperative arrhythmias. The duration and severity of these disturbances, combined with assessment of cardiac function, often determines what therapy is instituted. Self-terminating arrhythmias, in the setting of a transient stress and without overt cardiac disease, often need no therapy at all. On the other hand, the development of a transient, hemodynamically significant arrhythmia in a patient likely to remain under stress for some time, points to a need for therapy aimed at preventing recurrence.

BRADYCARDIAS

Sinus Node Dysfunction. Bradycardias associated with sinus node dysfunction include sinus bradycardia, sinus pause, sinoatrial block, and sinus arrest. In the perioperative setting, these disturbances often result from increased vagal tone caused by an intervention, such as spinal or epidural anesthesia, laryngoscopy, or surgical intervention (3). Initial therapy for postoperative bradyarrhythmias does not differ from other acute circum-

Table 1. Rhythm diagnosis

Locate the P wave
Analyze the relationship between the P and QRS
Examine the QRS morphology
Search for other clues
Establish the rhythm in the clinical setting

Table 2. Differential diagnosis of common atrial arrhythmias

Arrhythmia	Atrial Activity	Atrial Rate (beats/min)	Ventricular Activity	A-V Relation
Sinus tachycardia	Normal P wave; PR often short	100–180	Normal QRS; regular RR	1:1
Paroxysmal SVT			Normal QRS; regular RR	
A-V nodal reentry	P wave in or after QRS	140–220	Normal QRS; regular RR	1:1[a]
Ectopic atrial tachycardia	Abnormal P wave; normal PR	120–220	Normal QRS; regular RR	1:1[a]
Junctional tachycardia	A-V dissociation or retrograde activity	80–150	Normal QRS; regular RR	1:1
Atrial flutter	"Saw-tooth" waves in leads II, III, aVF	260–320	Normal QRS; regular RR	Usually 2:1, but not always
Atrial fibrillation	Chaotic	—	Normal QRS; irregularly irregular RR	—
Multifocal atrial tachycardia	Three or more distinct P wave morphologies	110–170	Normal QRS; irregular RR	1:1

A-V, atrioventricular; SVT, supraventricular tachycardia.
[a]A-V block may be seen.

stances. If bradycardia is transient and not associated with hemodynamic compromise, no therapy is necessary. If it is sustained or severe enough to compromise end-organ perfusion, therapy with antimuscarinic agents (atropine) or β agonists may be initiated. Transcutaneous or transvenous pacing may be necessary in some patients.

Patients with a combination of bradycardia, paroxysmal atrial tachycardias, and preexisting conduction system disease can be challenging to manage, inasmuch as pharmacotherapy for slow rhythms predisposes to fast ones and vice versa (6). In these patients, insertion of a pacemaker may facilitate the administration of agents with negative chronotropic effects.

HEART BLOCK

The most common cause of acquired chronic A-V heart block is fibrosis of the conducting system. Although preexisting conduction system disease is a risk factor for the development of complete heart block, no single laboratory or clinical variable identifies patients at risk for progression to high degree A-V block (7). Acute myocardial infarction is an important cause of transient A-V block. Criteria for insertion of temporary and permanent pacemakers in postoperative infarction are the same as the criteria for any infarction (7).

High-grade second- or third-degree A-V block persisting for 7–14 days after cardiac surgery is an indication for permanent pacing (7), and it seems reasonable to extrapolate these recommendations to patients with high-degree A-V block persisting after noncardiac surgery. Whether one needs to wait this long is unclear, because these patients usually have no obvious reversible cardiac injury. The role of permanent pacing in patients with transient postoperative A-V block and residual bifascicular block has not been established.

SUPRAVENTRICULAR TACHYCARDIAS

Supraventricular arrhythmias are common after surgery. Their frequency rate was estimated at 4% in a large registry of patients undergoing major noncardiac procedures (8), 3.2% in a multicenter study of patients undergoing abdominal aortic aneurysm repair (9), and almost 13% in 295 patients undergoing thoracotomy for lung cancer (10). Supraventricular tachyarrhythmias can be broken down into sinus tachycardia, atrial tachycardias (including multifocal), A-V nodal reentrant tachycardias, and A-V reciprocating tachycardias.

Sinus tachycardia is common and may be appropriate. Common potential causes in the postoperative setting include anxiety, pain, fever, hypovolemia, anemia, hypoxemia, medications, and, occasionally, alcohol withdrawal. Less common causes include thyrotoxicosis, pheochromocytoma, and methemoglobinemia (11). It is important to recognize appropriate sinus tachycardia, because treatment should be directed at the underlying cause rather than at the rhythm.

Atrial tachycardias can arise from either an automatic focus or a reentrant pathway. They comprise approximately 8% of paroxysmal supraventricular tachycardias in adults and are more common in children. They are diagnosed by identification of a P wave morphology different from that in sinus rhythm. Potential mechanisms include abnormal automaticity or triggered activity; digitalis toxic-

ity should also be considered (12). Short bursts of atrial tachycardia do not require specific drug therapy unless they are frequent and symptomatic. Multifocal atrial tachycardia usually occurs in acutely ill, elderly patients, or in patients with pulmonary disease (13). Multifocal atrial tachycardia is diagnosed by the presence of three or more different P wave morphologies and an irregularly irregular rhythm. The most useful therapy for multifocal atrial tachycardia therapy is to treat the underlying causes, including hypoxemia and hypercapnia, myocardial ischemia, congestive heart failure, or electrolyte disturbances. β blockers and calcium-channels blockers can slow the ventricular rate (14). These agents should be used cautiously in patients with decreased left ventricular contractility. Amiodarone may be helpful in patients refractory to more conservative measures.

Atrioventricular nodal reentry is the most common paroxysmal supraventricular tachycardia, accounting for ~60% of patients (12). Dual A-V nodal pathways with different conduction velocities and refractory periods are usually present, setting up the substrate for reentry. P waves are usually not seen on the electrocardiogram caused by near simultaneous retrograde atrial and antegrade ventricular activation. Carotid sinus massage or other vagal maneuvers may restore sinus rhythm. An infusion of 6–12 mg of iv adenosine is the preferred initial drug treatment because its extremely short half-life minimizes side effects. Cardioversion may be necessary for circulatory insufficiency. β blockers or calcium-channel blockers can also be used; the latter should not be used in patients with known or suspected preexcitation for fear

Table 3. Drugs used for postoperative arrhythmias

Drug	Dosing	Indications	Side Effects
Adenosine	6 mg; then 12 mg iv	Paroxysmal SVT; diagnosis of wide or narrow QRS tachycardias	Transient heart block, flushing, chest pain
Atropine	0.4–1 mg iv	Bradycardia or A-V block	Excessive tachycardia; myocardial ischemia
Diltiazem	10–20 mg iv bolus; then infusion at 5–15 mg/hr	Rate control	Hypotension; CHF
Esmolol	0.5 mg/kg bolus and infusion at 0.05 mg/kg/hr; ↑ by 0.05 mg/kg/hr every 5 mins	Rapid rate control	Bronchospasm; hypotension, exacerbation of CHF
Metoprolol	5 mg iv every 5 mins × 3	Rate control	Bronchospasm; hypotension, exacerbation of CHF
Digoxin	0.25 mg iv every 4–6 hrs up to 1 mg	Chronic AF	Delayed onset; arrhythmias, nausea, vomiting
Ibutilide	1 mg iv over 10 mins; may repeat × 1	Conversion of AF	QT prolongation; torsades de pointes
Amiodarone	150 mg iv over 10 mins, then 1 mg/min × 6 hrs, then 0.5 mg/min	Refractory VT or VF; rate control and conversion of AF	Occasional mild hypotension with bolus; heart block

SVT, supraventricular tachycardia; A-V, atrioventricular; CHF, congestive heart failure; AF, atrial fibrillation; VT, ventricular tachycardia; VF, ventricular fibrillation.

of precipitating rapid atrial fibrillation with conduction down the accessory pathway.

Approximately 30% of patients with paroxysmal supraventricular tachycardia will be found to have a concealed accessory pathway (no antegrade conduction) between the atrium and the ventricle (12). Conduction during tachycardia occurs retrograde along the bypass tract and antegrade down the A-V node; the QRS complex is narrow. This tachycardia is called orthodromic A-V reciprocating tachycardia (12). When antegrade accessory pathway conduction is present (Wolff-Parkinson-White syndrome) a delta wave can be observed and the baseline QRS complex is wide. Most tachycardias will still be orthodromic. Preexcited tachycardia may also develop with antegrade accessory pathway conduction and normal retrograde conduction (via the His-Purkinje system and A-V node; these are called antidromic tachycardias (12). Tachycardias that use accessory pathways for both their antegrade and retrograde limbs may occur in patients with multiple accessory pathways. Therapy for orthodromic tachycardias includes vagal maneuvers, adenosine, and β blockers. Calcium-channel blockers should be used very cautiously and digoxin must be avoided because of the potential for developing preexcited atrial flutter or fibrillation (12). For antidromic accessory pathway reentrant tachycardias or wide complex tachycardias suspected to be supraventricular, intravenous procainamide or amiodarone are the drugs of choice (15). In both circumstances, cardioversion is indicated for hemodynamic collapse.

Atrial flutter or fibrillation in the setting of a manifest accessory pathway is a dangerous situation caused by the potential for extremely rapid antegrade conduction down the accessory pathway with resultant rapid ventricular rates. In this situation, the ventricular rate is modulated by competition between A-V nodal conduction and conduction down the bypass tract. Intravenous digoxin, diltiazem, and verapamil block the A-V node and can thus, increase preexcited ventricular rates and lead to the potential for ventricular fibrillation (16). Digoxin may decrease antegrade accessory pathway refractory periods (in about one-third of patients), further increasing the risk of cardiac arrest. Drugs that slow conduction down the accessory pathway, such as procainamide and amiodarone can be used. Cardioversion is often necessary to restore hemodynamic stability promptly.

ATRIAL FIBRILLATION AND FLUTTER

As noted, supraventricular arrhythmias are common after noncardiac surgery. The precise frequency rate is highly dependent on the definitions used, on the intensity of monitoring, and on whether data from the intraoperative period are included. Of these, atrial fibrillation is by far the most common arrhythmia with the potential for serious consequences. In one prospective series of 916 patients >40 yrs old undergoing major noncardiac surgery, the frequency rate of supraventricular tachycardia was 4%; atrial flutter and fibrillation accounted for 63% of these arrhythmias (8).

Atrial fibrillation and flutter are even more common after cardiac surgery, with rates ranging from 12% to 40% after coronary artery bypass surgery (17, 18). The frequency rate is even higher after valve replacement, and may be as high as 60% (19). Because of its frequency, potential morbidity, and association with increased length of stay, atrial fibrillation has been well studied after cardiac surgery. Given the fact that many, if not most, patients with atrial fibrillation after noncardiac surgery will have underlying cardiac disease, it seems reasonable to extrapolate some of the findings in patients with atrial fibrillation after cardiac surgery to the setting of noncardiac surgery.

Risk factors associated with an increased risk of postoperative atrial fibrillation after cardiac surgery include increased age, postoperative electrolyte shifts, pericarditis, a history of preoperative atrial fibrillation, a history of congestive heart failure, and chronic obstructive pulmonary disease (4, 20, 21). Most of these risk factors are probably also predictive after noncardiac surgery, although this has been less well studied. Atrial fibrillation generally occurs 2–4 days after open-heart surgery. Episodes tend to be transient, frequent, and recurrent.

For stable patients with atrial fibrillation lasting >15 mins, initiation of therapy to control the ventricular rate is recommended. Intravenous β blockers are a logical choice in postoperative patients with high sympathetic tone. Intravenous calcium-channel blockers and intravenous amiodarone are alternatives for rate control (Table 3). Digoxin has a delayed onset of action and works by increasing vagal tone, and thus, is typically ineffective for acute rate control in this setting.

Intravenous ibutilide, a recently approved class III antiarrhythmic agent effective in termination of atrial fibrillation and flutter, is an alternative for acute cardioversion in the hemodynamically stable patient (22). Ibutilide is administered as a 1 mg bolus >10 mins, which can be repeated if necessary, provided the

QT interval is not excessively prolonged. QT prolongation induced by ibutilide is associated with a risk of torsade de pointes, and therefore, continuous electrocardiogram monitoring is recommended for 4 hrs after ibutilide administration or until the QT interval returns to normal (4).

Atrial fibrillation with a rapid ventricular response can worsen diastolic filling caused by decreased filling time and loss of atrioventricular synchrony. Associated symptoms may include chest pain, shortness of breath, and dizziness. For patients with hypotension, pulmonary edema, or severe unstable angina, urgent cardioversion is indicated.

Most episodes of postoperative atrial fibrillation are self-limiting, although they tend to be recurrent. Atrial fibrillation persisting for >48 hrs is associated with a increased risk of stroke or transient ischemic attack (23). Thus, after 48 hrs of atrial fibrillation, anticoagulation should be considered, weighing potential benefits against the risk of postoperative bleeding.

The high frequency rate of postoperative atrial fibrillation after cardiac surgery, along with the observation that its onset is generally late, has fueled interest in prophylactic measures to prevent its development. β blockers are effective in preventing atrial fibrillation and other supraventricular arrhythmias after cardiac surgery (19). Whereas the low frequency rate of atrial fibrillation may mitigate against prophylactic therapy after noncardiac surgery, these data do support an antiarrhythmic effect of β blockers given to high-risk patients for other indications (24). Prophylactic use of sotalol, a class III antiarrhythmic agent with β blocking activity, decreases the frequency rate of atrial fibrillation after bypass surgery (25). Several studies have examined the effects of amiodarone on the frequency rate of atrial fibrillation after cardiac surgery. Both oral amiodarone started 1 wk before bypass surgery (26) and intravenous amiodarone given shortly after the completion of surgery (27, 28) reduce the frequency rate of atrial fibrillation after cardiac surgery. Extrapolation to the entire population of patients undergoing noncardiac surgery may not be warranted, but these studies do support the potential efficacy of these modalities in selected patients with heart disease undergoing noncardiac procedures.

VENTRICULAR TACHYCARDIAS

Ventricular tachyarrhythmias can be classified as benign or malignant. The chief distinction, in addition to duration and hemodynamic consequences, is the presence of significant structural heart disease. This distinction is important when evaluating premature ventricular contractions and nonsustained ventricular tachycardia. In patients without structural heart disease, the risk of sudden death or hemodynamic compromise is minimal. Therapy is rarely necessary in the absence of symptoms. In patients with severe coronary artery disease, history of myocardial infarction, or cardiomyopathy, these arrhythmias may represent harbingers of more malignant ventricular tachyarrhythmias meriting prompt and thorough assessment. An expeditious evaluation for and reversal of precipitating factors, such as ischemia and electrolyte abnormalities is indicated. Although specific antiarrhythmic therapy may not be needed in all patients, empirical β blockade and nitroglycerin should be considered.

A recent clinical trial evaluated empirical β blockade in patients undergoing noncardiac surgery. Two hundred high-risk patients (defined as having two or more of the following risk factors: age >65 yrs, hypertension, currently smoking, cholesterol >240, and diabetes) undergoing noncardiac surgery were randomized to atenolol or placebo, started preoperatively and continued until hospital discharge (24). Atenolol produced a 15% absolute reduction in the combined end point of myocardial infarction, unstable angina, congestive heart failure requiring hospital admission, myocardial revascularization, or death at 6 months, and reduced mortality at both 6 months and 2 yrs (24). Although this trial does not directly address the use of β blockade as postoperative antiarrhythmic therapy, the findings tend to support empirical postoperative initiation of atenolol in high-risk patients with arrhythmias.

Serious ventricular arrhythmias are becoming uncommon after cardiac surgery, and are even less common after noncardiac surgery. In one study, the frequency rate of sustained ventricular tachycardia and ventricular fibrillation after coronary artery bypass surgery was reported to be 1.2% (29), with most cases occurring on the first postoperative day. Antecedent causes included perioperative infarction, hypoxia, medications, hypoka-

Because the length of the QT interval is proportional to the length of the RR interval, patients with acquired QT prolongation and torsade de pointes may benefit from use of isoproterenol or temporary pacing to increase the ventricular rate.

lemia, and hypomagnesemia. Other series have also found similarly low rates of *de novo* ventricular arrhythmias after cardiac surgery (30). Serious ventricular arrhythmias after noncardiac surgery most commonly occur in the setting of postoperative infarction and underlying structural heart disease (31). In this context, they may be even more worrisome than after cardiac surgery because underlying coronary artery disease may not have been corrected. Nonetheless, definitive prognostic data are sparse.

Sustained monomorphic ventricular tachycardia is a reentrant rhythm most commonly occurring late (>48 hrs) after myocardial infarction or in the setting of cardiomyopathy. Acute ischemia, a frequent cause of polymorphic ventricular tachycardia or ventricular fibrillation, does not commonly produce sustained monomorphic ventricular tachycardia. Initial management of sustained monomorphic VT depends on its rate, duration, and the extent of underlying cardiac disease. Unstable ventricular tachycardia (presenting with either hemodynamic compromise or angina) is an indication for direct current cardioversion. For hemodynamically stable patients felt to have a risk of imminent circulatory collapse, therapy with lidocaine can be initiated. Procainamide can be considered an alternative, but its use is limited by hypotension and the potential for negative inotropic effects. Amiodarone can be administered intravenously and represents a good alternative in patients with compromised left ventricular function.

As noted, polymorphic ventricular tachycardia most commonly occurs in

the setting of myocardial ischemia or infarction. Although it is often faster than sustained monomorphic VT and can lead to hemodynamic instability, most episodes of polymorphic VT terminate spontaneously. Initial management for polymorphic VT is similar to that for monomorphic VT, with direct current cardioversion when necessary and antiarrhythmic drugs that do not prolong the QT interval for sustained episodes (32). In addition, therapy for coronary disease with β blockers and intravenous nitroglycerin should be initiated. If these arrhythmias are unremitting in the setting of coronary artery disease, intra-aortic balloon pumping and coronary revascularization should be considered.

Torsade de pointes is a syndrome consisting of polymorphic VT associated with QT prolongation. Acquired QT prolongation can result from drugs (most notably type IA or type III antiarrhythmic agents, tricyclic antidepressants, nonsedating antihistamines, erythromycin, pentamidine, and azole antifungal agents), electrolyte abnormalities (most notably hypomagnesemia and hypokalemia), and other conditions including hypothyroidism, cerebrovascular accident, and liquid protein diets (33). Empirical magnesium should be given to all patients with suspected torsade de pointes, because the risk is low and the potential benefit high. Because the length of the QT interval is proportional to the length of the RR interval, patients with acquired QT prolongation and torsade de pointes may benefit from use of isoproterenol or temporary pacing to increase the ventricular rate (34).

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