

Management of Common Arrhythmias: Part II.

Ventricular Arrhythmias and Arrhythmias in Special Populations

A. KESH HEBBAR, M.D., and WILLIAM J. HUESTON, M.D., Medical University of South Carolina, Charleston, South Carolina

In patients without established cardiac disease, the occurrence of premature ventricular complexes without sustained ventricular tachycardia is more an annoyance than a medical risk, and treatment is not required. In contrast, patients with established heart disease and premature ventricular complexes have a higher likelihood of developing ventricular tachycardia or fibrillation. These patients should be treated with a beta blocker or class I antiarrhythmic drug. Treatment of arrhythmias in pregnant women is rarely needed. When treatment is required, amiodarone should be avoided, and beta blockers should be used with caution, because these agents have been associated with fetal growth retardation. The most important rhythm abnormality in athletes is ventricular tachycardia associated with hypertrophic cardiomyopathy. If the presence of the disease is confirmed by echocardiography, beta-blocker therapy is necessary, and these patients should be limited to participation in nonstrenuous sports. Acute arrhythmias in children with Wolff-Parkinson-White syndrome can be treated with adenosine. Radiofrequency ablation of the accessory pathway can provide long-term control. (*Am Fam Physician* 2002;65:2491-6. Copyright© 2002 American Academy of Family Physicians.)

Members of various family practice departments develop articles for "Practical Therapeutics." This article is one in a series coordinated by the Department of Family Medicine at the Medical University of South Carolina, Charleston. Guest editor of the series is William J. Hueston, M.D.

Atrial fibrillation, supraventricular tachycardias, and sinus nodal arrhythmias are the rhythm abnormalities most commonly encountered by family physicians. Ventricular ectopy is also common. The management of ventricular ectopy can be troublesome, because some treatments may actually worsen outcomes rather than improve them. Part II of this two-part article reviews the management of ventricular arrhythmias and approaches to rhythm disturbances in selected populations.

Premature Ventricular Complexes

A premature ventricular complex is characterized by the premature occurrence of a QRS complex that is bizarre in shape and lasts longer than 120 msec. The T wave is large and usually of opposite polarity to the QRS complex. A premature ventricular complex is usually followed by a full compensatory pause.

The term "ventricular bigeminy" refers to alternating normal sinus and premature ventricular complexes. Three or more successive premature ventricular complexes are arbitrarily defined as ventricular tachycardia.

Premature ventricular complexes become

more prevalent with increasing age and occur in association with a variety of stimuli (Table 1). It is important to determine whether underlying structural heart disease is present and left ventricular function is impaired. Other common causes include electrolyte abnormalities, stimulants, and some medications.

Attempts have been made to estimate the risk of chronic premature ventricular com-

TABLE 1
Causes of Ventricular Arrhythmias

Cardiac causes

Acute and chronic ischemic heart disease
Cardiomyopathy
Valvular heart disease
Mitral valve prolapse

Noncardiac causes

Stimulants: caffeine, cocaine, alcohol
Metabolic abnormalities: acidosis, hypoxemia, hyperkalemia, hypokalemia, hypomagnesemia
Drugs: digoxin (Lanoxin), theophylline, antipsychotics, tricyclic antidepressants, antiarrhythmics with proarrhythmic potential (e.g., flecainide [Tambocor], dofetilide [Tikosyn], sotalol [Betapace], quinidine)

This is part 2 of a two-part article on common arrhythmias. Part I, "Supraventricular Arrhythmias," appears on page 2479 in this issue.

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plexes based on their frequency and waveforms. Several studies^{1,2} have demonstrated an increased risk for life-threatening arrhythmias with 10 or more ectopic impulses per hour or the presence of impulse salvos (i.e., three to five consecutive impulses). However, structural heart disease and poor left ventricular function are the key factors in determining whether treatment is warranted and what the prognosis may be.

MANAGEMENT

Patients Without Heart Disease. In the absence of heart disease, premature ventricular complexes are associated with little or no increased risk of developing a dangerous arrhythmia. In this situation, the risk-to-benefit ratio of antiarrhythmic drug therapy does not support routine treatment.³ It is important to review medications, determine if stimulants are being used, and correct electrolyte abnormalities. If no underlying cause is found, the optimal approach is patient reassurance.

Patients should be made aware of the potential dangers of antiarrhythmic drug therapy as determined in the Cardiac Arrhythmia Suppression Trials (CAST and CAST II).^{4,5} CAST showed that the risk of dying increased, rather than decreased, with successful long-term suppression of premature ventricular complexes after myocardial infarction in older patients. At best, CAST II showed no impact on long-term survival from drug treatment that successfully suppressed premature ventricular complexes.

If patients with multiple premature ventricular complexes have severe, disabling symptoms, beta blockers are the safest initial choice. Referral to a cardiologist is indicated if beta-blocker therapy is not effective. In this situation, the next agents to be tried would be class I antiarrhythmic drugs, such as flecainide (Tambacor) and amiodarone (Cordarone), although radiofrequency ablation of an ectopic focus may also be an appropriate treatment.

Patients with Structural Heart Disease. The occurrence of premature ventricular complexes in patients with structural heart disease has been shown to significantly

increase the risk of subsequent morbidity and mortality. Coronary heart disease, cardiomyopathy, and congestive heart failure are the major cardiac diseases associated with unfavorable outcomes in patients with premature ventricular complexes.

Ventricular Tachycardia

Ventricular tachycardia refers to a rhythm originating from a ventricular ectopic focus at a rate greater than 100 beats per minute. The electrocardiogram shows a wide-complex tachycardia with no associated P waves.

In patients with bundle branch block, Wolff-Parkinson-White syndrome, or aberrant conduction, supraventricular tachycardia can resemble ventricular tachycardia. Because of the morbidity and mortality associated with untreated ventricular tachycardia, any wide-complex tachycardia should be assumed to be ventricular tachycardia until proved otherwise. Physicians should keep in mind that patients with ventricular tachycardia can have minimal symptoms.

MANAGEMENT

The mortality rate within two years is reported to be higher than 30 percent in patients with nonsustained ventricular tachycardia on Holter monitoring and impaired left ventricular function.⁶ Two large multicenter trials^{7,8} showed a clear advantage for automatic cardioverter-defibrillator implantation over drug therapy in patients who had a malignant ventricular arrhythmia or who had been resuscitated from sudden cardiac death.

The selection of high-risk patients for defibrillator implantation should be based on left ventricular function and the findings of electrophysiologic studies. Implantable defibrillators appear to be most beneficial in patients with a low ejection fraction who are noted to have frequent premature ventricular complexes, nonsustained ventricular tachycardia on Holter monitoring, and a history of syncope or near-syncope. It is critical to rule out coronary heart disease and to optimize the treatment of congestive heart failure in these patients.

When ventricular tachycardia is diagnosed in relatively asymptomatic patients, medical treatment should be attempted. New recommendations from the American Heart Association⁹ emphasize the initial use of 300 mg of intravenously administered amiodarone, followed by repeated 150-mg intravenous doses every eight to 10 minutes, in patients with pulseless ventricular tachycardia.

Patients with stable ventricular tachycardia should be given 150 mg of amiodarone intravenously over 10 minutes, followed by an infusion at 1 mg per minute for six hours and then at 0.5 mg per minute until the ventricular tachycardia converts to sinus rhythm or another less dangerous rhythm. The alternative treatment is intravenously administered lidocaine (Xylocaine), given first in a 100-mg bolus (or 1 mg per kg) and then in an infusion at 1 to 4 mg per minute.

In hemodynamically unstable patients, electrical cardioversion should be attempted in accordance with the recently revised advanced cardiac life support (ACLS) protocols.⁹

Automatic implantable cardioverter-defibrillators are considered the most effective treatment for patients with life-threatening ventricular tachycardia or fibrillation. According to expert recommendations, implantation of an automatic cardioverter-defibrillator should be considered in these situations¹⁰: (1) cardiac arrest resulting from ventricular fibrillation or tachycardia not caused by a transient or reversible cause; (2) spontaneous, sustained ventricular tachycardia; (3) syncope of undetermined origin and sustained ventricular tachycardia or fibrillation induced during electrophysiologic studies; and (4) non-sustained ventricular tachycardia with coronary artery disease and left ventricular dysfunction if ventricular tachycardia or fibrillation is induced during electrophysiologic studies.

Treatment of Arrhythmias in Special Populations

PREGNANT WOMEN

The incidence and severity of atrial and ventricular ectopy are reported to increase during pregnancy.^{11,12} The reasons remain unclear, although it may be that more frequent contact with health care providers allows greater opportunity for cardiac disturbances to be recognized.

Isolated atrial and ventricular ectopic beats in pregnant women without existing heart disease are usually benign.¹³ Increases in atrial extra beats and sustained atrial arrhythmias may be associated with the use of sympathomimetic drugs such as pseudoephedrine.¹³ Thus, it is important to inquire about the use of over-the-counter medication in pregnant women who complain about palpitations or extra heartbeats.

Most medications that are safe in nonpregnant patients are safe in pregnant women. Amiodarone is the only antiarrhythmic drug that has been associated with signif-

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icant fetal abnormalities. In addition to cardiac disturbances, amiodarone can cause fetal goiter, neonatal hypothyroidism, and fetal growth retardation.¹⁴ Based on these observations, amiodarone should not be given to pregnant women. When used for hypertension management during pregnancy, propranolol (Inderal) and atenolol (Tenormin) have been associated with intrauterine growth restriction. These beta blockers can also cause hypoglycemia in the newborn.^{15,16}

Most antiarrhythmic drugs are safe for use in lactating women. The exceptions are amiodarone and acebutolol (Sectral). Amiodarone should not be used because it is secreted in breast milk. Acebutolol becomes concentrated in breast milk; therefore, breastfed infants receive a much larger dose than they would with other beta blockers. As a result, these infants may develop neonatal bradycardia or hypoglycemia. If lactating women need a beta blocker, an agent other than acebutolol should be used.¹⁴

ATHLETES

Well-trained athletes usually have a slow heart rate, with occasional sinus pauses and, frequently, multiple benign escape beats. If no symptoms are present and the sinus pauses last three seconds or less, no further evaluation is necessary.¹⁷ The rhythm changes are caused by increased vagal tone. During exercise, vagal tone is reduced, and appropriate heart rate acceleration occurs. The maximal heart rate is unchanged, although more physical exertion may be required to achieve it.

Malignant ventricular tachycardia, the arrhythmia of most concern in athletes, is usually associated with idiopathic hypertrophic cardiomyopathy. In one series,¹⁸ 48 of 131 athletes who experienced sudden cardiac death were found to have this disease, and another 14 probably had it. Symptoms of syncope or near-syncope with exercise or a family history of sudden cardiac death in a close relative are red flags for the presence of idiopathic hypertrophic cardiomyopathy. Athletes who have an aortic murmur that increases with Valsalva's maneuver should also be

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evaluated for hypertrophic cardiomyopathy before they are allowed to participate in sports.

When hypertrophic cardiomyopathy is identified, treatment with a beta blocker or calcium channel blocker can reduce cardiac contractility and limit heart rate during exertion. An alternative to pharmacologic management is the insertion of an implantable cardioverter-defibrillator.

Expert panels have recommended that athletes with identified hypertrophic cardiomyopathy be barred from participation in strenuous sports.¹⁹ These persons may participate in low-intensity sports such as bowling, golf, billiards, and cricket.²⁰ High-performance athletes who have attempted to engage in their sport while taking medications frequently complain of fatigue or diminished performance. Consequently, they may not comply with treatment and thereby increase their risk of sudden cardiac death.

CHILDREN

Supraventricular tachycardias are the most common sustained pathologic arrhythmias in children younger than 12. These arrhythmias are usually caused by an accessory atrioventricular pathway or Wolff-Parkinson-White syndrome.²¹

The same medications are used to treat supraventricular tachycardias in children and adults. Adenosine (Adenocard) in a dose of 100 mcg per kg administered intravenously can usually disrupt the arrhythmia. If the initial dose is not successful, it may be doubled and repeated. For long-term control, radiofrequency ablation is the recommended definitive treatment, with a success rate of 85 to 95 percent when performed by experienced pediatric cardiologists.²²

Atrial and ventricular extra beats are also common in children. These extra beats are not cause for concern if they resolve with exercise in otherwise healthy children. However, ventricular extra beats are associated with a higher risk of death in children who have existing structural heart disease or cardiomyopathies.²¹ These children should be referred for additional evaluation.

PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Some form of rhythm abnormality is present in 90 percent of patients with acute myocardial infarction.²³ Serious arrhythmias, such as ventricular fibrillation, occur early in the acute phase of myocardial infarction, with the risk declining rapidly after 24 hours. The most common arrhythmias are sinus tachycardia and premature ventricular complexes. Sinus bradycardia often develops in patients with acute inferior infarction. Common arrhythmias in patients with acute myocardial infarction are summarized in *Table 2*.²⁴

Accelerated idioventricular rhythm is defined as a ventricular rhythm with a rate of 60 to 125 beats per minute. This arrhythmia is sometimes called "slow ventricular tachycardia." Accelerated idioventricular rhythm is present in up to 20 percent of patients after an acute myocardial infarction.²⁵ It occurs with equal frequency in anterior and inferior infarctions and does not usually have a negative impact on hemodynamic status. Accelerated idioventricular rhythm is also common after successful reperfusion with thrombolytics, but it is not considered a reliable indicator of reperfusion. Most episodes are self-limited and do not require treatment.

Premature ventricular complexes do not usually require treatment. However, if premature ventricular complexes and sinus tachycardia are both present, they may be controlled with orally administered beta blockers. Early intravenous administration of these drugs can reduce the incidence of ventricular fibrillation in patients with evolving acute myocardial infarction.²⁴

Nonsustained ventricular tachycardia in the immediate peri-infarction period does not appear to be associated with an increased risk of death, and antiarrhythmics have not been shown to have a beneficial effect on morbidity and mortality rates.²⁶ However, nonsustained ventricular tachycardia occurring after 48 hours in patients with left ventricular dysfunction is a marker for sudden cardiac death. These patients should be observed closely and referred for electrophysiologic studies. Sustained ventricular tachycardia (lasting longer than 30 seconds) is a medical emergency and should be managed in accordance with ACLS protocols.

Atrial fibrillation occurs in 10 to 15 percent of patients with acute myocardial infarction.²⁴ It is most often associated with larger anterior infarcts and signals an increased risk for stroke, as well as increased mortality.²⁷

Sinus bradycardia is particularly common in patients

TABLE 2
Common Arrhythmias in Patients with Acute Myocardial Infarction

<i>Arrhythmia</i>	<i>Frequency</i>	<i>Recommendations and comments</i>
Premature ventricular complexes	Common	Usually require no treatment; check for metabolic and electrolyte abnormalities.
Accelerated idioventricular rhythm	15% to 20%	Observation; no treatment usually needed
Ventricular tachycardia	Up to 60%	If nonsustained, immediate intense observation If sustained, give lidocaine (Xylocaine) in bolus of 1.0 to 1.5 mg per kg, followed by maintenance infusion at 1 to 4 mg per minute or direct-current cardioversion.
Ventricular fibrillation	5%	Defibrillation; if defibrillation is not initially successful, give amiodarone (Cordarone) in 300-mg bolus or lidocaine in 100-mg bolus.
Sinus tachycardia	Common	Rule out hypoxemia and hypovolemia; provide adequate analgesia; evaluate for congestive heart failure.
Atrial fibrillation	10% to 15%	Slow ventricular rate with beta blocker (e.g., propranolol [Inderal]) or calcium channel blocker (e.g., diltiazem [Cardizem]).
Paroxysmal supraventricular tachycardia	<10%	Give adenosine (Adenocard), 6 to 12 mg by IV push over 1 to 3 seconds; verapamil (Calan), diltiazem, or propranolol can be used as alternatives.
Sinus bradycardia	Up to 40% in acute inferior MI	If symptoms are present, give 0.5 to 1.0 mg of atropine IV.
First-degree atrioventricular block	15% in acute inferior MI	Observation; if symptoms are present, give atropine.
Mobitz type I block (Wenckebach block)	Up to 10%	Observation; if symptoms are present, give atropine.
Mobitz type II block	<1%	Temporary external or transvenous demand pacemaker
Complete atrioventricular block	5% to 15%	Ventricular pacemaker

IV = intravenous; MI = myocardial infarction.

Information from Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald E, ed. Heart disease: a textbook of cardiovascular medicine. 6th ed. Philadelphia: Saunders, 2001:1114-28.

with acute inferior and posterior infarction. If symptoms are present, atropine should be given. First-degree atrioventricular block occurs in 15 percent of patients with acute myocardial infarction and is sometimes exacerbated by medications.²⁴ No therapeutic measures are necessary in these patients, but monitoring should be continued.

Mobitz type I block (Wenckebach block) occurs when there is ischemia to the atrioventricular node. This arrhythmia is commonly associated with inferior myocardial infarction. It rarely progresses to complete atrioventricular block, and temporary pacing is almost never required. Temporary pacing support is required in most patients with Mobitz type II block or third-degree heart block.

The authors indicate that they do not have any conflicts of interest. Sources of funding: none reported.

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