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Review Article

Perioperative Cardiac Dysrhythmias: Diagnosis and Management

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Since a review by Katz and Bigger in 1970, [1] no discourse on recognition and management of perioperative cardiac dysrhythmias has appeared in Anesthesiology, despite important advances in diagnosis and treatment. Cardiac electrophysiology and mechanisms for perioperative dysrhythmias were reviewed in the journal by Atlee and Bosnjak in 1990. [2] Advances in diagnosis and management of dysrhythmias since 1970 include 1) elucidation of mechanisms for most clinical dysrhythmias, with application of this knowledge to more effective preventive, remedial, and specific management with drugs or devices (pacemakers, cardioversion) [3-14]; 2) availability of new drugs for intravenous use, including adenosine, amiodarone, bretylium, diltiazem, esmolol, ibutilide, and verapamil; 3) recognition of the potential for lethal ventricular prodysrhythmia with antidysrhythmic drugs [6,13,15-20]; 4) technologic advances with noninvasive transcutaneous and transesophageal pacing [21-23]; and 5) use of surgical or catheter ablation of dysrhythmic foci or reentrant pathways for management of atrial and ventricular tachydysrhythmias. [24]

The purpose of this article is to inform anesthesiologists of developments pertinent to recognition and management of perioperative cardiac dysrhythmias in perioperative settings. Therefore, the emphasis is on acute dysrhythmia management. However, longer term measures also are mentioned because they may affect perioperative management. Management for patients with pacemakers or antitachycardia devices is discussed elsewhere.

[25,26] Finally, a pathophysiologic approach to management, with specific targets for drug-device therapy, is stressed. [3,27]

Perspectives

Incidence and Outcomes

The incidence of intraoperative dysrhythmias depends on the definition (e.g., any dysrhythmia vs. only potentially dangerous ones), continuous surveillance versus casual observation, patient characteristics, and the nature of surgery. [2] Incidences greater than 90% are possible with continuous monitoring in patients having cardiothoracic surgery. The Multicenter Study of General Anesthesia reported a 70.2% incidence of tachycardia, bradycardia, or dysrhythmias in 17,201 patients having general anesthesia for a variety of surgical procedures. [28,29] Most patients (90.7%) were American Society of Anesthesiologists (ASA) physical status 1 and 2. Severe adverse outcomes, defined as need for significant management (i.e., antidysrhythmic drugs, electrical devices, or cardiopulmonary resuscitation) with or without chronic disability or death resulting from dysrhythmias, occurred in 1.6% of these patients. [28,29] Comparable data are not available for patients in postanesthetic or surgical intensive care units.

Physiologic Impact

The ventricular rate and duration of tachydysrhythmias and myocardial functional impairment are the most important factors determining outcomes. Rapid ventricular rates with tachycardia can cause diastolic encroachment, reduce cardiac output, and result in hypotension or myocardial ischemia. [30,31] Chronic tachycardia may produce tachycardiomyopathy and cardiac failure. [32,33]

Bradydysrhythmias, especially with loss of atrial transport function, may impact severely on patients with systolic or diastolic ventricular dysfunction. [34-37] Cardiac output more than doubled after atrial pacing for overdrive suppression of AV junctional rhythm (AVJR) in some patients with presumed left ventricular dysfunction. [38]

Genesis of Dysrhythmias

Perioperative dysrhythmias are more likely to occur in patients with structural heart disease, and the initiating factor is often transient imbalance (Table 1). [39] Therefore, structural heart disease and imbalance provide a substrate for reentry, triggered activity, or abnormal automaticity. [3,40] For example, patients with chronic coronary artery disease have elements of normal and abnormal conduction interacting with nonuniform myocardial refractoriness. [40] They also may have occasional or frequent ventricular extrasystoles as a manifestation of

their heart disease. The addition of transient imbalance (e.g., ischemia, catecholamines, or electrolyte abnormalities) may be all that is necessary to trigger lethal ventricular tachydysrhythmias. [40-42]

Table 1

Approach to Management

If transient imbalance alters normal or abnormal myocardium to aggravate dysrhythmias, then management should include treatment for structural heart disease and corrective intervention for obvious imbalance. This may reduce or even remove the need for specific therapy with drugs or devices, with potential adverse effects of their own. However, dysrhythmias that cause severe circulatory insufficiency or are inherently unstable require immediate cardiac electroversion, followed by indicated antidysrhythmics and remedial measures to prevent recurrences.

A group of leading cardiologists (The Sicilian Gambit) has suggested a pathophysiologic approach to management of antidysrhythmic drug treatment. [3,27] How this may be applied to management of perioperative AVJR is illustrated in Figure 1. In this example, AVJR occurs as a result of altered normal automaticity or depolarization-induced (abnormal) automaticity. [2,4,39,43] Specific antidysrhythmic therapy is targeted at suppressing enhanced automaticity of the junctional pacemakers (overdrive pacing, beta-blockers), increasing sinus node automaticity (anticholinergics, beta$_1$ agonist), or opposing myocardial ischemia (beta-blockers, nitroglycerin, nicardipine).

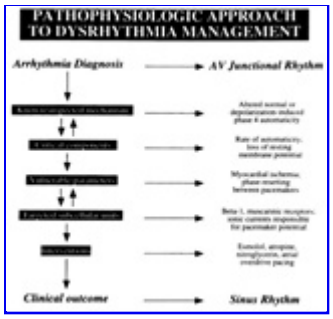

Figure 1

Diagnosis of Dysrhythmias

A systematic approach to electrocardiographic (ECG) diagnosis (Table 2) can improve the accuracy and likelihood of success with subsequent management. Especially important are the rate and regularity of rhythm, appearance of P waves, relation of P waves to QRS complexes, and finding the cause for widened or bizarre QRS complexes (ventricular

aberration).

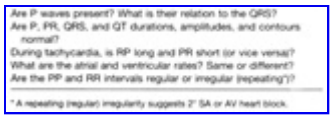
Are P waves present? What is their relation to the QRS?
Are P, PR, QRS, and QT durations, amplitudes, and contours normal?
During tachycardia, is RP long and PR short (or vice versa)?
What are the atrial and ventricular rates? Same or different?
Are the PP and RR intervals regular or irregular (repeating*)?
* A repeating (regular) irregularity suggests 2° SA or AV heart block.

Table 2

Rate and Regularity of Rhythm

The rate of normal sinus rhythm is between 60 and 100 beats/min in adults. Slower or faster rates are defined as bradycardia or tachycardia, respectively. The definition of clinically relevant bradycardia or tachycardia should be individualized. For example, resting sinus bradycardia of 40 beats/min can be normal in trained athletes, whereas sinus rate of 100 beats/min during sleep or the inability to increase rate above 100 beats/min during strenuous exercise is abnormal.

Sinus rhythm is not precisely regular. When PP cycle length variation exceeds 10% with breathing, respiratory (phasic) sinus dysrhythmia exists. Similar variation not related to breathing (nonphasic sinus dysrhythmia) is abnormal and is a result of sinus node dysfunction, aging, or digitalis intoxication. [31,44] The absence of a recurring pattern of QRS complexes (i.e., "irregularly" irregular [45]) with nonapparent P waves suggests underlying atrial fibrillation, even with QRS aberration (widened QRS complexes). However, even monomorphic ventricular tachycardia (VT) can be somewhat irregular, with up to 20 ms RR interval variation during VT considered normal. [45-47] RR variation of more than 20 ms during monomorphic, wide QRS tachycardia suggests atrial fibrillation.

Appearance of P Waves and Relation to QRS

Complex tachydysrhythmias often require more than two simultaneous leads for diagnosis. At least one of these should be a lead that maximizes P waves (II, III, aVF, or V1) to ascertain the relationship between atrial and ventricular depolarizations. P waves are best seen by recording from esophageal (Figure 2), transvenous, or epicardial pacing leads. [23,48,49] Strip-chart recordings or 12-lead ECG may also be needed for diagnosis and are necessary to document responses to management.

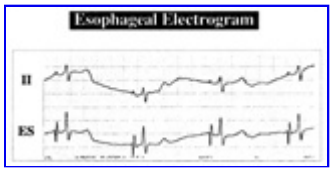

Figure 2

Atrial depolarization normally proceeds caudad and to the left, producing upright P waves in the inferior limb leads (II, III, aVF) and a biphasic P wave in V1. The PR interval is fairly constant, with the range 0.11-0.21 s in healthy adults at rest. Longer or shorter PR intervals

suggest AV heart block or ventricular preexcitation, respectively. Biphasic, isoelectric, or inverted P waves in the inferior limb leads with a normal PR interval suggest atrial enlargement or ectopic atrial depolarization. Inverted P waves after the QRS complex (with constant RP interval) in the inferior limb leads suggest retrograde atrial activation from AV junctional or ventricular beats. Finally, when the atria and ventricles are controlled by independent pacemaker foci with similar rates, P waves appear to "march in and out" of the QRS complex. This is termed isorhythmic AV dissociation. [31,50]

Cause for Widened QRS Complexes

Widened (> 0.10 s) or bizarre QRS complexes result from abnormal ventricular conduction of supraventricular beats (ventricular aberration) or abnormal ventricular activation with ventricular beats. This distinction is important. Aberrant beats are easily misdiagnosed and mismanaged as ventricular dysrhythmias. The term QRS aberration does not apply to fixed ventricular conduction defects (i.e., fascicular or bundle branch block). QRS aberration has many possible causes, [31,51] all of which are explained by differential or altered refractoriness of the ventricular specialized conducting tissues. For example, the higher incidence of right versus left bundle branch block pattern QRS aberration with premature supraventricular beats or tachycardia (SVT) is explained by longer refractoriness of the right bundle branch. Also, QRS aberration is more likely with slow heart rates or when a short cycle beat follows a long cycle beat in patients with atrial fibrillation (Ashman phenomenon; Figure 3). Aberration caused by the Asmann phenomenon occurs because refractoriness is longer after the long cycle beat, so that the subsequent short cycle beat propagates, whereas the distal conducting system is still partially refractory. [45,46] In contrast, with abnormal ventricular activation, the QRS complex has about an equal likelihood of having right or left bundle branch block morphology. [47]

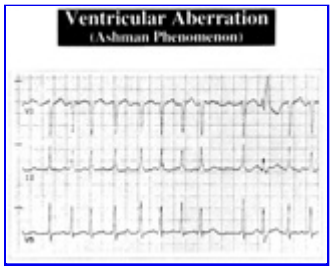

Figure 3

Pharmacologic Management

Antidysrhythmic Action

Cellular mechanisms for clinical cardiac dysrhythmias include altered normal automaticity of primary and latent pacemakers, abnormal automaticity in partially depolarized fibers, triggered activity from early or delayed after depolarizations, and reentny. [2,4,9]

Antidysrhythmic drugs oppose these mechanisms by altering pacemaker currents, reducing intracellular Ca^2+ overload, affecting Ca^2+ plateau or K sup + repolarization currents, or producing alterations in conduction and refractoriness that are nonconductive to reentry. [52-57] Examples of perioperative dysrhythmias with likely mechanism, desired antidysrhythmic drug action, and useful drugs are listed in Table 3.

Table 3

Prodysrhythmia

In addition to adverse side effects and toxicity, antidysrhythmic drugs may aggravate or provoke dysrhythmias at therapeutic concentrations, a phenomenon termed prodysrhythmia. [6,13,15,18] A classic example of prodysrhythmia is torsades de pointes with quinidine. [58] Prodysrhythmia excludes dysrhythmias resulting from drug toxicity or drug-caused bradycardia in patients with intrinsic sinus node dysfunction or impaired conduction. [13] Factors that can increase susceptibility to prodysrhythmia include structural heart disease, left ventricular dysfunction, ventricular preexcitation, QT interval prolongation, T wave alternans, myocardial ischemia, preexisting ventricular dysrhythmias, atrial flutter or fibrillation, and electrolyte imbalance. [6,13,15,18,54]

Ventricular prodysrhythmia have not been confirmed for beta-blockers (except sotalol) or calcium channel blockers (CCB) but may occur in up to 20% of patients with some class IC drugs. [58] Although lidocaine has caused ventricular prodysrhythmia in a canine infarction model, it does so only rarely in humans. [59]

Antidysrhythmic Drugs

Drugs under the Vaughan Williams classification [60,61] are discussed first, followed by unclassified adenosine and digitalis. Emphasized are parenteral drugs used for acute management of dysrhythmias. Dosing, indications, and precautions for these are summarized in Table 4. [58,62-64] For information on chronic management, including indications, adverse effects, and oral dosing, the reader can consult other works. [58,62-64]

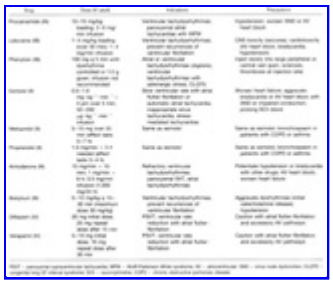

Table 4

Class IA drugs. Vaughan Williams class IA drugs (quinidine, procainamide, disopyramide) primarily block open Na sup + channels with intermediate time constants for recovery from block (300-1500 ms). [58,62-65] They also (especially quinidine) block K sup + repolarization currents (a class III action). Therefore, class IA drugs slow conduction, prolong action potential duration and repolarization, and increase effective refractoriness of atrial and ventricular myocardium. Because onset of block is rapid and because cardiac electrophysiologic (EP) effects are more pronounced at fast rates (use-dependence), class IA drugs are effective for suppressing reentrant atrial and ventricular dysrhythmias. [52,65] Class IA drugs slow SA node automaticity and increase AV node conduction time and refractoriness, but antimuscarinic actions (especially for quinidine and disopyramide) oppose these effects. Nevertheless, increased atrial refractoriness and antimuscarinic effects promote 1:1 AV conduction in patients with atrial flutter or increase ventricular rate in some patients with atrial fibrillation. Procainamide undergoes renal excretion (50-70%) or hepatic acetylation to N-acetylprocainamide (NAPA). NAPA has mostly class III activity, and toxic concentrations can produce severe ventricular prodysrhythmia. [58,62-65] Procainamide and NAPA concentrations should be monitored in patients with renal insufficiency or in those receiving chronic therapy. Quinidine and disopyramide cause significant vasodilation and negative inotropy, which makes them undesirable for intravenous use. Hypotension is also common with intravenous procainamide.

Class IB drugs. Class IB drugs (lidocaine, mexiletine, and tocainide) block inactivated and open Na sup + -channels. Phenytoin is classified by some as a Class IB drug. [52,64] However, much of its antidysrhythmic activity results from reduced central sympathetic efferent traffic (especially in patients with digitalis toxicity). [47,64,66] Class IB drugs have a rapid onset of action and short time constant for recovery from block (300-400 ms). [63,65,66] Consequently, they exert greater effects in depolarized (ischemic, hypoxic) tissues and at fast heart rates. [52,56,63,65,67] Cardiac EP effects also depend on the tissue studied and extracellular K sup + concentration. Because atrial action potentials are short and because Na sup + -channel block resolves between beats, class IB drugs are not effective against atrial dysrhythmias. [63,68] Class IB drugs decrease action potential duration and refractoriness in ventricular fibers as a result of block of slowly inactivating (plateau phase) Na sup + current or by increasing K sup + repolarization currents. [63,66] Also, lidocaine hyperpolarizes Purkinje fibers depolarized by stretch or low extracellular K sup +. [69] If so, the resultant

increased conduction velocity and shortened refractoriness could oppose reentry by removing an area of unidirectional conduction block or facilitate reentry by converting an area of bidirectional to unidirectional conduction block. At usual concentrations, class IB agents do not have significant EP effects on SA or AV nodal tissue. However, they can suppress the rate of sinus or escape rhythms in patients with intrinsic sinus node dysfunction (SND) but not the rate of depolarization-induced (abnormal) automaticity. [63,66] Lidocaine is commonly used for management or prophylaxis of ventricular dysrhythmias after open heart surgery, with acute myocardial infarction, and in patients with chronic ischemia. [58,63,64,66] Tocainide and mexiletine, structural analogs of lidocaine, have similar antidysrhythmic activity but better oral absorption. Although sometimes used alone to suppress ventricular dysrhythmias, they are probably more effective combined with a class 1A drug. [58,63,66]

Class IC drugs. Available class IC drugs (flecainide, moricizine, propafenone) are approved for oral use only. All block Na channels in the open state (with long recovery times from block, 1.5-10 s) and Ca^2+ and K sup + currents. [52,63,65] Therefore, they suppress automaticity of the SA node, slow AV node, His-Purkinje, and ventricular conduction times and prolong the PR, QRS, and QT intervals. They have little effect on action potential duration or refractoriness at normal heart rates. However, flecainide prolongs atrial action potential duration more at faster, compared with slower, heart rates, which makes this drug particularly useful for management of paroxysmal atrial tachydysrhythmias. [63,65,66] This contrasts with quinidine, which causes more prolongation at slower heart rates. [70] The efficacy of class IC drugs to suppress ventricular dysrhythmias is low and risk of serious toxicity high, so that these drugs are rarely used for this indication today. [63,66] The Cardiac Arrhythmia Suppression Trial (CAST) investigators demonstrated approximate threefold increased mortality rate as a result of dysryhthmias with class IC drugs for suppression of chronic, asymptomatic, nonsustained ventricular dysrhythmias. [16,17] The risk of prodysrhythmia with class IC drugs appears to be greatest in patients with ventricular dysfunction, [13,15,66,71] and these drugs can exacerbate heart failure in these same patients. [58,64,72,73] Flecainide is often prescribed to prevent paroxysmal atrial tachydysrhythmias in patients without structural heart disease. [63,72,74,75] Propafenone is a weak Ca^2+ -channel blocker and apparently one fortieth as potent as propranolol is as a beta-blocker. [72,76-79] It is used to manage life-threatening ventricular dysrhythmias and to suppress paroxysmal atrial tachycardias in patients with and without Wolff-Parkinson-White (WPW) syndrome. [72,77-79] Moricizine is approved only for management of life-threatening ventricular dysrhythmias; its usefulness for suppression of supraventricular tachydysrhythmias remains undetermined. [72]

Class II drugs. Class II drugs are beta-adrenergic blockers. Beta-Blockers approved for antidysrhythmic use include acetbutolol, esmolol, metoprolol, and propranolol. [80] Of these, acetbutolol and esmolol are approved for oral and intravenous use only, respectively.

Metoprolol and propranolol can be administered by either route. Sotolol, one fourth as potent as propranolol as a beta-blocker, has primarily a class III action. [58,63-65,81,82] Whereas cardiac EP effects of beta-blockers result primarily from specific block of catecholamine actions, at least some block Na sup + channels (acebutolol, labetalol, metoprolol, oxyprenolol, pindolol, propranolol, and others) have intrinsic agonist activity (acebutolol, labetalol, oxyprenolol, pindolol). [62,63,80,83] The latter may reduce effectiveness against catecholamine-mediated dysrhythmias. The cardiac EP effects of beta-blockers are greatest when catecholamines or digitalis excess contribute to dysrhythmias. EP effects include slowing the rate of SA node and latent pacemaker automaticity, preventing depolarization-induced (abnormal) automaticity and increasing sino-atrial (SA) and AV node refractoriness and conduction times. Because catecholamines are elevated in patients with congestive heart failure, undergoing cardiac surgery, or with myocardial infarction, beta-blockers have been used to manage related supraventricular or ventricular dysrhythmias with varying success. [58,64,80] However, they can worsen heart failure or cause peripheral vasoconstriction or coronary spasm (as a result of unopposed alpha stimulation). Finally, there is substantial evidence that beta-blockers reduce mortality from dysrhythmia or reinfarction after acute myocardial infarction. [80,84-86]

Class III drugs. Class III antidysrhythmics block K sup + repolarization currents and increase action potential duration and refractoriness in atrial and ventricular muscle and in Purkinje fibers. [52,56,63,87,88] Class III drugs approved for intravenous use include amiodarone, bretylium, and ibutilide.* Sotalol is approved only for oral use in the United States. Amiodarone exhibits all four antidysrhythmic class actions. [52,56,63,87,88] Class I effects contribute to slowing the rate of ventricular tachycardia and to improving hemodynamic tolerance to the disturbance. Class II effects slow the rate of automaticity and increase AV node conduction time and refractoriness. They may also protect against sudden death after myocardial infarction. [89-91] Class III effects increase atrial, accessory pathway (AP), and ventricular refractoriness, useful for managing PSVT or atrial flutter-fibrillation in WPW patients. [88,92] Amiodarone's class IV action slows the ventricular rate with atrial flutter-fibrillation, opposes paroxysmal SVT (PSVT) caused by AV node reentry, and possibly suppresses torsades de pointes as a result of early afterdepolarizations. [53] Oral amiodarone may cause sinus arrest, AV heart block, or severe circulatory compromise in the presence of volatile anesthetics. [93-96] Amiodarone was recently approved for intravenous use against drug-refractory ventricular tachydysrhythmias. One study found comparable efficacy but better tolerance with intravenous amiodarone compared with bretylium for destabilizing ventricular dysrhythmias. [87] Although the manufacturer's suggested intravenous dose is 1000 mg/24 h (Table 4), a recent study of 273 patients found no statistically significant difference in success rates or incidence of complications with 500, 1000, and 2000 mg/24 h dosing. [97] However, there was a trend toward increased antidysrhythmic response with larger doses. [97] Infusing amiodarone too rapidly can precipitate heart failure in patients with poor left ventricular function.** Amiodarone's multiple class actions suggest possible

efficacy for other refractory dysrhythmias. It has been used intravenously in Europe with success comparable with class IC drugs for pharmacologic conversion of recent onset atrial fibrillation. [98,99] However, another study suggests it is no more effective than placebo for this purpose. [100]

Bretylium concentrates selectively in sympathetic ganglia and postganglionic nerve terminals. [63,64,82] Initially, it releases norepinephrine (and thereby may aggravate dysrhythmias) and then prevents norepinephrine release by depressing sympathetic nerve terminal excitability. It does not deplete catecholamines, interfere with sympathetic transmission, or decrease adrenergic responsiveness. Bretylium is preferred management for ventricular fibrillation, especially in those with acute myocardial infarction, and for therapy of recurrent VT that has not responded to lidocaine, procainamide, or $MgSO_4$. [101] Bretylium has not been used for acute supraventricular tachydysrhythmias.

Sotalol has no intrinsic sympathomimetic or Na sup + channel blocking activity; otherwise, its cardiac EP actions are consistent with beta-blockade and class III activity. [56,63,82,102-106] Oral sotalol is approved for chronic management of ventricular dysrhythmias, although it is also effective against supraventricular tachydysrhythmias. [55,82,102] Intravenous sotalol has been used for prophylaxis and management of SVT after coronary bypass surgery. [107,108] Others have not found sotalol effective for this indication. [109] Sotalol's untoward EP effects are related to beta-blockade (dose-related) and class III actions (torsades de pointes). [82] The incidence of ventricular prodysrhythmia with sotalol is similar to that with class 1A and 1C drugs. [82]

Class IV drugs. Class IV drugs are CCBs, but among these, only diltiazem and verapamil are effective for antidysrhythmic use. CCB delay recovery from inactivation of the L-type cardiac Ca^2+ channel to slow SA rate and increase AV node conduction time and refractoriness. [110,111] Reflex actions of dihydropyridine CCB (e.g., nifedipine, nicardipine, nimodipine) reverse any direct depressant effects on SA or AV node tissue. [110,111] CCB are used primarily to terminate or suppress PSVT due to AV or SA node reentry, and to reduce ventricular rate with atrial flutter-fibrillation. [31,58,64,110] CCB also may be effective against multiform atrial tachycardia in patients with chronic pulmonary disease. [112] However, CCB should not be administered intravenously to WPW patients with atrial flutter-fibrillation because of danger of provoking ventricular fibrillation and probably should be avoided for chronic use in all WPW patients whose AP refractory periods are short or unknown. [113] This is because 1) increased AV node refractoriness with CCB encourages AV conduction over accessory pathways (AP); 2) CCBs do not increase AP conduction and refractoriness, or 3) AP conduction may be enhanced and refractoriness shortened by increased adrenergic activity during tachycardia or resulting from CCB-mediated vasodilation. CCBs are not effective against VT, except for VT caused by coronary spasm or by certain specific and uncommon types of sustained VT in patients without demonstrable heart disease. [110] Finally, a number

of animal studies have demonstrated severe myocardial depression, sinus arrest, or AV heart block when diltiazem or verapamil are administered during anesthesia with potent volatile agents. [114-118]

Adenosine. Adenosine has clinical cardiac EP effects similar to those of diltiazem and verapamil. It stimulates cardiac adenosine-1 receptors to increase K sup + current, shorten action potential duration, and hyperpolarize membrane potential. [58,63,119-121] Further, adenosine antagonizes catecholamine-stimulated adenylate cyclase to reduce cyclic AMP, inward Ca^2+ current, and pacemaker current. It has little effect on atrial, AP, His-Purkinje, or ventricular fibers. Adenosine is eliminated with a half-life of seconds by carrier-mediated cellular uptake. [63] Therefore, its cardiovascular effects are shortlived (less than 1 min). It is used to terminate PSVT, with efficacy comparable with CCB. [120] Adenosine may be useful for the differential diagnosis of wide or narrow QRS tachycardias with nonapparent P waves (Table 5). However, there should be strong suspicion as to a supraventricular origin for tachycardia. Wide QRS tachycardia in patients with structural heart disease should be presumed to be ventricular in origin and treated as such. [122,123] Adenosine may accelerate the ventricular rate with atrial fibrillation in patients with WPW and is ineffective for most VT (exception to be discussed). [8] Adenosine can also 1) initiate atrial fibrillation [119,121,124]; 2) accelerate the ventricular rate or cause bradyasystole with atrial flutter [125]; 3) initiate polymorphic VT [124,126]; and 4) cause transient asystole in patients receiving CCBs or beta-blockers or with SND. [119,121] The initial dose of adenosine (Table 6) can probably be halved with central bolus administration. [127] Methylxanthines inhibit adenosine action by binding to the adenosine-1 receptor, [63,119] so that the dose may have to be increased in patients receiving theophylline or using caffeine. Dipyridamole (adenosine uptake inhibitor) and cardiac transplantation (denervation hypersensitivity) potentiate adenosine's effects. [63,124]

Table 5

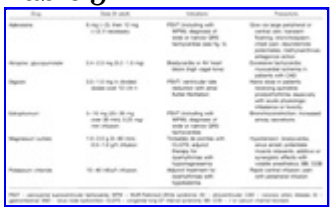

Table 6

Digoxin. Direct and autonomic effects contribute to digoxin's EP actions. [58,62,63] It inhibits the Na sup + -K sup + pump, thereby enhancing Na sup + -Ca^2+ exchange and increasing intracellular Ca^2+ to increase contractility. Toxic concentrations are associated with intracellular Ca^2+ -overload, the probable mechanisms for some ectopic dysrhythmias with digoxin toxicity. Indirect actions result from centrally mediated increased vagal tone

(therapeutic concentrations) and sympathetic tone (toxic concentrations). The former increases AV node refractoriness, and the latter shortens atrial and AP refractoriness. Thus, although digoxin slows ventricular rate with atrial flutter-fibrillation, it can also increase flutter rate. Further, it may dangerously accelerate ventricular rate with atrial flutter-fibrillation in patients with WPW.

Digoxin toxicity can cause virtually any cardiac rhythm disturbance, but some are more characteristic. [63] Atrial tachycardia with block is archetypal, but ventricular bigeminy, AV junctional tachycardias, bidirectional VT (rare), and AV block also occur. With advanced toxicity, severe hyperkalemia may develop as a result of Na sup + -K sup + pump poisoning, which can cause bradycardia unresponsive to pacing or atropine. Although brady- or tachydysrhythmias with early toxicity may respond to atropine, phenytoin, lidocaine, potassium, or MgSO sub 4, [128] patients with advanced toxicity should be treated with digoxin-specific Fab antibody fragments (Fab). Fab binds digoxin to enhance its renal excretion. [58,63,129]

Digoxin is used for management of heart failure and ventricular rate reduction with atrial flutter-fibrillation, especially in patients who may not tolerate beta-blockers or CCBs. However, in many patients with advanced heart disease, sympathetic activity is so high that therapeutic concentrations (0.5-2.0 ng/ml) may be ineffective for reducing rate.

Miscellaneous Drugs

Miscellaneous intravenous drugs useful for diagnosis and management of perioperative dysrhythmias include antimuscarinics, edrophonium, magnesium, and potassium. These will be reviewed, with suggested dosing, indications, and precautions presented in Table 6.

Antimuscarinics. Atropine or glycopyrrolate is used to increase heart rate and speed AV node conduction. Central nervous system side effects and modest cardioacceleration with scopolamine limit its usefulness. [130] Effects of atropine on lower pacemakers or ventricular conduction may be minimal or absent (Figure 4(B)) because vagal innervation is sparse. Atropine is most effective when bradycardia is caused by increased vagal tone, with effects most pronounced in young adults. [130] In infant and elderly populations, even large doses may fail to accelerate heart rate. [130] Small doses of atropine (< 0.6 mg) may cause transient slowing of rate in conscious adults. [130] Whether they do during general anesthesia is unknown. Palmisano et al. [131] found no decrease in heart rate with administration of atropine in infants and children anesthetized with halothane and N_2 O.

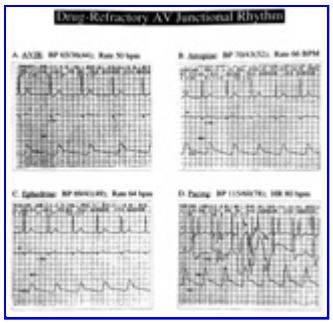

Figure 4

Edrophonium. There is precedent for use of edrophonium for diagnosis and management of dysrhythmias. [132-134] Its effects are a result of accumulation of acetylcholine at muscarinic and nicotinic sites. Sufficient doses produce the following effects: 1) stimulate muscarinic receptors at all cholinergic sites; 2) stimulate, then depress, nicotinic receptors in autonomic ganglia; and 3) stimulate, then depress central nervous system cholinergic receptors. [135] However, with suggested dosages (Table 6), muscarinic effects prevail. Edrophonium slows SA rate and increases AV-node conduction time and refractoriness but has no effect on atrial or ventricular conduction, contractility, or vascular tone. Caution is advised in patients with asthma or chronic pulmonary disease because of the possible occurrence of bronchoconstriction.

Magnesium sulfate. Magnesium (Mg^2+) strongly influences cardiac cell membrane ion transport function and is essential for the activity of many cellular enzymes. [12,63,136,137] Mg^2+ activates the Na sup + -K sup + exchange pump and contributes to maintaining the Ca sup 2+ gradient through Mg^2+ -dependent, Ca^2+ -AT-Pase. Dysrhythmias with Mg^2+ -deficiency resemble those with hypokalemia or digitalis toxicity. [12,63,64,136,137] Mg^2+ is used to manage supraventricular and ventricular tachydysrhythmias, including with digitalis. [128,138-140] With the possible exception of torsades de pointes, a cause-and-effect relationship has not been established for dysrhythmias with Mg^2+ deficiency. [63,141-146] Serum Mg^2+ levels can be normal despite significant intracellular deficits because only 5% of the available pool is extracellular. [136] $MgSO_4$ has been used to control ventricular dysrhythmias with heart failure or coronary artery disease, [10,11,146,147] PSVT, [148-150] and after open heart surgery. [151,152] Except for torsades de pointes with QT interval prolongation, [101,145] $MgSO_4$ s advised only as adjunct management for dysrhythmias with Mg^2+ deficiency (Table 7), hypokalemia, or digitalis toxicity [12,137,142]

Cachexia, starvation, malnutrition
Critical illness, cardiopulmonary bypass
Renal tubular damage, hemodialysis
Alcoholism, cirrhosis, diabetes
Myocardial infarction, chemotherapy
K-wasting diuretics, hypokalemia

Table 7

Potassium. Hypokalemia causes many clinically important dysrhythmias. [142,153,154] Hypokalemia commonly results from dialysis, thiazide or loop diuretics, mechanical hyperventilation, and management with insulin or beta$_1$ -adrenergic agonists. Cellular EP changes with hypokalemia provide a trigger (enhanced automaticity) and substrate (altered repolarization conductive to reentry) for tachydysrhythmias. [142] EP changes and dysrhythmias with hypokalemia are similar to those with digitalis or beta-adrenergic agonists. [142] However, there is no serum K sup + value below which there is undisputed risk of serious dysrhythmias. A serum K sup + value of 3.5 mEq/l may be low for a patient with chronic hypokalemia.*** Also, one should consider the risk of sinus node suppression, AV heart block, or ventricular fibrillation with overly aggressive repletion therapy. When is potassium replacement justified? Vitez et al. found no evidence for increased risk of intraoperative dysrhythmias with chronic hypokalemia K sup + 2.6-3.4 mEq/l. [155] However, there were limitations to this study: 1) K sup + values were not reconfirmed; 2) it was impossible to ascertain acuteness of hypokalemia; 3) the sample size was too small to detect differences between normo- and hypokalemic patients; and 4) most patients were at low risk for dysrhythmias. The risk of serious dysrhythmias with hypokalemia is increased in hypertensive patients receiving diuretics; with digitalis, acute myocardial infarction, high catecholamines, heart failure, malnutrition, and acute alcohol intoxication or withdrawal; and after cardiac surgery. [10,142] It is the author's belief that delay of anesthesia for acute potassium replacement is not required for hypokalemia (K sup +, 2.5-3.5 mEq/l) without other risk factors or characteristic dysrhythmias.

Pacing and Cardiac Electroversion

Cardiac pacing and electroversion (cardioversion or defibrillation) have advantages over drugs for the management of perioperative dysrhythmias. Their therapeutic effect is more prompt, and the "dose" (i.e., pacing mode, rate, current) is easier to titrate than with drugs. Also, drugs may not produce the desired effect or aggravate dysrhythmias. Finally, drug effects may last longer than required. Nonetheless, there are recognized disadvantages to pacing, including risk of sepsis, hemorrhage, or direct myocardial injury with invasive methods, stimulation of tachydysrhythmias, and inability to pace some patients. Modalities for temporary pacing and indications for cardiac electroversion will be discussed. Perioperative treatment for patients with pacemaker or antitachycardia devices is discussed elsewhere. [156,157]

Temporary Cardiac Pacing

Epicardial, transvenous (endocardial), transcutaneous, and transesophageal routes are used for temporary pacing. Epicardial, endocardial, or transesophageal leads are also useful for ECG diagnosis because of their proximity to myocardium. Epicardial, atrial, ventricular, or dual-chamber pacing is used in cardiac surgery to increase heart rate, suppress bradycardia-dependent tachycardia, overdrive escape rhythms, suppress atrial or ventricular

extrasystoles, and to terminate reentrant SVT or atrial flutter. [48,158-160] Atrial or dual-chamber is preferred to ventricular pacing to preserve atrial transport function. [34-37] Endocardial atrial, ventricular, or dual-chamber pacing is used for most other temporary pacing. Pacing pulmonary artery or balloon-flotation electrode catheters that do not require fluoroscopy for positioning are preferred by many anesthesiologists. [48,161]

Noninvasive transcutaneous pacing (TCP) is used if invasive pacing is not feasible or is impractical. [48,161-166] Current adult advanced cardiac life support guidelines emphasize early use of TCP in emergency cardiac care, [101] but results are discouraging when not instituted early after cardiac arrest. [101,164,165] Limitations include 1) nonphysiologic (ventricular) pacing, [34,167] 2) inability to capture in some patients, 3) restricted access (sterile fields, patient position), and 4) discomfort in conscious patients. Transesophageal (TE) pacing probes are approved for atrial pacing. [49,161,168,169] TE ventricular pacing and electroversion are being investigated. [170-173],**** TE pacing has been used to manage bradycardia in adults, [38,174-178] bradycardia and tachydysrhythmias in neonates and children, [21,76,179] and reentrant SVT or atrial flutter. [180-185]

Cardiac Electroversion

Cardiac electroversion includes cardioversion (synchronized shocks) and defibrillation (nonsynchronized shocks). [64,186-189] Both use high-energy capacitor discharges to simultaneously depolarize a sufficient mass of myocardium to terminate dysrhythmias. Cardioversion is not indicated for automatic or triggered tachydysrhythmias (especially with digitalis) because it merely resets the cycle of automaticity or triggered activity and may initiate VF. The lowest possible energy shocks should be used to reduce the risk of myocardial injury. [64,101] External shocks of 25-50 J (internal, 5-20 J) will terminate most SVT or VT, although 50-200 J (internal, 10-50 J) is needed for atrial flutter-fibrillation. Current for defibrillation (external, 200-360 J; internal, 5-50 J) is higher because far more myocardium should be depolarized.

Specific Dysrhythmias

Patients requiring oral antidysrhythmics should be continued on therapy until the time of surgery. Consultation is advised for patients who require pacing-cardioverter devices (PCD) to suppress or terminate tachydysrhythmias. Initial management for perioperative dysrhythmias does not differ from other acute circumstances. With life-threatening circulatory compromise, prompt pacing or electroversion is required. Obvious imbalance should be corrected and management provided for underlying heart disease. Specific antidysrhythmic drugs are used to suppress dysrhythmias and prevent recurrences.

Sinus Node Dysfunction

Bradydysrhythmias associated with sinus node dysfunction (SND) include sinus bradycardia, sinus pause, sino-atrial block, and sinus arrest. [31,190,191] These rhythms may produce symptoms or result in the emergence of alternative pacemakers, producing a variety of ECG observations, including wandering atrial pacemaker or paroxysmal atrial tachycardia. In perioperative settings, these disturbances are often transient and often caused by autonomic imbalance as the result of an intervention (spinal or epidural anesthesia, laryngoscopy, surgical stimulation, and so on) or by the effects of drugs on the sinus node or perinodal tissue. [2,192,193] These rhythms require management if bradycardia compromises end-organ perfusion. $Beta_1$ agonists are more reliable and longer acting than antimuscarinic agents for increasing rate. Cardiac pacing is effective and may be initiated by either the transesophageal or transvenous route. [38,176,177] Patients scheduled for surgery who are known to have symptoms attributable to bradydysrhythmias associated with SND may have a prophylactic temporary pacemaker inserted before induction of anesthesia.

Management of the combination of bradycardia with paroxysmal tachydysrhythmias (bradytachy syndrome) is more challenging. Chronotropic drugs used to manage or prevent bradycardia may aggravate or produce more frequent episodes of sinus tachycardia or tachydysrhythmias (often atrial flutter or fibrillation). Treatment in these patients may include a beta-blocker for suppression of sinus tachycardia or appropriate antidysrhythmic drug for tachydysrhythmias and a permanent pacemaker for management of bradycardia. [31,190,191]

Atrial Tachycardia

Atrial tachycardia is a supraventricular tachycardia that originates in atrial muscle and does not include the sinus node or AV node. Uniform atrial tachycardia (UAT) is characterized by a single P-wave morphology, whereas multiform atrial tachycardia (MAT) has three or more distinct P-wave morphologies. Heart rates may range from 100-250 beats/min, and there may be rate-dependent PR interval variation. Varying degrees of AV block may be present with fast heart rates, especially in patients receiving digitalis or other drugs that increase AV node refractoriness and conduction time.

Although atrial tachycardia accounts for up to 20% of narrow QRS tachycardias in children, it is much less common in adults. [194,195] UAT may be observed in patients with or without structural heart disease; however, MAT is more commonly seen in acutely ill patients, in elderly patients, or in those with pulmonary disease.

Postulated mechanisms for UAT and MAT are similar and include enhanced normal or abnormal automaticity or triggered activity within the atrium. These mechanisms result in an incessant tachycardia with variation in rate depending on autonomic tone. Chronic atrial tachycardia may produce a cardiomyopathy. [196,197] When reentry is the cause (UAT), onset and termination of tachycardia are paroxysmal. A rare cause for UAT today is digitalis intoxication, wherein one may see 2:1, 3:1, or higher AV conduction ratios (previously called

atrial tachycardia with block).

Management of atrial tachycardia is twofold. First, controlling the ventricular rate, and second, identifying and removing the cause whenever possible. Short bursts of tachycardia (unless very frequent) generally do not require specific drug management. When definitive management is indicated, an intravenous beta-blocker or CCB may be tried. [31,194,195] These agents will reduce the ventricular rate and may suppress tachycardia. Unless caused by reentry, atrial tachycardias are not likely to be terminated by pacing or cardioversion. If digitalis intoxication is the cause, Fab antibodies are indicated. Amiodarone, flecainide, and magnesium (especially with hypomagnesemia) have been reported to terminate some types of atrial tachycardia. [112,194,198-201] However, experience with these agents is limited, and their effectiveness in the perioperative arena remains to be determined. The short duration of action of adenosine makes it ineffective for controlling ventricular rate. It may terminate atrial tachycardia because of reentry, although this is a rare mechanism for UAT.

AV Junctional Rhythm Disturbances

AV junctional rhythm is a nonparoxysmal, narrow QRS rhythm with retrograde or nonapparent P waves and a rate less than 70 beats/min (Figure 4, panels A-C). If faster, usually less than 130 beats/min, it is termed accelerated AVJR [202] or nonparoxysmal AV junctional (or nodal) tachycardia. [31,203] AVJR is common during general anesthesia and was observed in 6% of patients having noncardiac surgery in one series. [38] An important cause for accelerated AVJR is digitalis toxicity, recognized by regularization of the ventricular rate in patients with atrial fibrillation. [31,202] Accelerated AVJR occurs in up to 10% of patients with acute myocardial infarction and also in patients having cardiac surgery or with acute rheumatic fever. [202] AVJR can produce circulatory compromise as a result of nonsynchronized atrial contractions and bradycardia.

Esmolol has been effective treatment in some patients having coronary revascularization, [204] possibly because ischemia and catecholamines contributed to AVJR. Atropine or ephedrine may restore sinus rhythm in some patients, although these drugs may have no effect or may accelerate AVJR in others (Figure 4, panels B and C). Therefore, temporary pacing is advised, especially in patients with ischemic heart disease. AVJR and accelerated AVJR are suppressed by atrial or dual-chamber overdrive pacing (Figure 4, panel D). [38,176] With increased coronary perfusion, it is usually possible to terminate pacing within a short timespan.

Paroxysmal Supraventricular Tachycardia

AV node (Figure 5, panel A) and accessory pathways (AP) reentry (Figure 5, panel B), in about equal proportions, account for 85-90% of paroxysmal supraventricular tachycardia (PSVT). [31,205-207] Sinus node and intraatrial reentry (mechanisms for PAT discussed

previously) account for the remainder. Note that with orthodromic and AP antidromic reentry (Figure 5, panel B), the AP and AVN are obligate for sustaining reentry. APs are electrophysiologically similar to atrial muscle and can manifest or concealed. Manifested APs conduct anterogradely during sinus rhythm to preexcite the ventricles (short PR interval, delta wave). Patients with WPW syndrome have preexcitation and paroxysmal tachycardias. [208] Concealed APs conduct retrogradely during orthodromic AP reentry tachycardia (Figure 5, panel A) but not anterogradely to cause preexcitation. AP reentry accounts for up to 80%, atrial fibrillation for 15-30%, and atrial flutter up to 5% of tachydysrhythmias in patients with WPW. [31] Ninety to 95% of AP reentry is of the orthodromic variety. [5,206,209]

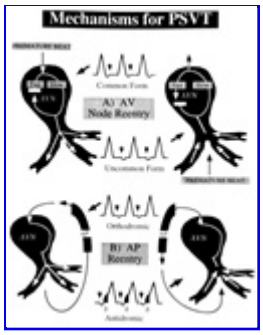

Figure 5

Paroxysmal supraventricular tachycardia caused by the common form of AV node and orthodromic AP reentry is characterized by its abrupt onset after a premature beat of atrial origin. The P wave of the premature beat initiating tachycardia usually has a different morphology than those during tachycardia, which is in contrast to AAT (discussed previously). [210] PSVT also terminates suddenly, often followed by a brief period of asystole or bradycardia. Finally, PSVT is a regular tachycardia with rates between 120 and 300 beats/min, and most patients are children or young adults without heart disease. [31,206,207,209]

Paroxysmal supraventricular tachycardia in patients without syndrome. After vagal maneuvers, drugs that increase AV node refractoriness (adenosine, CCB, esmolol, and other beta-blockers) are preferred initial therapy for any narrow QRS PSVT or PAT resulting from SA node or atrial reentry. [31,205-207,209,211-213] With circulatory insufficiency, prompt cardioversion is advised. Adenosine is preferred by some for initial drug treatment of PSVT because it: 1) has efficacy comparable with CCB; 2) is shorter-acting; 3) does not cause cardiovascular collapse or ventricular fibrillation if administered to a patient with VT or preexcited atrial tachycardia***** 4) has little effect on AP in patients with orthodromic AP reentry tachycardia; and 5) can slow retrograde AP or AV node conduction to terminate antidromic AV reentry PSVT. [121,205,211,214-218]

Narrow QRS paroxysmal supraventricular tachycardia in patients with Wolff-Parkinson-White syndrome. Caution is advised with using digitalis, diltiazem, or verapamil as a single drug for initial treatment of narrow QRS PSVT in patients known to have the

WPW syndrome. [5,31,206,207,209,219] All can accelerate the ventricular rate with atrial flutter or fibrillation, and some patients will develop atrial flutter or fibrillation during treatment for orthodromic AP reentry PSVT. Otherwise, initial treatment is similar to that for PSVT in patients without WPW (i.e., vagal maneuvers, adenosine, beta-blockers, cardioversion).

Wide QRS paroxysmal supraventricular tachycardia in patients with Wolff-Parkinson-White syndrome. Antidromic AP reentry PSVT is a preexcited (wide QRS) tachycardia with rates up to 250 beats/min. [209] Differential diagnosis includes all other causes for wide QRS tachycardia, including preexcited atrial tachycardias, any SVT with aberrant ventricular conduction, or ventricular tachycardia. Intravenous procainamide or amiodarone are initial drugs of choice for termination of known antidromic AP reentry PSVT or wide QRS tachycardia in a patient with patient, unless the patient needs immediate cardioversion. [31,209]

Atrial Flutter

Atrial flutter is a paroxysmal disturbance, usually lasting only minutes to hours before changing to sinus rhythm or atrial fibrillation. [31,220-222] Flutter waves, best seen in leads II, III, aVF, and V1, often have a saw-tooth appearance with no isoelectric line (Figure 6, panel A). The ratio of atrial to conducted beats is either fixed (Figure 6, panel A) or variable (Figure 6, panel B). Two types of atrial flutter are distinguished based on response to rapid atrial pacing. [49,158,159,184,221,223] Type I flutter (Figure 6, panels A and B; atrial rate < 340 beats/min) usually is, but faster type II flutter (Figure 6, panel C) is not, as it is interrupted by pacing.

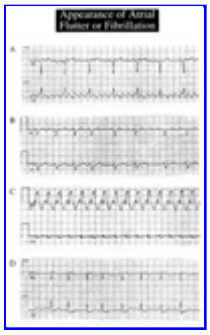

Figure 6

Atrial flutter is seen commonly in patients with chronic pulmonary disease, open heart surgery, dilated cardiomyopathy, inflammatory conditions affecting the heart, ethanol intoxication, and thyrotoxicosis. [31,220-222] With circulatory compromise, cardioversion (usually 50 J or less) or rapid atrial pacing (type I atrial flutter) are preferred initial management. [31,158,159,173,184,220,221,223] Drugs do not reliably convert flutter, and ventricular rate reduction can be difficult. If cardioversion fails to restore sinus rhythm or converts flutter to atrial fibrillation, higher energy shocks may be used or the patient may be left in atrial fibrillation. The latter may revert to sinus rhythm, or rate control may be easier.

In the author's experience, acute ventricular rate reduction with atrial flutter has been satisfactory with either esmolol or edrophonium (Table 4). Digoxin alone is not recommended for acute ventricular rate reduction. [66,220,221,224-226] CCBs or beta-blockers, alone or with digoxin, are more reliable, safer, and faster (Table 4). [66,220,221,224-226] However, CCB or digitalis can shorten atrial and AP refractoriness in patients with WPW (discussed previously). Class IA, IC, and III antidysrhythmics are used to prevent recurrences of atrial flutter, except that IA and IC drugs are contraindicated in patients with coronary disease and left ventricular dysfunction (ventricular prodysrhythmia). [227,228] Further, class IA drugs can accelerate AV conduction during atrial flutter as a result of antimuscarinic effects.

Atrial Fibrillation

Atrial fibrillation (Figure 6, panel D) is paroxysmal or chronic [31,92,220,222] and is the most common tachydysrhythmia in patients with the sick sinus syndrome. Other associations include hypertension, chronic pulmonary disease, coronary and other structural heart disease, and after cardiothoracic surgery. Paroxysmal atrial fibrillation may be poorly tolerated in patients with heart disease caused by diastolic encroachment and loss of atrial transport function. There is an approximate 5% annual risk of thromboembolism in patients with paroxysmal or recent onset (< 48 h) atrial fibrillation not receiving anticoagulant therapy. [227-229] Patients at increased risk are those with rheumatic heart disease, sick sinus syndrome, mural thrombi, left atrial enlargement, hypertension, left ventricular dysfunction, and aged more than 75 yr. [92,227,228] Anticoagulation is advised for 3 or 4 weeks before elective DC cardioversion for all patients with atrial fibrillation of more than 2 days or unknown duration, [31,92,220,222] although one study suggests that patients without echocardiographic evidence of mural thrombi can be safely cardioverted without previous anticoagulation. [230]

Atrial pacing is not feasible in patients with atrial fibrillation. Ventricular pacing is used for those with deleterious bradycardia. DC cardioversion (usually 100-200 J) is the most effective method to convert atrial fibrillation to sinus rhythm. It is most likely to be successful with paroxysmal or recent onset atrial fibrillation and small heart size. [31,92,220,222] Rarely are class IA drugs used for this purpose today, although they are prescribed to prevent recurrences. Class IC drugs, [92,222,226-228] amiodarone, [231] but not sotalol, [226] are also effective for preventing recurrences of atrial fibrillation.

Beta-Blockers, diltiazem, or verapamil, alone or with digoxin, are used for acute ventricular rate reduction with atrial fibrillation. [212,225,227,228,232-234] In one report, verapamil and esmolol were equally effective and safe for this purpose, but conversion to sinus rhythm was significantly more likely with esmolol. [235] Edrophonium has also been used for acute ventricular rate reduction. [47,132,133] However, edrophonium, digitalis, diltiazem, or

verapamil can all shorten atrial and AP refractoriness to increase the ventricular rates with atrial fibrillation or flutter in patients with WPW.

In patients with fast ventricular rates and hemodynamic compromise, prompt cardioversion is advised. [31,92,220,22,227,228] Procainamide, amiodarone flecainide, and propafenone are used to prevent recurrences. Any of these drugs may chemically convert some episodes of paroxysmal atrial fibrillation in those with WPW and in other patients. [98-100,107,231]

Ventricular Premature Beats and Non-sustained Tachycardia

Appearance and significance. A distinction is made between ventricular premature beats (VPB), NSVT, and sustained VT (lasting longer than 30 s). [6,15-17,30,31,40,47,236,237] Sustained VT is usually a manifestation of structural heart disease, has an adverse hemodynamic effect, and is managed with antidysrhythmic drugs or antibradycardia pacing to prevent recurrences. VPB and NSVT can occur without heart disease, often are hemodynamically inconsequential, and do not necessarily require antidysrhythmic drug treatment.

Prognosis. Although frequent VPB or NSVT may worsen long-term prognosis with structural heart disease, especially ischemic, dilated, or hypertrophic cardiomyopathy, they do not in patients with normal hearts. [236,237] This probably applies to patients having general anesthesia. The Multicenter Study of General Anesthesia reported a 6.3% incidence of ventricular dysrhythmias in 17,201 patients, [28] but only 107 of these suffered severe adverse outcomes (0.62% incidence). [29] Some adverse outcomes could have been related to presumptive or overly aggressive management of VPB or NSVT. Predictors for adverse outcomes included preexisting ventricular dysrhythmias, myocardial ischemia, chronic myocardial infarction, abdominal or cardiovascular surgery, and halothane versus fentanyl-based, enflurane, or isoflurane anesthesia. [29]

Suggested treatment priorities. When and how VPB or NSVT should be managed in perioperative circumstances is uncertain, even for patients having cardiac surgery. [238,239] Consideration should be given to the risks of management with antidysrhythmics compared with need for and likelihood of achieving a beneficial effect. Appearance and frequency of PVB or NSVT (e.g., Lown-Wolf grading system) [240] are probably insufficient criteria on which to base the decision to manage with antidysrhythmics. [241] Other factors to consider are hemodynamic impact, underlying pathophysiology, adequacy of treatment for concurrent disease, and whether VPB or NSVT initiate more ominous dysrhythmias.

Treatment. Specific drug management for VPB or NSVT is with beta-blockers (especially with adrenergic stress), lidocaine, and procainamide. An exception is rare, repetitive, idiopathic monomorphic VT, for which verapamil and adenosine are effective. [237,242,243] Specific management should probably not be provided for chronic PVB and NSVT in patients

with structural heart disease, unless they increase in frequency or duration, are associated with circulatory compromise, or initiate sustained dysrhythmias. The author has commonly observed a reduction in VPB and NSVT in patients during general anesthesia. Finally, the occurrence of frequent VES or NSVT in patients not known to have cardiac disease should alert anesthesiologists to occult disease, myocardial ischemia, imposed physiologic imbalance, or drug toxicity.

Accelerated Idioventricular Rhythm

Idioventricular rhythm (IVR) is a uniform, widened QRS rhythm with a rate of 60 beats/min or less. [31,244-246] It is termed accelerated IVR (or "slow" VT) when the rate is between 60 and 100 beats/min. A supraventricular pacemaker may intermittently capture all or a portion of the ventricles to cause capture or fusion beats, respectively. IVR is caused by a focus of altered normal or abnormal automaticity within the ventricles. Accelerated IVR occurs in approximately 12% of patients with acute myocardial infarction, but it does not usually progress to rapid VT or VF. [247] It also occurs with digitalis toxicity immediately after reperfusion of a previously occluded coronary artery, during cardiac surgery, and in patients with rheumatic fever or cardiomyopathy. [31,247] IVR may produce severe circulatory compromise in patients with ventricular dysfunction. Atropine has little effect on the rate IVR because the His-Purkinje system receives little vagal innervation. However, it may accelerate the atrial rate to suppress IVR. Caution is advised with lidocaine, procainamide, or beta-blockers if IVR is a tolerated escape rhythm. Temporary overdrive pacing by a route that preserves AV synchrony is advised for IVR with inadequate hemodynamics. With improved perfusion, sinus rhythm may be restored.

Sustained Monomorphic Ventricular Tachycardia

With sustained monomorphic VT (SMVT), uniform, widened QRS complexes (longer than 0.12 s) occur at rates between 100 and 250 beats/rain for more than 30 s. [31,46,47,248] SMVTs slower than 100 and faster than 250 beats/min are accelerated idioventricular rhythm and ventricular flutter, respectively. [447,66] At least 90% of patients with SMVT have coronary artery disease with severe left ventricular dysfunction caused by a previous infarction. [247-249] Acute ischemia, which often causes polymorphic VT or VF, seldom produces SMVT. [4,248] SMVT usually occurs late after infarction (more than 48-96 h) and is more likely a result of reentry. [4,248] Aside from SMVT with chronic coronary disease, other associations include dilated, hypertrophic, and infiltrative cardiomyopathies. Uncommon, specific types of SMVT include bundle branch reentry VT, idiopathic monomorphic VT (subdivided as right ventricular outflow tract VT and dysrhythmogenic right ventricular dysplasia VT), and idiopathic left ventricular VT. [31,46,242,248,250,251] There is no evidence that anesthesia aggravates SMVT in patients with coronary disease. Enflurane and halothane oppose reentrant VT in animal models of infarction. [252-255] Comparable data for newer or intravenous anesthetics are not available, nor are there reports of the effects of anesthetics

on clinical SMVT or other forms of VT.

Initial management of SMVT depends on the rate and duration of tachycardia, tolerance, and the extent of underlying heart or other major organ system disease. Identifiable imbalance should be managed. For patients not in danger of imminent circulatory collapse, treatment is started with lidocaine. If this is unsuccessful, some prefer DC cardioversion to bretylium or procainamide because of possible hypotension with these drugs. [101] With circulatory collapse, immediate cardioversion followed by intravenous lidocaine is recommended. [31,46,47,101,248] The initial shock should be 200 J, followed by 300 and 360 J if necessary. If lidocaine suppresses SMVT after DC conversion, the drug should be infused continuously. If it fails to suppress SMVT, then bretylium or procainamide should be administered, followed by continuous infusion of whichever drug is effective. Lidocaine is considered relatively ineffective for treating SMVT in patients with chronic infarction [46] because it has little EP effect on nondepolarized fibers. [256] Procainamide is more effective. [46,257] However, lidocaine has a clearly defined role for treating SMVT in patients with ischemia, with digitalis excess, or direct myocardial injury. [46] For refractory cases, amiodarone should be considered. [87,97] Finally, most patients with a history of circulatory collapse or sudden death caused by SMVT will receive chronic therapy with class I or III antidysrhythmic drugs or antitachycardia devices. [31,46,47,248]

Polymorphic Ventricular Tachycardia and Torsades de Pointes

Polymorphic ventricular tachycardia (PMVT) in the absence of QT interval prolongation is PMVT and that with QT interval prolongation is torsades de pointes (TdP). [31,47,258-261] PMVT and TdP are characterized by continuously changing, wide QRS complexes in any single ECG lead. The rate is often 100-200 beats/min, and prognosis is more ominous compared with SMVT. Although PMVT or TdP faster than 200 beats/min can be associated with hemodynamic collapse and degeneration into VF, the majority of episodes terminate spontaneously. [66]

Initial management for PMVT and SMVT are similar. [31,47,101,259,260] Because PMVT often occurs in association with coronary disease, indicated management for this is necessary (e.g., nitroglycerine, beta-blockers, circulatory assist devices, acute revascularization, and so on). However, if it is uncertain whether the patient has QT interval prolongation, then $MgSO_4$ should be tried. If PMVT is associated with bradycardia, isoproterenol or temporary pacing should be considered, except that isoproterenol is contraindicated with ischemic heart disease. [259]

Torsades de pointes can occur with acquired or congenital QT interval prolongation, and management differs. [258-261] With acquired QT prolongation (bradycardia- or pause-dependent TdP), temporary pacing or isoproterenol (not with ischemic heart disease [259]) to

increase the heart rate to 100-120 beats/min is part of acute management. In addition, causes for QT interval prolongation should be removed (Table 8). MgSO sub 4 is very effective in suppressing pause-dependent TdP. [146,258,259,262] Antidysrhythmic drugs that do not increase the QT interval (class IB, bretylium) are used in refractory cases. [7,258,259,263]

Drugs	Antidysrhythmic drugs (class 1A and 1C; class 3, amiodarone, sotalol; class 4, bepridil, lidoflazine); antibiotics (erythromycin, ampicillin, pentamidine, trimethoprim-sulfamethoxazole); phenothiazines; tricyclic and tetracyclic antidepressants; lithium; probucol; doxorubicin; ketanserin; nonsedating antihistamines, astemizole and terfenadine (especially in patients taking ketoconazole, itraconazole or erythromycin); steroids; organophosphate insecticides; diuretics; vasopressin; chloral hydrate poisoning; α blockers
Imbalance	Hypomagnesemia, hypokalemia, hypocalcemia; altered nutritional states, cachexia, liquid protein diets, starvation, anorexia nervosa, prenyamine; hypothyroidism
Disease	Hemorrhage; stroke, subarachnoid hemorrhage, encephalitis, sympathetic gangliontis; mitral valve prolapse, sinus node dysfunction, AV heart block, myocarditis, rheumatic fever, cardiomyopathies, coronary heart disease (especially, coronary artery spasm)
Other	Right radical neck dissection; carotid surgery; neurosurgical procedures; anaphylactic reactions; hypothermia

Table 8

For TdP with congenital QT interval prolongation (adrenergic-dependent TdP), beta-blockers and antidysrhythmic drugs that do not prolong the QT interval are used for initial and preventive therapy. [7,258-261] Left cervicothoracic sympathectomy is considered for patients whose symptoms are not controlled with beta-blockers. [264] A recent study suggests that verapamil also may be effective. [265] Antidysrhythmic drugs that prolong the QT interval (Table 8) are contraindicated. If bradycardia precludes management with beta-blockers, then temporary pacing may be required. Finally, because gene mutations affecting inactivation of Na sup + - and K sup + -currents have been linked to congenital QT prolongation, [266-268] future therapy (e.g., K sup + -channel openers pinacidil and nicorandil) may target these defects. [258,260,269,270]

Ventricular Flutter and Fibrillation

Ventricular fibrillation (VF) is the most common cause of sudden cardiac death. [31,101,271-275] Up to 75% of victims have coronary disease, and VF commonly develops after VT. The incidence of VF during contemporary anesthesia is unknown, but it is probably less than 0.62% incidence of severe adverse outcomes with ventricular dysrhythmias in the Multicenter Study of General Anesthesia (presumably, some dysrhythmias were VF. [28,29]

The distinction between ventricular fibrillation and flutter (collectively, VF) is moot because both are incompatible with life, and management is the same. [31,101,271,274,275] With either, there are continuous regular (flutter) or irregular undulations of the ECG baseline. [31,46,66] P and T waves are absent in the surface ECG leads. Fibrillatory waveform amplitude is coarse at the onset of fibrillation and becomes fine as the VF persists. Fine fibrillation identifies patients with worse survival rates and may be misdiagnosed as asystole. [64]

The only effective management for VF is defibrillation with transthoracic shocks of 200-360 J delivered as soon as possible after presumptive diagnosis of VF. [31,101,271,276] Time should not be wasted administering drugs to improve defibrillation success. Lidocaine, bretylium, procainamide, and amiodarone are used only to prevent recurrences of VF. A precordial thump is occasionally effective in terminating VF, but should be attempted only if a defibrillator is not available immediately because chest thumps may convert some VT to VF. [271] Initial success with defibrillation depends on the duration of VF. [277-279] If present for only a few seconds to minutes and if fibrillatory waves are coarse, initial success is high. Conversely, with continued VF and fine fibrillatory waves, defibrillation is more difficult. Also, if VF continues for more than 4 min, there may be irreversible damage to the brain and other vital organs. [280]

Heart Block

Atrioventricular (AV) heart block is a temporary or permanent conduction disturbance resulting from anatomic or functional impairment. [281] It is distinguished from physiologic interference, whereby impulse propagation is delayed or blocked because of persistent refractoriness from previous excitation. AV heart block (AVHB) is diagnosed when: 1) supra-ventricular impulses are conducted to the ventricles with delay (1 degree AVHB, PR greater than 0.21 s in adults or 0.18 s in children); 2) some but not all impulses are conducted (2 degrees AVHB); or 3) no impulses are conducted (complete or 3 degrees AVHB). Second degree AVHB is subdivided as type I (Wenckebach) and type II (Mobitz) block. With type I or II block, respectively, there is progressive or no PR interval prolongation before dropped beats.

Fascicular block is block of the left anterior or posterior fascicles (LAF, LPF) of the left bundle branch (LBB) or the right bundle branch (RBB). Bifascicular heart block is block of any two of these fascicles. LBB block or RBB with LAF block are by far most common types of bifascicular block. [281-283] Although complete heart block is often preceded by bifascicular block, progression to complete heart block is not common. Further, no single laboratory or clinical variable identifies patients at high risk for progression to high degree AV block, [281] and there is no evidence that bifascicular block will progress to high degree AVHB during the course of anesthesia and surgery. [187]

By far the most common cause for acquired chronic AVHB is idiopathic, progressive fibrosis of the conducting system (Lev's or Lenegre's disease). [282,283] Although acute myocardial infarction is an important cause of transient AV heart block, chronic coronary disease infrequently produces persistent AV heart block. [282] Other causes for acquired AV or fascicular heart block are listed in Table 9. Volatile anesthetics may produce transient high-grade AV block with CCB [114-118,284-287] or amiodarone. [93-96] Also, although beta-blockers depress AV node conduction, this action highly depends on prevailing adrenergic tone. The potential for additive effects appears greatest with enflurane and

halothane compared with isoflurane (data for desflurane and sevoflurane are not available). [288,289]

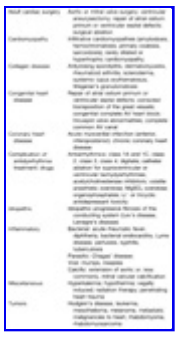

Table 9

There is no consensus among anesthesiologists as to indications for temporary perioperative pacing for patients with AV or fascicular heart block. The American College of Cardiology and American Heart Association Joint Task Force Committee on Pacemaker Implantation has published guidelines for permanent pacing in adults and children and temporary pacing in those with acute myocardial infarction. [281] Emphasis is placed on concurrence of symptoms with bradycardia as a result of heart block or sinus node dysfunction, especially in children. As for temporary perioperative pacing, pacing is indicated whenever bradycardia caused by heart block threatens vital organ perfusion. Temporary pacing should also be considered for bradycardia-dependent tachydysrhythmias (e.g., torsades de pointes). Finally, stand-by ventricular pacing should be available for patients with LBB block who require a pulmonary artery catheter for perioperative monitoring.

Summary and Recommendations

Cardiac dysrhythmias are common during anesthesia and surgery and occur in patients with structural heart disease or normal hearts. The inciting or aggravating factor is often physiologic imbalance unique to perioperative settings (e.g., anesthetic or adjuvant drugs, adrenergic stress, acid-base or electrolyte imbalance, hypoxia, hypercapnia). [2] If so, it is important to correct or remove such imbalance. Not only may this be sufficient therapy, but it also will enhance indicated specific treatment with drugs or devices.

There are important limitations to treatment with anti-dysrhythmic drugs. Among these is prodysrhythmia, whereby antidysrhythmics may paradoxically aggravate dysrhythmias. Others are adverse drug interactions, troublesome side effects, and cardiovascular depression. Therefore, to reduce complications with treatment, the Sicilian Gambit's pathophysiologic compared with more empiric approaches to drug selection has been stressed. [3,290] With the Gambit's approach, drugs are targeted at specific, vulnerable parameters affecting the genesis of dysrhythmias (e.g., beta-receptor, L-type Ca^{2}+ channels, Na sup + - and K sup + -currents). Finally, although cardiac pacing is useful for prevention and management, current technology is too invasive and costly for routine use. However,

expected improvements with noninvasive technology will make pacing increasingly attractive as a therapeutic modality.

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*Ibutilide was approved in May 1996 for intravenous use for rapid conversion of atrial fibrillation or flutter of recent onset (< 90 days) to sinus rhythm. It has primarily a class III action. [14]

**Waxman H. Acute pharmacologic management of supraventricular and ventricular arrhythmias. ACC Current Journal Review 4:29-32, 1995.

***The author recalls a chronic renal failure patient with usual serum K sup + values above 5.5 mEq/L who underwent coronary bypass surgery. This patient had prominent U waves and frequent atrial extrasystoles after cardiopulmonary bypass, despite serum K sup + values of 4.5 and 4.7 mEq/L. The extrasystoles resolved following administration of K sup +.

****Atlee J, Bilof R. Transesophageal ventricular pacing in anesthetized adults. Anesthesiology 1993;79:A75.

*****Antidromic AP reentry PSVT, atrial flutter-fibrillation, or sino-atrial tachycardias with preexcited QRS complexes.

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