VITEBSK STATE MEDICAL UNIVERSITY

CHAIR OF THE FACULTY THERAPY

**FACULTY THERAPY**

(Methodics work out for foreign students, studying the course of faculty therapy)

Part 1

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**ECTOPIC SUPRAVENTRICULAR ARRHYTHMIAS**

***A trial and Junctional Premature Beats***

Single ectopic supraventricular beats can originate in the atria or in of the AV node.The former are called atrial premature beats (or premcontractions); the latter, junctional premature beats. These are connomena, neither indicating underlying cardiac disease nor requiring They can, however, initiate more sustained arrhythmias.

An atrial premature beat can be distinguished from a normal sinus beat by the *contour* of the P wave and by the *timing* of the beat.

*Contour.* Because an atrial premature beat originates at an atrial site distant from the sinus node, atrial depolarization does not occur in the usual manner and the configuration of the resultant P wave differs from that of the sinus P waves. If the site of origin of the atrial premature beat is far from the sinus node, the *axis* of the atrial premature beat will also differ from that of the normal P waves.

*Timing.* An atrial premature beat comes too early, *i.e.,* it intrudes itself before the next anticipated sinus wave.The third beat is an atrial premature beat. The P wave is shaped differently from the other, somewhat unusual-looking P waves, and the beat is clearly premature.

With junctional premature beats, there is usually no visible P wave, but sometimes a retrograde P wave may be seen. This is just like the case with the junctional escape beats seen with sinus arrest.

***Ectopic Ventricular Arrhythmias***

Ventricular arrhythmias are rhythm disturbances arising below the AV node.

*Premature Ventricular Contractions*

*Premature ventricular contractions,* or PVCs, are certainly the most common of the ventricular arrhythmias. The QRS complex of a PVC appears wide and bizarre because ventricular depolarization does not follow the normal conduction pathways. However, the QRS complex may not appear wide in all leads, so scan the entire 12-lead EKG before making your diagnosis. A retrograde P wave may sometimes be seen, but it is more common to see no P wave at all. A PVC is followed by a prolonged pause before the next beat appears.

Isolated PVCs are common in normal hearts and rarely require treatment An isolated PVC in the setting of an acute myocardial infarction, however, is more ominous, because it can trigger ventricular tachycardia or ventricular fi­brillation, both of which are life-threatening arrhythmias.

PVCs may occur randomly or may alternate with normal sinus beats in ,1 regular pattern. If the ratio is one normal sinus beat to one PVC, the rhythm is called *bigeminy. Trigeminy* refers to two normal sinus beats for every one PVC and so on.

When should you worry about PVCs? This is a subject of much debate, and the debate is far from over. Certain situations have been identified in which PVCs appear to pose an increased risk of triggering ventricular tachycardia, ventricular fibrillation, and death. These situations are summarized in *the rules of malignancy:*

1. Frequent PVCs
2. Runs of consecutive PVCs, especially three or more in a row
3. Multiform PVCs, in which the PVCs vary in their site of origin and hence in their appearance
4. PVCs falling on the T wave of the previous beat, called the "R on T" phe­nomenon. The T wave is a vulnerable period in the cardiac cycle, and a PVC falling there appears to be more likely to set off ventricular tachycardia.
5. Any PVC occurring in the setting of an acute myocardial infarction.

Although PVCs meeting one or several of these criteria are associated with an increased risk of developing a life-threatening arrhythmia, there is no evidence that suppressing these PVCs with antiarrhythmic medication reduces mortality in any setting.

**PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA**

Supraventricular tachycardia (SVT), a common clinical condition, is any tachyarrhythmia that requires only atrial and/or atrioventricular (AV) nodal tissue for its initiation and maintenance. It is usually a narrow-complex tachycardia that has a regular, rapid rhythm; exceptions include atrial fibrillation (AF) and multifocal atrial tachycardia (MAT). Aberrant conduction during SVT results in a wide-complex tachycardia. SVT occurs in persons of all age groups, and treatment can be challenging.

Paroxysmal supraventricular tachycardia (PSVT) is episodic, with an abrupt onset and termination. Manifestations of SVT are quite variable; patients may be asymptomatic or they may present with minor palpitations or more severe symptoms. Results from electrophysiology studies have helped determine that the pathophysiology of SVT involves abnormalities in impulse formation and conduction pathways. The most common mechanism identified is reentry (Denes, 1973; Rosen, 1974; Akhtar, 1984; Waldo, 1993). This article focuses on SVT, including the pathophysiology, clinical presentation, diagnosis, management, and treatment options of this condition.

**Frequency**

PSVT incidence is approximately 1-3 cases per 1000 persons. The incidence rate of the WPW pattern on ECG tracings is 0.1-0.3% in the general population, although not all patients develop SVT (Klein, 1979; Montoya, 1991; Ganz, 1995; Xie, 1998; Al-Khatib, 1999). In a population-based study, the prevalence of PSVT was 2.25 cases per 1000 persons, with an incidence of 35 cases per 100,000 person-years (Orejarena, 1998). AVNRT is more common in patients who are of middle age or older, while adolescents are more likely to have SVT mediated by an accessory pathway. PSVT is not only observed in healthy individuals, it is also common in patients with previous myocardial infarction, mitral valve prolapse, rheumatic heart disease, pericarditis, pneumonia, chronic lung disease, and current alcohol intoxication (Klein, 1979; Montoya, 1991; Ganz, 1995; Xie, 1998). Digoxin toxicity also may be associated with PSVT (Ganz, 1995; Xie, 1998; Josephson, 2001).

PSVT may start suddenly and last for seconds or days. Patients may or may not be symptomatic, depending on their hemodynamic reserve and their heart rate, the duration of the PSVT, and coexisting diseases. PSVT can result in heart failure, pulmonary edema, myocardial ischemia, and/or myocardial infarction secondary to an increased heart rate in patients with poor left ventricular function (Ganz, 1995; Xie, 1998; Josephson, 2001). In fact, one study found that one third of patients with SVT experienced syncope, required cardioversion, or had an episode of sudden death (Wood, 1997).

Most series of catheter ablation reflect a higher proportion of female patients with AVNRT than male patients. This may reflect a true higher incidence in women, or it may reflect the sample of patients who are referred (or choose) to undergo extensive evaluation and/or catheter ablation.

In a population-based study, the risk of developing PSVT was twice as high in women compared to men (Orejarena, 1998).

**Pathophysiology**

The development of intracardiac electrophysiology studies has dramatically changed the classification of SVT. Intracardiac recordings have identified the various mechanisms of SVT. Depending on the site of origin of the dysrhythmia, SVTs may be classified as an atrial or AV tachyarrhythmia (Klein, 1987; Basta, 1997).

**CLINICAL**

**History**

Because symptom severity depends on the presence of structural heart disease and on the hemodynamic reserve of the patient, individuals with PSVT may present with mild symptoms or severe cardiopulmonary complaints. Some common presenting symptoms are listed below (Wood, 1997; Al-Khatib, 1999). Palpitations and dizziness are the most common symptoms reported by patients with SVT. Chest discomfort may be secondary to a rapid heart rate, and it frequently subsides with the termination of the tachycardia.

Common presenting symptoms of PSVT and their frequency rates are as follows:

Palpitation - Greater than 96%

Dizziness - 75%

Shortness of breath - 47%

Syncope - 20%

Chest pain - 35%

Fatigue - 23%

Diaphoresis - 17%

Nausea - 13%

History should include time of onset, any triggers, any previous episodes or arrhythmia, and previous treatment. A detailed past medical and cardiac history and a complete list of all medications should be obtained.

Patients who are hemodynamically unstable should be resuscitated immediately with cardioversion. An ECG should be performed as soon as possible.

Many patients with frequent episodes of PSVT tend to avoid activities such as exercising and driving due to past episodes of syncope or near-syncope.

**Physical**

Pertinent findings are generally limited to cardiovascular and respiratory systems. Patients often appear quite distressed. Tachycardia may be the only finding in patients who are otherwise healthy and have significant hemodynamic reserve.

Patients who have limited hemodynamic reserve may be tachypneic and hypotensive. Crackles may be auscultated secondary to heart failure. An S3 may be present, and large jugular venous pulsations may also be visualized (Ganz, 1995; Wood, 1997; Xie, 1998).

**Lab Studies**

A cardiac enzyme evaluation should be ordered for patients with chest pain; patients with risk factors for myocardial infarction; and patients who are otherwise unstable and present with heart failure, hypotension, or pulmonary edema. Young patients with no structural heart defects have a very low risk of myocardial infarction.

Electrolyte levels should be checked because electrolyte abnormalities can contribute to PSVT.

A complete blood cell count helps assess whether anemia is contributing to the tachycardia or ischemia.

The results from thyroid studies are rarely diagnostic of hyperthyroidism.

Obtain a digoxin level for patients on digoxin because PSVT is one of the many dysrhythmias that can be caused by supratherapeutic levels of this drug.

**Imaging Studies**

ECG findings allow classification of the tachyarrhythmia, and they may allow a precise diagnosis. P waves may not be visible; when present, they may be normal or abnormal depending on the mechanism of atrial depolarization (Ganz, 1995; Obel, 1997; Xie, 1998).

ECG characteristics of the SVTs are as follows:

Atrial tachycardia - Heart rate 120-250 bpm; P-wave morphology different from sinus rhythm; long RP interval (in general); AV block does not terminate tachycardia

Obtain a chest radiograph to assess for the presence of pulmonary edema. Infections such as pneumonia, which are associated with PSVT in certain cases, can also be confirmed with findings from this imaging method (Ganz, 1995; Pieper, 1995; Xie, 1998; Trohman, 2000; Josephson, 2001).

A transthoracic echocardiogram may be helpful if structural heart disease is suggested.

**Procedures**

Electrophysiology studies and radiofrequency catheter ablation

Electrophysiology studies have dramatically changed the diagnosis of SVT. Intracardiac recordings have helped map accessory pathways and reentry circuits in patients, and they have also assisted cardiologists and electrophysiologists in understanding the mechanisms behind these tachyarrhythmias.

At present, electrophysiologic studies are generally performed in combination with radiofrequency catheter ablation. Catheter ablation is indicated in patients with severe symptoms, symptomatic preexcitation syndrome, incessant tachycardia, and those who do not tolerate or do not desire medical therapy. Catheter ablation procedures are generally performed in an outpatient setting or with an overnight stay for observation.

**TREATMENT**

Medical Care

* Vagal maneuvers are the first-line treatment in hemodynamically stable patients. Vagal maneuvers, such as breath-holding and the Valsalva maneuver (ie, having the patient bear down as though having a bowel movement), all slow conduction in the AV node and can potentially interrupt the reentrant circuit.
* Carotid massage is another vagal maneuver that can slow AV nodal conduction. Massage the carotid sinus for several seconds on the nondominant cerebral hemisphere side. This maneuver is usually reserved for young patients. Due to the risk of stroke from emboli, auscultate for bruits before attempting this maneuver. Do not perform carotid massage on both sides simultaneously.
* Synchronized cardioversion starting at 50 J can be used immediately in patients who are hypotensive, have pulmonary edema, have chest pain with ischemia, or are otherwise unstable.

Short-term medical management

When SVT is not terminated by vagal maneuvers, short-term management involves intravenous adenosine or calcium channel blockers. Adenosine is a short-acting drug that blocks AV node conduction. Adenosine does not usually terminate atrial tachycardia, although it is effective for terminating SNRT. Typical adverse effects of adenosine include flushing, chest pain, and dizziness. These effects are temporary because adenosine has a very short half-life of 10-20 seconds (Siberry, 2000).

Another alternative for the acute treatment of SVT is verapamil. Verapamil is a calcium channel blocker that also has AV blocking properties. Verapamil has a longer half-life than adenosine and may help maintain sinus rhythm following the termination of SVT. It is also advantageous for controlling the ventricular rate in patients with atrial tachyarrhythmia.

Electrical cardioversion is the most effective method for restoring sinus rhythm. If AF has been present for longer than 24-48 hours, defer cardioversion until the patient has been adequately anticoagulated to prevent thromboembolic complications (Pieper, 1995; Campbell, 1997; Connors, 1997; Levy, 1997; Reimold, 1997; Gold, 1999; Siberry, 2000; Trohman, 2000).

Long-term medical management

The choice of long-term therapy for patients with SVT depends on the type of tachyarrhythmia and the frequency and duration of episodes, symptoms, and risks associated with the arrhythmia (eg, heart failure, sudden death). Evaluate patients on an individual basis, and tailor the best therapy for the specific tachyarrhythmia.

Patients with PSVT may initially be treated with calcium channel blockers, digoxin, and/or beta-blockers. Class IA, IC, or III antiarrhythmic agents are used less frequently because of the success of radiofrequency catheter ablation (Pieper, 1995; Campbell, 1997; Connors, 1997; Levy, 1997; Reimold, 1997; Gold, 1999; Siberry, 2000; Trohman, 2000). Consider radiofrequency ablation for any patient with symptomatic PSVT in whom long-term medical treatment is not effectively tolerated or desired. In addition, because of the risk of sudden cardiac death, perform catheter ablation on patients with symptomatic WPW syndrome. Radiofrequency catheter ablation is more than 90% effective in curing PSVT (Ganz, 1995; Pieper, 1995; Xie, 1998; Trohman, 2000; Ganz, 2002).

Radiofrequency ablation involves focally ablating the crucial component of the arrhythmia mechanism. For example, in AVNRT, the slow pathway is ablated, which prevents the reentry cycle. The accessory pathway is targeted in patients with AVRT. Focal atrial tachycardia, atrial flutter, and, in some cases, AF can also be cured with ablation. Radiofrequency ablation has a high success rate and is performed using conscious sedation in an outpatient setting or with overnight hospitalization. Complications, which occur at a rate of 1-3%, include deep vein thrombosis, systemic embolism, infection, cardiac tamponade, and hemorrhage. The risk of death is approximately 0.1%. The lifetime risk of fatal malignancy as a result of radiation exposure is low.

Radiofrequency ablation is cost-effective for patients who have frequent episodes of SVT that require antiarrhythmic agents and frequent emergency visits. It is also indicated for patients with incessant tachycardia and for patients with symptomatic WPW syndrome. The optimal management strategy for patients with asymptomatic preexcitation syndromes remains uncertain (Scheinman, 1992; Strickberger, 1993; Lesh, 1994; Ganz, 1995; Ganz, 2002).

**Further Inpatient Care**

Patients who require cardioversion, are unstable, and have comorbid illnesses should be admitted to the hospital. Patients who are young, healthy, and asymptomatic may be discharged and advised to have a follow-up examination with their primary physician or cardiologist. If the patient is having more frequent episodes of PSVT and medical therapy is not successful or desired, then radiofrequency ablation should be proposed.

**Further Outpatient Care**

Patients treated medically should be monitored regularly. Patients cured with radiofrequency catheter ablation are typically seen once in a follow-up examination following the procedure, then as needed for recurrent symptoms.

**Complications**

Rare complications of PSVT include myocardial infarction, congestive heart failure, syncope, and sudden death.

Potential complications of radiofrequency catheter ablation include hematoma, bleeding, infection, pseudoaneurysm, myocardial infarction, cardiac preformation, heart block that requires a pacemaker, thromboembolic complications, stroke, need for emergency surgery, radiation burn, increased risk of malignancy, and death.

**Prognosis**

Patients with symptomatic WPW syndrome have a small risk of sudden death. Otherwise, prognosis is dependent on any underlying structural heart disease. Patients with PSVT in the setting of a structurally normal heart have an excellent prognosis.

**Ventricular Tachycardia**

Ventricular tachycardia (VT) refers to any rhythm faster than 100 beats per minute arising distal from the bundle of His. The rhythm may arise from working ventricular myocardium and/or the distal conduction system. VT may cause important symptoms such as palpitations, dyspnea, and syncope. Depending upon the underlying heart disease, VT often is associated with an increased risk of sudden death. The most common setting for VT is ischemic heart disease, in which myocardial scar is the substrate for electrical reentry. It often, but not always, is associated with hemodynamic compromise, particularly if the left ventricle is impaired or the heart rate is especially fast. When sustained VT causes signs or symptoms of diminished perfusion, emergent treatment is necessary.

VT is distinguished from ventricular fibrillation (VF), which is a grossly disorganized rapid ventricular rhythm that varies in both interval and waveform. VF may be difficult to distinguish from rapid polymorphic VT. Sudden death accounts for approximately half of all deaths from cardiovascular disease and generally is caused by VT and fibrillation.

**Frequency**

The incidence of VT in the United States is not well quantified because of the clinical overlap of VT with VF. Examination of sudden death data provides a rough estimate of VT incidence. Most sudden cardiac deaths are caused by VT/VF, at an estimated rate of approximately 300,000 deaths per year in the United States, or about half of the estimated cardiac mortality in this country. This translates to an incidence of 0.1-0.2.0% per year. This is only a rough estimate of VT incidence because many patients have nonfatal VT and because some sudden deaths are associated with VF or bradycardia, rather than VT.

The incidence of VT in developed countries is thought to be similar to that of the United States. In other regions, VT incidence correlates with the prevalence of coronary artery disease.

Occasional pockets of unusual heart disease cause locally increased risk of VT. Examples include the Greek island of Naxos (right ventricular dysplasia), parts of South America (Chagas disease), and northeastern Thailand (idiopathic VF).

Torsade de pointes is one of the few mechanisms of sudden death in structurally normal hearts. When polymorphic VT is observed in the absence of a cardiac channel defect, the most common causes are ischemia and myocarditis.

Sex: VT is observed more frequently in men because ischemic heart disease is more prevalent among men. Among patients with coronary artery disease in the Framingham study, male deaths were more common than female deaths (46% versus 34%) (Gordon, 1971).

Age: The incidence of ischemic VT increases with age, regardless of sex, as the prevalence of coronary artery disease increases.

Among patients younger than 35 years, the most common causes of sudden death, and presumably of VT, include hypertrophic cardiomyopathy, right ventricular cardiomyopathy, myocarditis, and long QT syndrome. Idiopathic VT is not associated with sudden death and can be observed at any age.

**Causes**

The most common cause of VT is coronary artery disease.

Reentrant circuits may form and typically include the border zone between electrically inactive scar tissue and electrically active myocardial tissue. Paradoxically, the slower electrical conduction within such border zones predisposes to the development of fast reentrant arrhythmia circuits.

In developed countries, coronary artery disease is the most common cause of myocardial scar. The substrate for electrical reentry (VT) may occur with any process that creates myocardial scar tissue, including the dilated cardiomyopathies, hypertrophic cardiomyopathy, right ventricular dysplasia, Chagas disease, and surgical incisions in the ventricle.

The torsade de pointes form of polymorphic VT is related to acquired or congenital QT interval prolongation.

Acquired QT prolongation is observed with certain potassium channel blocking medications. Most of the causative drugs block the delayed rectifier cardiac potassium current, IKr (HERG), and include quinidine, erythromycin, haloperidol, and many others. For a current list of these medications, see QTDrugs.org.

Congenital long QT syndrome is a group of genetic disorders involving abnormal cardiac ion channels, most commonly the potassium channels that determine the rate of ventricular repolarization.

In both acquired and congenital long QT syndromes, prolonged repolarization predisposes to torsade de pointes, which is presumed to be a reentrant rhythm. Other inherited ion channel abnormalities may cause idiopathic VF and familial polymorphic VT in the absence of QT interval prolongation.

In some patients, monomorphic VT occurs in the absence of structural heart disease (idiopathic VT).

These VTs are named according to their sites of origin, often are exercise dependent, and most often arise from the right and left ventricular outflow tracts and the left ventricular septum. The QRS morphology during tachycardia can be used to predict the VT site.

Clinical behavior generally is consistent with an automatic, rather than reentrant, mechanism of origin. This becomes a critical point when treatment is considered. Although idiopathic VTs often respond to calcium channel blockers, these agents can cause hemodynamic collapse and death when administered to VT patients with structural heart disease.

Triggers of VT include ischemia and electrolyte abnormalities.

Hypokalemia is the most important arrhythmia trigger clinically, followed by hypomagnesemia. Hyperkalemia also may predispose to VT and VF, particularly in patients with structural heart disease.

Occasionally, VT is triggered by aggressive adrenergic stimulation, as is observed with cocaine use.

VT can be broadly classified according to its electrocardiographic appearance.

When the same QRS electrocardiographic wave repeats itself, the VT is considered monomorphic. This implies that the sequence of electrical activation within the ventricle is repetitive.

Monomorphic VT most commonly is due to repetitive activation of the same reentrant circuit within the ventricle.

Occasionally, monomorphic VT is related to repetitive automatic beats arising from the same ectopic focus.

When the QRS complex varies from beat to beat, the rhythm is described as polymorphic VT and suggests a variable electrical activation sequence. The most notorious, and probably the most common, form of polymorphic VT is torsade de pointes, a French term suggesting a "twisting of the points" of the QRS complexes over time. This term now is reserved for polymorphic VT observed in the setting of a prolonged QT interval. Other polymorphic VTs occasionally are observed during ischemia.

**Pathophysiology**

VT is a general term that includes any rapid rhythm, faster than 100-120 beats per minute, arising in the ventricle. Regardless of the arrhythmia mechanism, the severity of clinical symptoms will determine the urgency with which VT must be treated. During VT, cardiac output is reduced due to the rapid heart rate and lack of a properly timed or coordinated atrial contraction. Hemodynamic collapse is more likely when underlying left ventricular dysfunction is present or with very rapid rates. Diminished cardiac output may cause a cascade of diminished myocardial perfusion, worsening inotropic response, and degeneration to VF, resulting in sudden death.

When the ventricular activation sequence is constant, the electrocardiographic pattern remains the same, and the rhythm is called monomorphic VT. Alternatively, polymorphic VT occurs when the ventricular activation sequence varies.

Although most patients with VT have underlying structural heart abnormalities, monomorphic VT is observed occasionally in patients with structurally normal hearts. The clinical behavior of these VTs may be more consistent with triggered activity or abnormal automaticity. Polymorphic VTs occasionally are observed in structurally normal hearts when patients have genetic abnormalities of cardiac ion channels. Examples include long QT syndrome, idiopathic VF, Brugada syndrome, and certain familial adrenergic polymorphic VTs.

**CLINICAL**

**History**

Sustained VT may precede a significant hemodynamic collapse. When this rhythm is present, it should be addressed rapidly.

The main symptoms of VT are palpitation, lightheadedness, and syncope. Because VT often is nonsustained, patients frequently present with recurrent syncopal episodes in the setting of underlying structural heart disease.

Some patients describe a sensation of neck fullness, which may be related to increased central venous pressures and cannon A waves.

Dyspnea may be related to increased pulmonary venous pressures and occasional left atrial contraction against a closed mitral valve.

Faster VT rates are associated with lightheadedness or syncope related to diminished cerebral perfusion.

Anxiety often is present, regardless of whether syncope occurs.

Risk factors include prior myocardial infarction (see Myocardial Infarction), other known structural heart disease, or a family history of premature sudden death. VT must be considered part of the differential diagnosis in any syncopal patient with such a history.

Any patient with a strong family history of premature (<35 y) sudden death should be evaluated for long QT syndrome, Brugada syndrome, arrhythmogenic right ventricular dysplasia, and hypertrophic cardiomyopathy.

**Physical**

Physical findings during VT include tachycardia, which often is associated with hypotension and tachypnea.

Signs of diminished perfusion may be present, including diminished level of consciousness, pallor, and diaphoresis.

Jugular venous pressure may be high, and cannon A waves may be observed if the atria are in sinus rhythm.

The first heart sound may vary in intensity, and rales may be present.

Physical findings during sinus rhythm are associated with underlying structural heart disease and may include displacement of the point of maximal impulse (PMI), murmurs related to valvular heart disease, or hypertrophic cardiomyopathy, and an S3 gallop.

Rales may be present during sinus rhythm if uncompensated congestive heart failure is present.

Sinus rhythm often is interrupted by ventricular extrasystoles.

**Lab Studies**

Include electrolytes in an acute evaluation.

Consider serum and urine toxicology screens for cocaine metabolites and tricyclic antidepressants in accordance with the clinical history.

Check cardiac enzymes if clinical symptoms or signs of ischemia are present.

In some patients with spontaneous VT, genetic studies may be appropriate. Spontaneous polymorphic VT may be related to genetic mutations affecting ion channels, as occurs in long QT syndrome, Brugada syndrome, and idiopathic VF. Finally, some patients with drug-induced ventricular arrhythmias have underlying genetic and channel defects.

**Other Tests**

Signal-averaged ECG is a noninvasive test that often produces abnormal results in patients with VT related to prior infarct or right ventricular dysplasia. Quantification of T wave alternans also has been proposed as a noninvasive risk stratifier for sudden death risk.

Electrocardiography is the criterion standard for diagnosis of VT. If no pacing device is present, the challenge is to discriminate between VT and aberrantly conducted SVT. Brugada et al (1991) have developed the best diagnostic criteria. Specific findings for VT include the absence of RS complexes in the precordial ECG leads (V1-V6), RS duration greater than 100 milliseconds in any precordial lead, and ventriculoatrial dissociation.

Patients with tachyarrhythmias may present with syncope. The ECG should be screened carefully for myocardial infarction, conduction abnormalities, QT interval prolongation, precordial T-wave inversions, ventricular preexcitation, and ventricular hypertrophy.

Patients with pacemakers represent a special challenge because they can present with wide complex tachycardia (WCT) that is secondary to rapid ventricular pacing.

Possibilities include tracking of an atrial tachyarrhythmia in a dual-mode, dual-pacing, dual-sensing (DDD) or an atrial-triggered, ventricular-inhibited (VDD) device; endless loop tachycardia; inappropriate rate responsive pacing due to sensor problems or incorrect sensor programming; and overt pacemaker failure (runaway pacer).

The most common problem involves the patient whose device is tracking atrial fibrillation or flutter. In the absence of a mode-switching algorithm, a VDD or DDD pacer will respond by pacing the ventricle at the programmed upper rate limit of the device. Application of a magnet to the pacer generator may terminate endless loop tachycardia or drop the paced rate enough to allow diagnosis of the underlying atrial tachyarrhythmia.

Occasionally, patients with structural heart disease present with recurrent syncope or palpitations. In this setting, an arrhythmic cause of syncope may be sought. Options include Holter monitoring, which has a low yield, or event recording. The goal is to document the patient's rhythm during symptoms.

**TREATMENT**

The acute management strategy depends upon the immediate hemodynamic consequences of the arrhythmia. VT associated with loss of consciousness or hypotension is a medical emergency requiring immediate cardioversion. In a normally sized adult, this typically is accomplished with a 200- to 360-J monophasic shock or a clinically equivalent biphasic energy dose.

When the hemodynamic status is stable, the patient is well perfused, and no evidence for coronary ischemia or infarction is present, then a trial of intravenous medication may be considered. If left ventricular function is impaired, amiodarone, then lidocaine, are favored over procainamide due to the latter drug's potential for exacerbating congestive heart failure. If medical therapy is unsuccessful, synchronized cardioversion (50-200 J monophasic) following sedation is appropriate.

Amiodarone is a complex antiarrhythmic drug that deserves special mention. It generally is listed as a class III drug but has measurable class I, II, and IV effects.

In the setting of congestive heart failure, the best proven antiarrhythmic drug strategies appear to be the use of angiotensin-converting enzyme (ACE) inhibitors and chronic beta-receptor–blocking agents.

The ICD has changed the face of ventricular arrhythmia management. Like pacemakers, these devices can be implanted transvenously quickly in a low-risk procedure.

* Surgical Care

In the 1980s, ventricular arrhythmia surgery was explored at several centers, using excision and cryoablation of infarct zones to prevent recurrent VT. The high mortality rates of these procedures and the success of the ICD led to a decline in open surgical procedures for VT. Catheter ablation of VT remains an option for patients with recurrent ICD shocks or non-ICD patients with preserved ventricular function. Ablation is used to treat symptomatic VT, rather than to reduce sudden death risk.

Reentrant VT requires a slow conduction zone, and this usually is located along the border of a scarred zone of myocardium.

The small physical size of the slow conduction zone makes it an ideal target for focal ablation procedures.

Cell disruption can be achieved using radiofrequency energy or cryoablation via transvenous catheters during closed-chest procedures.

Because patients with ischemic VT often have multiple reentrant circuits, ablation typically is used as an adjunct to ICD therapy.

If VT arises from an automatic focus, the focus can be targeted for ablation.

In patients with structurally normal hearts, the most common form of VT arises from the right ventricular outflow tract (RVOT). Abnormal or triggered automaticity is the most likely mechanism, and focal ablation is curative in these patients. Ablation cure rates typically are greater than 90% in this setting.

Reentrant tachycardia occasionally arises from the RVOT in patients with right ventricular dysplasia and repaired tetralogy of Fallot patients. The tachycardia mechanism usually can be determined during EPS.

**Prognosis**

Prognosis varies with the specific cardiac process but is predicted best by left ventricular function. In patients with ischemic cardiomyopathy and nonsustained VT, sudden death mortality rates approach 30% in 2 years. In patients with idiopathic VT, the prognosis is excellent and the major risk is due to poorly timed syncopal spells. A few exceptions exist to the above rule. Patients with long QT syndrome, right ventricular dysplasia, and hypertrophic cardiomyopathy may be at increased risk of sudden death despite relatively preserved left ventricular function. These possibilities should be considered in any patient with a strong family history of premature sudden death.

**ATRIAL FIBRILLATION**

Atrial fibrillation (AF) is a common arrhythmia and a significant public health problem in the United States, affecting 2.2 million Americans and almost 5% of the population older than 69 years. The prevalence of AF increases with advancing age. Data from the Framingham heart study show that AF is associated with a 1.5- to 1.9-fold higher risk of death, which may be due to thromboembolic stroke. While patients can be asymptomatic, many experience a wide variety of symptoms, including palpitations, dyspnea, fatigue, dizziness, angina, and congestive heart failure (CHF). In addition, the arrhythmia can be associated with hemodynamic dysfunction, tachycardia-induced cardiomyopathy, and systemic embolism.

Overall, approximately 15-25% of all strokes in the United States (75,000/y) can be attributed to AF. Known risk factors include male sex, valvular heart disease (rheumatic valvular disease), CHF, hypertension, and diabetes. Additional risk factors, such as advanced age and prior history of stroke, diabetes, and hypertension, place patients with preexisting AF at even higher risk for further comorbidities such as stroke. Patients with nonvalvular AF and risk factors have a 5-fold increased risk for stroke. Patients with rheumatic heart disease and AF have an even higher risk for stroke (17-fold). At least 4 large clinical trials have clearly demonstrated that anticoagulation with warfarin decreases the risk of stroke by 50-80%.

Given the frequency of these comorbidities, management can result in significant medical costs. Therapeutic goals include rate control, maintenance of sinus rhythm, and prevention of thromboembolism. Additionally, current practice and economic pressures force many physicians to reconsider outpatient treatment options.

**Frequency**

AF affects 2.2 million Americans. It can occur in the absence of comorbidities, as it does in 10-15% of individuals (lone AF); however, AF is associated more frequently with hypertension; organic heart disease; CHF; ischemic heart disease; and valvular, dilated, hypertrophic, restrictive, and congenital cardiomyopathies. Paroxysmal AF is commonly associated with cardiac surgery, pulmonary disease, thyrotoxicosis, acute ethanol intoxication, and electrolyte imbalance. Given the almost epidemic proportions of patients with AF, clinicians must be aware of the multiple mechanisms and presentations and then correct the underlying etiology, if possible. For example, a logical decision may be to correct an overactive thyroid gland before attempting cardioversion.

AF is strongly age-dependent, affecting 4% of individuals older than 60 years and 10% of persons older than 80 years. The rate of ischemic stroke among elderly patients not treated with warfarin averages approximately 5% per year.

**Causes**

The most common cause is advancing age; however, uncontrolled hypertension, coronary disease, CHF, valvular heart disease, acute pulmonary processes, hyperthyroidism, acute alcohol intoxication (ie, holiday heart, Saturday night heart), and illicit narcotic abuse should also be considered.

**Pathophysiology**

Several classification schemas have been proposed for the study of AF, but none fully accounts for all aspects of AF. A number of different labels and nomenclature have been used to describe patterns of AF, including acute, chronic, paroxysmal, intermittent, and permanent. The vagaries of each of these definitions make comparing the results of studies assessing the magnitude and treatment of AF difficult.

Recently published guidelines from expert committees of the American College of Cardiology/American Heart Association and European Society of Cardiology on the treatment of patients with AF suggest that AF be classified into 3 patterns. These include a first detectable episode, irrespective of whether it is symptomatic or self-limited. Recurrent AF is considered to be present when a patient has 2 or more episodes of AF. If AF terminates spontaneously, then recurrent AF is designated as paroxysmal; if this arrhythmia becomes sustained, then AF is considered persistent (irrespective of whether AF is terminated with pharmacologic therapy or electrical cardioversion).

Persistent AF may be either the first presentation of AF or the result of recurrent episodes of paroxysmal AF. Patients with persistent AF also include patients with long-standing AF in whom cardioversion has not been indicated or attempted, often leading to permanent AF. Permanent AF is recognized as the accepted rhythm, and the only treatment goals are rate control and anticoagulation.

This classification schema pertains to cases that are not related to a reversible cause of AF (eg, thyrotoxicosis, electrolyte abnormalities, acute ethanol intoxication). The occurrence of AF secondary to acute myocardial infarction, cardiac surgery, pericarditis, pulmonary embolism, or acute pulmonary disease is considered separately because in these situations, AF is less likely to recur once the precipitating condition has been resolved and adequately treated.

Some patients with paroxysmal AF, typically younger patients, have been found to have distinct electrically active foci within their pulmonary veins. These patients generally have many atrial premature beats noted on Holter monitoring. Isolation or elimination of these foci can lead to elimination of the trigger for paroxysms of AF.

Patients can also have AF as a secondary arrhythmia associated with cardiac disease that affects the atria (eg, CHF, hypertensive heart disease, rheumatic heart disease, coronary artery disease [CAD]). These patients tend to be older, and AF is more likely to be chronic. Paroxysmal AF may progress to chronic AF, and aggressive attempts to restore and maintain sinus rhythm may prevent comorbidities associated with AF.

Persistent AF with an uncontrolled response can cause dilated cardiomyopathy and can lead to electrical remodeling in the atria (atrial cardiomyopathy). Therapy, such as drugs or atrioventricular (AV) nodal ablation and permanent pacemaker implantation, to control the ventricular rate can improve left ventricular (LV) function and improve quality-of-life scores.

New developments aimed at curing AF are being actively explored. By reducing the critical mass required to sustain AF with either surgical or catheter-based compartmentalization of the atria (ie, Maze procedure), fibrillatory wavelets collide with fixed anatomic obstacles, such as suture lines or complete lines of ablation, thus eliminating or reducing the chance of chronic AF. Some patients with focal origins of their AF also may be candidates for catheter ablation. Still, much remains to be accomplished before either of these procedures is appropriate for routine use.

**CLINICAL**

History

Focus the initial evaluation on hemodynamic stability and potential causes. Consider immediate electrical cardioversion for patients with impending hemodynamic collapse or evidence of cardiac ischemia.

Initial evaluation

A thorough history, physical examination, and 12-lead ECG are key to making an accurate diagnosis.

Examine the ECG tracings for evidence of Wolff-Parkinson-White syndrome, which may predispose a small percentage of patients to sudden cardiac death. In these patients, AF can lead to rapid ventricular rates and, subsequently, degenerate into ventricular fibrillation, especially when AV nodal blocking agents are used.

**Physical**

The physical examination determines whether patients need immediate restoration of sinus rhythm or whether they can be treated more conservatively.

Vital signs - Assessment of vital signs and oxygen saturation for evidence of pulmonary vascular congestion

Head and neck - May reveal elevated venous pressures or cyanotic lips

Pulmonary - May reveal rales and evidence of pleural effusion

Cardiac - May reveal CHF with a displaced point of maximal impulse and an S3 and other potential important murmurs

Abdomen - May reveal ascites and slight pressure in the area of the liver; may produce a hepatojugular reflex consistent with passive liver congestion and CHF

Lower extremities - May reveal edema

**Lab Studies**

Perform at least one thyroid function study to exclude hyperthyroidism.

Perform a CBC count.

Measure electrolytes and BUN/creatinine.

Measure the digoxin level if toxicity is suspected.

A toxicology screen or ethanol level may be appropriate in the right clinical scenario to rule out acute intoxication as the cause of AF.

**Imaging Studies**

* Electrocardiogram

ECG findings usually confirm the diagnosis of AF.

The ventricular rate typically is irregular.

Discrete P waves are absent; instead, undulating fibrillatory (f) waves are present.

* In hemodynamically stable patients, obtaining a transthoracic echocardiogram is reasonable to evaluate for LV function and cardiac chamber size, valvular heart disease, pulmonary hypertension, LV hypertrophy, and pericardial effusion.

In some instances, performing a transesophageal echocardiogram (TEE) is desirable to look for specific evidence of left atrial or LV thrombus prior to cardioversion. If thrombus is present, cardioversion should be delayed because the restoration of atrial mechanical contraction associated with conversion to sinus rhythm may cause embolization in the immediate postcardioversion period or as long as 2-3 weeks later. While left atrial smoke (or spontaneous contrast) is present in many patients, it does not imply thrombus. Synchronized cardioversion can be accomplished safely if left atrial and LV thrombi are not present. Despite a TEE finding that is negative for thrombus, patients still require anticoagulation with heparin and warfarin after cardioversion.

**Procedures**

* Synchronized direct current

Cardioversion is synchronized to the R wave to prevent the potential for a nonsynchronized shock to be delivered during the vulnerable phase of the T wave and the initiation of ventricular fibrillation.

Direct-current cardioversion is used to restore sinus rhythm if patients are anticoagulated adequately with a therapeutic international normalized ratio (INR) and remain in AF.

In most patients, the procedure can be performed safely in an outpatient setting.

Administer either intravenous sedation, according to sedation guidelines, or general anesthesia for patient comfort and safety.

Defibrillating patches are positioned in various positions, including the right anterior and left posterior positions to allow electrical vectors to defibrillate the atria, and in more standard positions such as anterior and lateral positions.

A high success rate should be expected, with the realization that some patients remain in sinus rhythm transiently, only to revert back to AF. If initially unsuccessful, consider using an intravenous infusion of ibutilide (1 mg), which reduces the defibrillation thresholds, or use different electrical vectors. Newer defibrillators with biphasic waveforms have been demonstrated to be more efficacious in converting AF to sinus rhythm. Occasionally, performing internal cardioversion may be necessary.

* Synchronized electrical cardioversion for acute onset

Consider immediate cardioversion for patients with AF of less than 48 hours' duration because the risk of embolic stroke is small. If the exact time cannot be determined accurately or is longer than 48 hours in duration, patients should receive either a TEE to assess for atrial thrombus prior to cardioversion or anticoagulation therapy for 4 weeks with an INR goal of 2-3 prior to elective cardioversion. If AF is longer than 48 hours in duration and the TEE shows no evidence of atrial thrombus, anticoagulation is still indicated postprocedure, and the initiation of intravenous heparin is warranted while warfarin therapy is initiated and while the INR rises to therapeutic levels. In the future, low–molecular-weight heparin may be used in outpatient settings in conjunction with warfarin therapy.

Alternatively, use the traditional right anterior and left lateral position of patches or external paddles. Once the patient is adequately sedated, a synchronized shock of 200-360 J is usually required for conversion to sinus rhythm. Body habitus and urgency are guides to choosing the shock energy. For example, a person who is obese with a large anteroposterior diameter probably will require 360 J, and an elderly person with thin body habitus might require only 200 J. Required energies are lower with biphasic waveforms.

Two sets of patches can be used to successfully cardiovert patients in whom a single maximal shock with 1 set of patches fails. Two external defibrillators and 2 sets of defibrillation patches are required. Vectors are crossed so that 1 set of patches is placed anteriorly left of the sternum and posteriorly right of the spine. The second set of patches is placed anteriorly right of the sternum and posteriorly left of the spine. The 2 defibrillators may be activated simultaneously, in synchronized fashion, or with a slight delay after the first discharge. This method may succeed when a single set of patches fails.

Internal cardioversion is also possible and requires an electrophysiologist and an electrophysiologic laboratory. Typically, intracardiac catheters are positioned in the right atrium and in the coronary sinus. Synchronized shocks are delivered between the 2 catheters, with energies from 1-100 J. Alternatively, defibrillating current may be passed between a single intracardiac catheter (right atrium, coronary sinus) and a single cutaneous patch placed anteriorly or posteriorly.

External cardiac defibrillators with biphasic shocking waveforms have received approval from the US Food and Drug Administration (FDA). In 2003, Neal et al reported that these devices have been shown to dramatically reduce energy requirements and improve efficacy compared with standard monophasic devices.

* Chemical conversion to sinus rhythm

Hemodynamically stable patients with AF can be converted to sinus rhythm with large doses of oral agents or with intravenous agents. Large single doses of flecainide (300 mg) or propafenone (450-600 mg) given orally have been shown to convert patients to sinus rhythm. The drug is given in a monitored setting, preferably in an inpatient setting. Patients should not have a QTc longer than 460 milliseconds and should not be taking concomitant antiarrhythmic agents.

Alternatively, patients can be given intravenous doses of procainamide (<18 mg/kg/h) or ibutilide (1 mg over 15 min, may be repeated after 30 min). Some clinicians also preload patients with intravenous magnesium (2 g) prior to infusing ibutilide as a preventative measure for torsade de pointes (TdP). Both agents should be administered in a highly monitored setting (ICU), with an external defibrillator at the bedside, and with special care to ensure electrolyte values are within normal limits. Monitor blood pressure and heart rate, and monitor for signs of QRS widening (ie, with frequent ECGs).

Patients with CHF and cardiogenic shock are not candidates for this type of procedure. Involving a cardiologist or electrophysiologist would be reasonable if intravenous conversion is being considered because TdP has been documented.

**TREATMENT**

The initial goal in the management of AF is rate control and anticoagulation, with an eventual goal of restoration and maintenance of sinus rhythm. However, few prospective, randomized controlled trial data are available to indicate to the clinician whether patients with symptomatic or asymptomatic AF do better simply with rate control and anticoagulation or with aggressive attempts to maintain sinus rhythm with repeated electrical cardioversion and antiarrhythmic agents.

Restoration of sinus rhythm with regularization of the heart's rhythm improves cardiac hemodynamics and quality of life and reduces the risk of thromboembolic complications. Additional benefits include maintaining the appropriate physiologic responses to exercise and the atrial contribution to the cardiac output, thus preventing atrial dilation and possible LV dysfunction. Thus, most clinicians initially focus on rhythm control, and rate control is pursued only when rhythm control fails.

* Rate control

Beta-blockers and calcium channel blockers are the drugs of choice for rate control. These drugs can be administered intravenously and orally. They are effective at rest and with exertion. Caution should be exercised in patients with reactive airway disease given beta-blockers.

Digoxin can be used in the acute setting but does little to control the ventricular rate in active patients. In addition, it has no antiarrhythmic effects and actually may promote AF by shortening atrial refractory periods. Digoxin is indicated in patients with reduced LV function.

* Anticoagulation

AF is recognized as a powerful risk factor for stroke. One of the most important concerns in treating patients is the need for anticoagulation. Effective anticoagulation in patients with AF reduces the risk of stroke 3-fold. Patients with newly diagnosed AF and patients awaiting electrical cardioversion can be started on intravenous heparin (activated partial thromboplastin time [aPTT] of 45-60 seconds).

Patients can be concomitantly started on warfarin in an inpatient setting while awaiting a therapeutic INR value. Many practices have developed specialized anticoagulation clinics to closely monitor INR values.

In the future, low–molecular-weight heparins will be used to facilitate initial outpatient anticoagulation until warfarin is therapeutic. Studies are currently being conducted to assess feasibility and safety.

* Long-term prophylaxis

Patients have a risk of stroke or peripheral embolism that is approximately 5 times that of people in sinus rhythm. This risk can be reduced greatly with warfarin.

* Acute conversion to sinus rhythm

The decision to restore sinus rhythm acutely depends on the hemodynamic status of the patient.

Consideration of impending hemodynamic collapse or acute cardiac ischemia may influence the decision to cardiovert immediately.

* Elective conversion to sinus rhythm

Exactly how long AF must be present before the risk of atrial thrombus and subsequent thromboembolism develops is uncertain. Certainly, the longer patients are in AF, the more likely they are to develop atrial thrombus. In general, if the arrhythmia is present for less than 48 hours, cardioversion can be accomplished safely without further need for anticoagulation. If uncertain, a TEE could be performed to exclude left atrial thrombus. In any case, acute conversion must be accompanied by anticoagulation.

In patients with AF longer than 48 hours in duration or of unknown duration, cardioversion is not recommended until sufficient anticoagulation is achieved. The most conservative route is to anticoagulate with warfarin for 3-4 weeks prior to any attempt to restore sinus rhythm.

Because embolic events can occur following cardioversion as atrial mechanical function returns, continue anticoagulation for an additional 4-6 weeks. Alternatively, initially perform a TEE, and if no thrombus is present, cardiovert and therapeutically anticoagulate for 4-6 weeks after sinus rhythm is restored.

* Pacing to prevent AF

The fact that ventricular-based pacing (VVI mode) does not prevent AF in patients with sick sinus syndrome is well documented. Retrospective data suggest that atrial-based pacing (AAI, DDD modes) reduces the risk of developing AF and increases the interval between episodes in patients with sick sinus syndrome.

In addition, biatrial pacing and dual-site atrial pacing may reduce the prevalence of AF. This is currently an active area of research. Which areas are the best for lead placement (ie, high right atrial, coronary sinus, left atrial sites) remain unknown.

* Atrial defibrillators

Early conversion to sinus rhythm is desirable because patients do not require long-term anticoagulation. AF-free intervals have been shown to be prolonged with early AF termination.

Defibrillation energy requirements can be higher than the patient's pain threshold. This can represent a problem in some patients. Painless therapy (eg, atrial burst pacing to extinguish AF) is successful approximately 20% of the time, making this form of therapy desirable in patients with poorly tolerated AF.

In addition, the next generation of ventricular defibrillators incorporates atrial defibrillation therapies. Patients with reduced LV function and a tendency toward heart failure with episodes of AF will benefit greatly from early conversion to sinus rhythm.

Antiarrhythmic agents are commonly used to prevent AF recurrence. Currently, the FDA has approved 5 antiarrhythmic drugs for the treatment of AF (ie, quinidine, flecainide, propafenone, sotalol, dofetilide); however, other antiarrhythmic agents (eg, amiodarone) are used in an off-label fashion with great clinical efficacy. Use antiarrhythmic agents with great caution because they can cause proarrhythmia, exacerbate a preexisting arrhythmia, or provoke a new arrhythmia. Proarrhythmia can be bradycardic or tachycardic, atrial or ventricular. In addition, adverse effects can cause severe comorbidities and even death. Consultation with a cardiac electrophysiologist or knowledgeable physician is recommended prior to antiarrhythmic drug initiation.

Surgical Care: Since its inception, surgical compartmentalization of the atria, or the Maze procedure, has evolved as an exciting procedure with a potential to cure AF. Quite simply, the atria are transected and resutured to reduce the critical mass required for the maintenance of AF. Early experience shows that atrial transport is restored postoperatively and that long-term anticoagulation is not required. The downside remains the need for an open chest procedure; however, thoracoscopic procedures may reduce hospitalization and recovery times in the future. The surgical Maze procedure remains an attractive procedure for patients with AF who are undergoing concomitant mitral valve procedures. Its role as a primary therapy for AF is doubtful. Catheter ablation has taken the following 3 paths in the attempt to cure or manage AF.

**ATRIAL FLUTTER**

Synonyms: AF, atrial flutter

Some patients have both atrial flutter and atrial fibrillation. The elimination of atrial flutter has been noted to reduce or eliminate episodes of atrial fibrillation. Left untreated, persistent atrial flutter can degenerate into chronic atrial fibrillation. Uncommon forms of atrial flutter have been noted during long-term follow-up in as many as 26% of patients with surgical correction of congenital cardiac anomalies.

**Frequency**

Atrial flutter is much less common than atrial fibrillation. Of patients admitted to US hospitals with a diagnosis of supraventricular tachycardia between 1985 and 1990, 77% had atrial fibrillation and 10% had atrial flutter.

Prognosis depends on the patient's underlying medical condition. Any atrial arrhythmia can cause a tachycardia-induced cardiomyopathy. Intervening to control the ventricular response rate or to return the patient to sinus rhythm is important. Thrombus in the left atrium has been described in patients with atrial flutter (0-21%). Thromboembolic complications have also been described.

Atrial flutter is associated with a male predominance. In one study of 100 patients with atrial flutter, 75% were men.

Patients with atrial flutter, as with atrial fibrillation, tend to be older adults. In one study, the average age was 64 years (range 27-86 y).

**Causes**

Unlike atrial fibrillation, type I atrial flutter requires the necessary electroanatomic elements to be in place.

These elements include an area of relatively slow conduction (the tricuspid valve isthmus).

Many patients with atrial flutter also have depressed LV function.

Type II atrial flutters are generally found in patients after heart surgery.

**Pathophysiology**

In most studies, approximately 30% of patients have no underlying cardiac disease, 30% have coronary artery heart disease, and 30% have hypertensive heart disease. Other conditions are also associated with atrial flutter, including cardiomyopathy, hypoxia, chronic obstructive pulmonary disease, thyrotoxicosis, pheochromocytoma, electrolyte imbalance, and alcohol consumption.

Animal models have been used to demonstrate that an anatomical block (surgically created) or a functional block of conduction between the superior vena cava and inferior vena cava, similar to the crista terminalis in the human right atrium, is key to initiating and maintaining the arrhythmia.

In humans, the common form of atrial flutter involves a single reentrant circuit with circus activation in the right atrium around the tricuspid valve annulus (in a counterclockwise direction), with an area of slow conduction located between the tricuspid valve annulus and the coronary sinus ostium (subeustachian isthmus). The crista terminalis acts as another anatomic conduction barrier, similar to the line of conduction block between the 2 venae cavae required in the animal model. The orifices of both venae cavae, the eustachian ridge, the coronary sinus orifice, and the tricuspid annulus complete the barrier for the reentry circuit. Common type I atrial flutter has caudocranial activation (ie, counterclockwise around the tricuspid valve annulus when viewed in the left antero-oblique fluoroscopic view) of the atrial septum. Uncommon type I atrial flutter has the opposite activation sequence (ie, clockwise activation around the tricuspid valve annulus).

Other atypical atrial flutters are less extensively studied and electroanatomically characterized. Atypical atrial flutters may originate from the right atrium (surgical scars [ie, incisional reentry]) or from the left atrium (pulmonary veins [ie, focal reentry] or mitral annulus). Tricuspid isthmus dependency is not a prerequisite for atrial flutter.

CLINICAL

**History**

The severity of symptoms and the patient's underlying cardiac condition dictate the initial management approach.

Address symptoms of other noncardiac conditions (eg, hyperthyroidism) that are associated with atrial flutter.

Most clinicians believe that patients with atrial flutter, like those with atrial fibrillation, of a duration greater than 36-48 hours require anticoagulation with warfarin prior to conversion to sinus rhythm, unless transesophageal echocardiography (TEE) is performed first and shows the absence of thrombus. Thus, the duration of the episode and the initial time of atrial fibrillation or flutter may affect the timing of cardioversion and the need to address anticoagulation.

The most common symptom is palpitations. Other symptoms include fatigue, dyspnea, and chest pain.

Often, atrial flutter is not as well tolerated as atrial fibrillation. This may be due to the rapid and difficult-to-control ventricular response, especially with minimal exertion.

Atrial flutter can cause hypotension, angina, or congestive heart failure due to rapid ventricular response in the setting of compromised left ventricular (LV) function.

**Physical**

The general appearance and vital signs of the patient are very important when determining the urgency with which to restore sinus rhythm. Thus, the initial cardiopulmonary evaluation and monitoring for signs of cardiac or pulmonary failure help guide initial management. With typical atrial flutter, atrioventricular (AV) conduction usually is 2:1, making the ventricular rate approximately 150 beats per minute (bpm).

Different subgroups of atrial flutter are described with electroanatomic correlations.

Type I atrial flutter is described well both anatomically and electrically, with atrial rates from 240-340 bpm.

Type II atrial flutter is associated with atrial rates from 340-433 bpm and is not as well characterized electrically as type I.

Type I atrial flutter can always be electrically entrained and interrupted by atrial pacing at faster rates than the intrinsic rate of the tachycardia, whereas type II atrial flutter cannot be electrically entrained.

For typical atrial flutter, pacing from the tricuspid valve isthmus during atrial flutter causes concealed entrainment such that the morphology of the paced flutter waves is identical to that of the native flutter waves.

Type I atrial flutter is also known as typical (or common form) or counterclockwise atrial flutter. In typical flutter, flutter waves are negative in ECG leads II, III, and aVF and positive in V1. The inferior leads show a "sawtooth" pattern.

The uncommon form of type I atrial flutter may have positive flutter waves in ECG leads II, III, and aVF and negative flutter waves in V1 (during clockwise flutter).

Both typical and uncommon forms of type I atrial flutter share the same reentrant circuit, but they activate the atria in opposite directions. The common form exhibits counterclockwise activation of the atria, and the uncommon form exhibits clockwise activation of the atria.

**Lab Studies**

The history and physical examination findings guide laboratory studies. Asymptomatic hyperthyroidism, especially in elderly patients, can manifest with atrial fibrillation or flutter; therefore, obtain thyroid function tests. Hyperthyroidism is a rare cause of atrial flutter and should be excluded with blood testing.

**Imaging Studies**

* Electrocardiography
  1. The common form of type I atrial flutter has sawtooth flutter (F) waves, best seen in leads II, III, and aVF, with atrial rates of 240-340 bpm and without an isoelectric interval between these F waves.
  2. The ventricular response may be regular or irregular.
  3. The ventricular rate is a fixed mathematical relationship of F waves and the resulting QRS complexes.

Variable AV conduction can also be seen (commonly present with 2:1 or 3:1 AV conduction). With 1:1 AV conduction, hemodynamic collapse may occur.

Morphology of the flutter wave can predict findings in the electrophysiology laboratory. A negative flutter wave in the inferior limb leads and a positive flutter wave in V1 are highly predictive of a counterclockwise circuit; however, with positive flutter waves in the inferior limb leads and negative flutter waves in V1, differentiating between clockwise type I atrial flutter and atypical forms of non–isthmus-dependent intra-atrial reentry is difficult.

* Transthoracic echocardiography can be useful when evaluating underlying cardiac function.

In addition, structural abnormalities, evidence of coronary artery disease, LV function, pericardial fluid, or evidence of LV thrombus can be sought.

If immediate cardioversion is considered, TEE may be useful to help exclude thrombus from the left atrium or appendage.

**Procedures**

Cardioversion

The timing of cardioversion depends on the hemodynamic need for sinus rhythm.

Patients with chronic atrial flutter benefit from anticoagulation in the same manner as patients with atrial fibrillation.

**TREATMENT**

Medical Care

General goals for the treatment of symptomatic atrial flutter are similar to those for atrial fibrillation and include

(1) control of the ventricular rate,

(2) termination of sustained episodes,

(3) prevention and decreased frequency or duration of recurrent episodes,

(4) prevention of thromboembolic complications,

(5) minimization of adverse effects from therapy.

However, these goals can be modified for each patient. In an acute setting with pending hemodynamic collapse, follow the adult advanced cardiac life support algorithms for managing atrial fibrillation and flutter. Consider immediate electrical cardioversion for patients who are hemodynamically unstable.

1. Ventricular rate control: Ventricular rate control is a priority because it may alleviate symptoms. Rate control is typically more difficult for atrial flutter than for atrial fibrillation.
   * Calcium channel blockers and beta-blockers

Ventricular rate control can be achieved with drugs that block the AV node. Intravenous calcium channel blockers (eg, verapamil, diltiazem) or beta-blockers can be used, followed by initiation of oral agents.

Hypotension and negative inotropic effects are concerns with the use of these medications.

A history of Wolff-Parkinson-White syndrome or evidence of ventricular preexcitation should be determined because agents that act exclusively at the level of the AV node may enhance accessory pathway conduction.

* Electrical cardioversion

The success rate of electrical cardioversion is higher than 95%.

Factors to consider include synchronization of shocks to R waves, adequate sedation, and electrode position (apex anterior, apex posterior, anteroposterior).

Atrial flutter generally requires less energy for conversion than atrial fibrillation, and as few as 50 joules may be necessary. Recent data show that an initial energy of 100 joules is more effective and is less likely to induce atrial fibrillation than 50 joules.

If cardioversion is not successful with one electrode configuration, switching may improve success. A second set of electrodes can be used with tandem or simultaneous shocks.

Newer devices with different outputs and biphasic external waveform may be more effective in restoring sinus rhythm.

A few points to remember about the cardioversion technique include a wide electrode separation in the anteroapex position, the application of pressure on paddles or electrodes to reduce thoracic impedance, and the placement of electrode patches under or lateral to the breasts in women.

* Pharmacological cardioversion

Procainamide is effective for converting atrial flutter to sinus rhythm in 0-13% of patients.

Flecainide is effective in approximately 10% of patients.

Dofetilide is effective in 70-80% of patients.

Ibutilide is effective, converting recent-onset atrial flutter to sinus rhythm in 63% of patients with a single infusion.

Large single oral doses of type IC antiarrhythmic agents, such as propafenone (450-600 mg) or flecainide (200-300 mg), have also been shown to be effective in converting recent-onset atrial fibrillation to sinus rhythm. Their use in atrial flutter can be assumed to have at least equal success.

Oral amiodarone during the loading period (>1 mo) converted 18% of atrial fibrillations or flutters to normal sinus rhythms. Intravenous amiodarone is also effective in converting atrial flutter to sinus rhythm and in slowing the ventricular rate in patients with a rapid ventricular response.

Atrial overdrive pacing: This procedure can be performed invasively or through the use of a transesophageal electrode to pace the left atrium, with a success rate of approximately 50%.

Combination of the above treatments: Antiarrhythmic medication prior to electrical cardioversion or rapid atrial pacing has been shown to improve the rate of conversion to sinus rhythm.

* Radiofrequency ablation: This treatment modality is not usually used in the acute setting.

Prevention (decrease frequency or duration of recurrence episodes): After the initial episode is terminated and the underlying disease is treated, the patient may not need any further intervention except avoidance of the precipitating factor (eg, alcohol, caffeine). For atrial fibrillation, approximately 30% of patients remain in sinus rhythm at 1 year without antiarrhythmic therapy.

* Thromboembolic

Patients with atrial flutter are at increased risk of thromboembolic complications compared with the general population. Most clinicians anticoagulate patients with atrial flutter as they would patients with atrial fibrillation. Thus, use the same anticoagulation strategy used in patients with atrial fibrillation.

Patients with atrial flutter and episodes of atrial fibrillation are at higher risk of thromboembolic events; however, determining whether episodes of atrial fibrillation are associated with episodes of atrial flutter is difficult.

Medications play a major role in the therapy of atrial flutter. Agents can be used to control the ventricular rate, terminate acute episodes, prevent or decrease the frequency or duration of recurrent episodes, and prevent complications. Various categories of drugs are used to treat atrial flutter. Drug initiation in an outpatient setting is generally accepted in patients without underlying structural heart disease who are in sinus rhythm. In addition, many specialists initiate outpatient drug therapy in patients with therapeutically anticoagulated atrial flutter who are awaiting outpatient electrical cardioversion in the near future. Regardless, close patient follow-up is mandated, with frequent ECG monitoring or transtelephonic monitoring for potential signs of proarrhythmia.

**Further Inpatient Care**

Base the decision to admit a patient with atrial flutter on hemodynamic considerations and the timing of cardioversion to sinus rhythm.

For atrial flutter of less than 48 hours in duration, attempt cardioversion as soon as possible. Postconversion anticoagulation is usually unnecessary, although data from TEE studies indicate that postconversion anticoagulation a reasonable option.

For episodes of atrial flutter of uncertain duration or greater than 48 hours, begin anticoagulation therapy. If cardioversion is needed sooner, anticoagulate patients with intravenous heparin and perform TEE as close to the time of cardioversion as possible. Patients still require anticoagulation for at least 4 weeks after cardioversion.

If thrombus is observed or suspected based on TEE findings, delay cardioversion. Rate control and therapeutic anticoagulation is required for a minimum of 4 weeks.

Cases have been reported of patients with atrial fibrillation who have experienced either a transient ischemic attack or stroke after cardioversion despite having no evidence of thrombus from the TEE. While atrial flutter is considered less thrombogenic than atrial fibrillation, neurological consequences can result from cardioversion.

In patients with recurrent episodes of common type I atrial flutter that are not well-tolerated or in patients in whom adverse effects from medical therapy (rate control or antiarrhythmic drugs) have developed, offer electrophysiologic study with an eye toward radiofrequency catheter ablation. Given the high success rate and low complication rate, radiofrequency ablation is superior to medical therapy. The authors believe the best plan is to offer radiofrequency ablation before antiarrhythmic medication.

Consider inpatient initiation of long-term antiarrhythmic drugs, especially for patients with underlying structural heart disease or baseline prolonged QT interval. The risk of proarrhythmia is probably greatest during the first 24-48 hours after the initiation of antiarrhythmics, but lack of proarrhythmia during antiarrhythmic drug loading may yield a false sense of security because proarrhythmia is often not predictable. Pause-dependent torsades de pointes can occur after conversion to sinus rhythm.

**Further Outpatient Care**

Closely monitor the patient's anticoagulation therapy, with a target INR of 2-3. Take special care when additional medications (including antibiotics) are added because they may cause dramatic alterations in INR values.

**In/Out Patient Meds**

Anticoagulant therapy (ie, heparin and/or warfarin) is indicated, especially when the onset of atrial flutter is of more than 48 hours' duration or is uncertain.

Patients need to maintain a therapeutic INR for 3 weeks prior to conversion and for at least 4 weeks after conversion to sinus rhythm.

Long-term anticoagulation is recommended for patients with chronic atrial flutter.

Anticoagulants are used to decrease thromboembolic complications.

Preferred medications that slow AV node conduction include beta-blockers (eg, atenolol, metoprolol, propranolol) and calcium channel blockers (eg, verapamil, diltiazem).

These medications are used to control ventricular rates.

Also use these medications in patients who are taking class IA or IC antiarrhythmic drugs (to prevent rapid ventricular response, which can occur when the atrial rate is slowed).

Antiarrhythmic drugs are indicated for the termination of acute episodes or the prevention of recurrent episodes.

For atrial flutter, electrical cardioversion is effective and usually requires less energy than for atrial fibrillation.

Catheter ablation offers a potential cure and may be safer than long-term use of an antiarrhythmic agent.

**Complications**

The major potential complication with atrial flutter (or atrial fibrillation) is neurologic insult, either transient ischemic attack or stroke. This risk can be minimized with proper anticoagulation. Consider patients with common type I atrial flutter for catheter ablation to eliminate the need for long-term anticoagulation and antiarrhythmic medications.

**Prognosis**

Atrial flutter itself is not considered a life-threatening arrhythmia; however, uncontrolled ventricular rates can lead to impaired ventricular function. Additionally, patients with Wolff-Parkinson-White syndrome can develop life-threatening ventricular responses. Consider these patients for catheter ablation of their accessory bypass tract. Data from the Framingham study suggest that patients with atrial fibrillation do not live as long as patients without atrial fibrillation (ie, controls). No data are available on atrial flutter.

The prognosis for patients with type I atrial flutter who undergo catheter ablation is excellent, with a very low recurrence rate. The picture is not as clear for patients with both atrial flutter and atrial fibrillation. Some reports have documented fewer episodes of atrial fibrillation after successful flutter ablation, while others have not. Atrial fibrillation is thought to possibly be more responsive to antiarrhythmic agents after atrial flutter has been eliminated.

Numerous reports indicate that patients with atrial fibrillation who are given class IC antiarrhythmic agents may convert to atrial flutter with faster ventricular rates. Thus, patients receiving type IC agents (flecainide) should also receive an AV nodal blocking drug such as a beta-blocker or calcium channel blocker.

**VENTRICULAR FIBRILLATION**

Ventricular fibrillation (VF) is a pulseless arrhythmia with irregular and chaotic electrical activity and ventricular contraction in which the heart immediately loses its ability to function as a pump. Sudden loss of cardiac output with subsequent tissue hypoperfusion creates global tissue ischemia; brain and myocardium are most susceptible. VF is the primary cause of sudden cardiac death (SCD).

**Frequency**

Incidence of arrhythmias is greater than 4,300,000 cases. VF has been described as the initial rhythm in almost 70% of out-of-hospital arrests. A circadian rhythm also has been reported.

Internationally: VF also is prevalent worldwide, with a reported predominance in the Northern Hemisphere. Among some European populations, annual incidence of cardiac arrests exceeds 6 per 10,000 people.

Cardiovascular diseases cause 12 million deaths in the world each year and are the primary causes of death in the United States. Although VF seldom is listed as the cause of death, it is thought to be responsible for more than 400,000 SCD cases in the United States annually. Untreated, VF causes loss of oxygen to vital organ systems and brain death in minutes.

SCD is more common among males than females, although the rates become similar for patients older than 70 years.

Incidence initially peaks during the first 6 months of life, then rapidly declines until a second peak in those aged 45-75 years.

**Causes**

VF and cardiovascular disease have common causes. VF is the major electrical mechanism for cardiac arrest and SCD. Causes of SCD and cardiac arrest include the following:

* Coronary heart disease (present in most patients who experience SCD)
* Myocardial hypertrophy
* Cardiac inflammatory diseases
* Cardiac valvular disease
* Electrophysiologic abnormalities
* Abnormal metabolic states
* Sympathetic nervous system disorders
* Proarrhythmic toxic exposures
* Electrocution
* Tension pneumothorax, trauma, and drowning
* Pulmonary embolism
* Hypoxia or acidosis

**Pathophysiology**

VF usually develops secondary to an area of myocardial ischemia with a lowered fibrillation threshold. Contributing factors include the following:

* Increased catecholamine levels
* Improper sympathetic stimulation
* Electrolyte imbalances
* Hypoxia or acid-base disturbances
* Toxic responses due to proarrhythmic drugs
* Hyperthermia/hypothermia
* Proarrhythmic conditions, such as prolonged QT syndromes

VF can be initiated in ischemic heart disease when a premature ventricular contraction (PVC) overcomes a lowered fibrillation threshold. Fibrillation is caused by simultaneous presence of abnormal irregular beats, conduction disturbances creating abnormal electrical propagation of the action potential, and reentry. An often-described R-on-T extrasystole phenomenon may account for many VF cases. Propagation to the whole myocardium implies that initial fibrillation reached sufficient critical mass.

In the absence of ischemic heart disease, VF may occur secondary to antiarrhythmic drugs, prolonged QT syndromes, preexcitation syndromes, and systemic hypoxemia. Ventricular tachycardia (VT) often precedes onset of VF in these cases; VT degenerates into VF.

Characteristics of ventricular waveforms in VF include an irregular heart rate, usually exceeding 300 beats/minute; an initial amplitude exceeding 0.2 mV; and a waveform that fades to a flat line within 15 minutes.

Primary VF is fibrillation in the absence of shock or cardiac failure (described by sudden ischemic events or proarrhythmic conditions).

Secondary VF is fibrillation occurring in the context of ongoing shock, myocardial infarction (MI), or congestive heart failure.

Ventricular flutter is a variant of VF in which the electrocardiogram appears as a sine wave. In contrast to the irregularity of VF, ventricular flutter may be difficult to distinguish from VT.

**CLINICAL**

**History**

VF often occurs without forewarning. The following symptoms, while not necessarily specific for SCD or VF, may develop before any major cardiac event:

Chest pain and other angina equivalents

Dyspnea

Easy fatigue

Palpitations

Syncope

Immediately preceding acute cardiac arrest, possible increase in heart rate, presence of PVCs, or period of VT

**Physical**

No pulse or respiration

Unconsciousness

Wide and chaotic QRS complexes on cardiac monitor

**Lab Studies**

* Electrocardiogram (ECG) to help identify ischemic or proarrhythmic conditions
* Serum electrolyte levels, including calcium and magnesium
* Cardiac enzymes to identify myocardial injury
* Complete blood count (CBC) to detect contributing anemia
* Arterial blood gases (ABGs) to assess degree of acidosis or hypoxemia
* Toxicologic screens and levels as clinically indicated

**Imaging Studies**

Chest x-ray may identify aspiration pneumonia, pulmonary edema, and injury (eg, secondary to cardiopulmonary resuscitation [CPR]).

**TREATMENT**

Prehospital Care

Because of the critical importance of early defibrillation, prehospital care is vital for arrests due to VF that occur outside the hospital. Interventions that impact survival and outcome of resuscitation include the following:

Early recognition of an arrest

Early activation of emergency medical services (EMS) system

Early access to trained personnel capable of performing CPR, defibrillation, and advanced cardiac life support (ACLS)

Automated external defibrillators (AEDs) have revolutionized prehospital VF management because they have eliminated the need for rhythm-recognition training. AEDs identify VF more rapidly than manual defibrillation techniques, are 92-100% specific for VF, and require less time to achieve defibrillation. Their use has resulted in increased survival rates and improved prognoses for patients in VF arrest.

AEDs are becoming increasingly available in workplaces, planes, remote areas, and police and first-response units; however, personnel still must be trained in rhythm recognition with manual defibrillators.

AEDs have a limited sensitivity (78-100%). One report describes a 50% sensitivity for fine VF and 90% for coarse VF.

Most AEDs deliver 3 consecutive jolts preset at 200 J, 200 J, and 360 J. No shock is delivered until the AED has finished analyzing the rhythm and confirmed VF. This sequence can be repeated if ACLS response has not arrived.

Do not use AEDs during transport by ambulance because patient movement creates the risk of inadvertent defibrillation.

Currently, AEDs cannot be used on children younger than 8 years.

Bystander CPR reportedly plays a significant role in prolonging the period (up to 12 min) in which VF may respond to a defibrillator. CPR may increase the number of patients in VF who benefit from defibrillation by response personnel (see Algorithm).

Emergency Department Care

Defibrillation

Electrical external defibrillation remains the most successful treatment of VF. A shock is delivered to the heart to uniformly and simultaneously depolarize a critical mass of the excitable myocardium. The objective is to interfere with all reentrant arrhythmia and to allow any intrinsic cardiac pacemakers to assume the role of primary pacemaker.

Successful defibrillation largely depends on the following 2 key factors: duration between onset of VF and defibrillation, and metabolic condition of the myocardium. VF waveform usually begins with a relatively high amplitude and frequency, and, then, it degenerates to smaller and smaller amplitude until asystole after approximately 15 minutes, possibly from depletion of the heart's energy reserves. Consequently, early defibrillation is vital; emergency response teams can perform defibrillation before arrival at the ED.

Defibrillation causes serum creatine phosphokinase to increase proportionate to the amount of electric energy delivered. If customary voltage is used to defibrillate a patient, the proportion of myocardial fraction (CK-MB) should remain within normal limits unless an infarction has caused myocardial injury.

* Precordial thump is less appropriate for VF than for VT because it is not as effective and may be detrimental. Use it only for witnessed monitored arrests where no defibrillator is immediately available.

**Algorithm**

Activate emergency response system

Initiate CPR

Verify patient is in VF as soon as possible (ie, AED and quick look with paddles)

Defibrillate (200 J adult; 2 J/kg child)

Defibrillate (200-300 J adult; 2-3 J/kg child)

Defibrillate (maximum 360 J adult; 4 J/kg child)

Check for pulses/rhythm

CPR for 1 minute, with attention to the following:

Properly positioning electrode

Attempting tracheal intubation and IV access

Giving epinephrine every 3 minutes

Correct the following if necessary and/or possible:

Hypoxia

Hypovolemia

Hyperkalemia/hypokalemia and metabolic disorders

Tension pneumothorax

Tamponade

Toxic/therapeutic substances

Thromboembolic/mechanical obstruction

Defibrillate (360 J adult; 4 J/kg child)

Lidocaine (1 mg/kg IV) or amiodarone (300 mg IV)

Defibrillate (360 J adult; 4 J/kg child)

Lidocaine (1 mg/kg IV), if amiodarone used in the first round

Other options are as follows: administer amiodarone (300 mg IV) now, if not used in the first round; repeat lidocaine; or use bretylium (5 mg/kg IV)

Amiodarone IV bolus is administered only once.

Defibrillate (360 J adult; 4 J/kg child)

Amiodarone (300 mg IV), if not used in the first or second round, or bretylium (5 mg/kg IV)

Following the third shock, intervals between consecutive jolts should not exceed 1 minute.

Assess use of the following in conjunction with subsequent defibrillation attempts; administer a shock 30 seconds after each dose of indicated drug:

Lidocaine (1-1.5 mg/kg IV; maximum 3 mg/kg total)

Amiodarone (0.5 mg/min following bolus dosing above, or IV loading protocol below)

Bretylium (5 mg/kg IV; may repeat at 10 mg/kg)

Epinephrine (0.1-0.2 mg/kg IV): for adults, 1 mg IV every 3 minutes usually is used; alternative dosing regimens are advocated (ie, high, intermediate, escalating)

Procainamide (30 mg/min IV, maximum 17 mg/kg total in refractory VF)

Magnesium sulfate (1-2 g IV push) in cases of suspected hypomagnesemia or refractory VF

Sodium bicarbonate (1 mEq/kg IV push) in cases of known preexistent hyperkalemia or known tricyclic antidepressant overdose

Three initial defibrillation attempts take precedence over CPR as soon as a defibrillator or AED is available.

Lidocaine and epinephrine can be administered through the endotracheal (ET) tube if IV attempts fail. Use 2.5 times the IV dose.

Refractory VF

Lack of response to standard defibrillation algorithms is challenging. Addition of magnesium and/or procainamide often is ineffective.

If not used earlier, consider amiodarone loading protocol, as follows: 15 mg/min for 10 minutes, followed by 1 mg/min for 6 hours, then 0.5 mg/min for 18 hours.

Such reported alternatives as transesophageal and intracardiac defibrillation or thoracotomy with internal defibrillation generally are impractical because of limited experience and availability of equipment and trained personnel.

**Postresuscitative care**

Antiarrhythmics used successfully should be continued. Maintain lidocaine at 1-4 mg/min, bretylium at 1-2 mg/min, and amiodarone at 0.5 mg/min.

Control any hemodynamic instability.

Administer vasopressors as indicated.

Postdefibrillation arrhythmias (mainly atrioventricular [AV] blocks) have been reported in up to 24% of patients. The incidence is related to the amount of energy used for defibrillation.

Check for complications (eg, aspiration pneumonia, CPR-related injuries).

**WOLFF-PARKINSON-WHITE SYNDROME**

Synonyms: preexcitation syndrome, WPW syndrome

Preexcitation was defined by Durrer et al in 1970 when he wrote, "Preexcitation exists, if in relation to atrial events, the whole or some part of the ventricular muscle is activated earlier by the impulse originating from the atrium than would be expected if the impulse reached the ventricles by way of the normal specific conduction system only." Of the various preexcitation syndromes, the most common is Wolff-Parkinson-White (WPW) syndrome. Emergency physicians should be familiar with this syndrome and the proper treatment of its associated arrhythmias to avoid unnecessary morbidity and mortality.

**Frequency**

* WPW affects approximately 0.15-0.2% of the general population. Of these individuals, 60-70% have no other evidence of heart disease.
* Death from WPW occurs secondary to the associated arrhythmias or from mistreatment of these arrhythmias with inappropriate medications. Little data are available regarding the mortality rate of such arrhythmias, but most studies report the incidence of sudden death in the 0-4% range.
* Men are affected more often (60-70%) than women. Typically, those affected are young, otherwise healthy individuals.
* Although this disease affects people of all ages, it most commonly is recognized in children and young adults presenting to the ED with an arrhythmia. Conduction speed in the accessory pathway appears to attenuate with age.

**Pathophysiology**

Accessory connections between the atrium and ventricle are the result of anomalous embryonic development of myocardial tissue bridging the fibrous tissues that separate the 2 chambers. Although dozens of locations for bypass tracts can exist in preexcitation, including atriofascicular, fasciculoventricular, intranodal, or nodoventricular, the most common bypass tract is an accessory atrioventricular (AV) pathway otherwise known as a Kent bundle. This is the anomaly seen in WPW syndrome. Conduction through a Kent bundle can be anterograde, retrograde, or both.

**History**

Patients may present with anything from mild chest discomfort or palpitations to severe cardiopulmonary distress or arrest. Occasionally, the disease is discovered on routine electrocardiography (ECG) performed for a reason other than acute cardiac symptomatology.

Patients commonly present with heart rates in the 250 beat per minute (bpm) range, often with associated hypotension.

Patients usually are aware of their cardiac condition, but they may be diagnosed in the ED.

**Physical**

WPW has no specific examination features except for those that may accompany the arrhythmias.

On physical examination, the patient may be cool, diaphoretic, and hypotensive.

Crackles in the lungs are common, as the high heart rate causes diastolic heart failure.

**Tests**

**Electrocardiogram**

The classic ECG morphology of WPW is described as a shortened PR interval, a widened QRS complex, and a delta wave. In reality, however, morphology varies greatly.

Depending upon the location of the accessory pathway in relation to the sinus node and the relative transmission characteristics of the accessory pathway and the AV node, the morphology of the ECG may vary from a classic presentation to near normal.

In some cases, the electrical impulse's arrival into the ventricle occurs slightly earlier through the accessory pathway, creating preexcitation.

QRS is widened because the ventricles initially are activated via the accessory pathway outside the normal conducting system in the muscle tissues, producing slow initial forces and a delta wave. This is known as a revealed accessory pathway because it is easily identifiable on ECG.

In other cases, however, arrival of the electrical impulse to the ventricle occurs nearly simultaneously through both the accessory pathway and the AV node.

When this occurs, preexcitation is absent and ECG appears normal.

Thus, morphology of the ECG depends directly upon the degree of preexcitation.

An accessory pathway that does not manifest on ECG is revealed when the rate exceeds the refractory period of the AV node. This has been described as latent.

A latent accessory pathway can conduct both anterograde and retrograde transmissions.

An accessory pathway in which only retrograde transmission of impulses can occur is called concealed and is used only during circus movement tachycardias (CMTs).

Although any type of arrhythmia can occur in a patient with WPW, the two most common are CMTs and atrial fibrillation (AFib). CMT is the more common arrhythmia of the two.

A critically timed premature atrial beat that occurs during the refractory period of the accessory pathway typically initiates CMT. The impulse, therefore, travels solely down the AV node but returns through the accessory pathway, resulting in CMT.

This CMT, termed orthodromic, shows a narrow complex rate limited by the refractory period of the AV node. The complex rate is narrow because the impulses travel only antegrade through the AV node and regular because circus movement occurs at a regular rate.

Differential diagnosis of this arrhythmia includes paroxysmal supraventricular tachycardia (PSVT), but differentiating the two in an emergent presentation is unnecessary because treatment for both is identical. Adenosine (6 mg rapid IV push; if unsuccessful, follow by 12 mg rapid IV push) temporarily blocks the AV node and interrupts CMT, usually converting the patient to sinus rhythm. Note that an orthodromic CMT is the only common WPW arrhythmia in a patient with a concealed accessory pathway.

Orthodromic CMTs are 10-15 times more likely than antidromic CMTs.

Antidromic CMTs are wide and potentially may be faster because of the relatively short refractory period of most accessory pathways. They are wide because anterograde transmission occurs down the accessory pathway, creating preexcitation of the ventricle adjacent to it, yet they are regular because of the regular nature of the circus movement.

Differential diagnoses include ventricular tachycardia (V tach), which also is regular (unless it is torsade de pointes) or PSVT with aberrancy. Consider any regular wide-complex tachycardia to be V tach until proven otherwise; however, as in regular narrow-complex tachycardias, a stable patient initially may be treated with adenosine.

Most cases of wide-complex CMT associated with WPW that are treated with adenosine consequently are converted to sinus rhythm.

AFib in patients with WPW is very common and has an incidence of 11-38%. It also is the deadliest arrhythmia for these patients because of the possibility of deterioration into ventricular fibrillation (V fib).

In normal hearts, an individual is protected from exceptionally high rates by the relatively long refractory period of the AV node. In patients with WPW, however, the accessory pathway often has a much shorter anterograde refractory period, allowing for much faster transmission of impulses and correspondingly higher rates.

In addition, sympathetic discharge secondary to hypotension may lead to further shortening of the refractory period and subsequent increase in the ventricular rate. If the rate becomes too high, V fib may result.

Note that AFib through an accessory pathway appears as a bizarre, wide-complex, irregular tachycardia on ECG, with rates often in the 250 bpm range or higher.

**TREATMENT**

* Prehospital Care: Therapy in the prehospital setting depends upon the patient's degree of stability and the specific arrhythmia.
* Emergency Department Care: Upon presentation, immediately place an IV line in the patient and connect him or her to cardiac, blood pressure, and pulse oximetry monitors. Administer oxygen if the patient is hypoxic. Immediately perform cardioversion on a patient who is grossly unstable. If a patient is in cardiac arrest, treat according to advanced cardiac life support (ACLS) guidelines. Treat arrhythmias associated with WPW with caution.

Treatment of AFib associated with WPW is necessarily different than for a patient with a normal heart. AFib is an irregular rhythm as opposed to the regular rhythm seen in CMTs.

The basic treatment principle in WPW AFib is to prolong the anterograde refractory period of the accessory pathway relative to the AV node. This slows the rate of impulse transmission through the accessory pathway and, thus, the ventricular rate.

If AFib were treated in the conventional manner by drugs that prolong the refractory period of the AV node (eg, calcium channel blockers, beta-blockers, digoxin), the rate of transmission through the accessory pathway likely would increase, with a corresponding increase in ventricular rate. This could have disastrous consequences, possibly causing the arrhythmia to deteriorate into V fib.

Procainamide (17 mg/kg IV infusion, not to exceed 50 mg/min; hold for hypotension or 50% QRS widening) blocks the accessory pathway, but it has the added effect of increasing transmission through the AV node. Thus, although procainamide may control the AFib rate through the accessory pathway, it may create a potentially dangerous conventional AFib that may require treatment with other medications. Prompt cardioversion of patients with WPW and AFib is recommended.

Medical management may be a viable option in some patients, but it may have unpredictable results. Note that cardioversion is always the treatment of choice in unstable patients.

Treatment of CMTs associated with WPW is similar to treating PSVT. Focus on breaking the cyclical transmission of impulses. This is best accomplished by temporarily prolonging the refractory period of the AV node with drugs such as adenosine.

In a stable patient, adenosine (6 mg rapid IV push; if unsuccessful, 12 mg rapid IV push) should be the first-line treatment in any regular tachycardia, regardless of whether the complex is wide or narrow.

Once the circus movement is broken, the patient usually converts to sinus rhythm. Note that whether the QRS complex is regular or irregular distinguishes CMTs from AFib on ECG.

If the QRS complex is regular, the arrhythmia can be treated safely with adenosine as if it were CMT or PSVT.

If the QRS complex is irregular, the arrhythmia is likely AFib. In this case, adenosine theoretically could increase the rate.

Cardioversion, or in some cases, procainamide, are the treatment choices in these situations (ie, irregular QRS complex), providing the necessary rate control.

If in doubt about the regularity of the rhythm, it is safer to err on the side of treating for AFib.

**Further Inpatient Care**

Arrange consultation with a cardiologist for patients admitted with WPW because these patients are at high risk of sudden death if they develop AFib. Consider ablation of the accessory pathway in all patients.

**Further Outpatient Care**

Education is the most important aspect of outpatient care for patients with WPW. Urge patients to carry a sample ECG in sinus rhythm and a medical identification bracelet in case of cardiac arrest.

**Prognosis**

Patients with WPW have an excellent prognosis when treated with ablation of the accessory pathway.

**LOWN-GANONG-LEVINE SYNDROME**

The Lown-Ganong-Levine syndrome (LGL) is usually considered in a class of preexcitation syndromes that includes the Wolff-Parkinson-White syndrome (WPW), LGL, and Mahaim-type preexcitation. Investigations into WPW have revealed that an accessory pathway for conduction, called a bundle of Kent, from the atria to the ventricles underlies the preexcitation observed in patients with WPW. Less is known regarding the structural anomalies underlying LGL. Theories proposed to explain LGL have centered around the possible existence of intranodal or paranodal fibers that bypass all or part of the atrioventricular (AV) node.

In 1938, Clerc et al first described the occurrence of frequent paroxysms of tachycardia in patients with a short PR interval and normal QRS duration. This syndrome was again described in 1952 by Lown, Ganong, and Levine, whose names form the eponym now used to describe it. In 1946, Burch and Kimball proposed that an atrio-His (AH) bundle pathway might explain the findings of the syndrome, although no such pathway had yet been identified anatomically. In 1961, James described fibers that originate in the low atrium and terminate low in the AV node. Brechenmacher et al reported anatomic findings of an AH bundle in 1974. Subsequent investigations into the origin of LGL have largely involved invasive electrophysiologic studies that have sought to identify structural and functional anomalies that might explain the findings of LGL.

Criteria for LGL include PR interval less than or equal to 0.12 second (120 ms), normal QRS complex duration, and occurrence of supraventricular tachycardia but not atrial fibrillation or atrial flutter.

Historically, some authors have referred to patients with a short PR interval and normal QRS duration as having LGL. However, this practice has been largely abandoned as more evidence has accumulated demonstrating that such patients without a history of tachycardia likely fall into a class of normal variants. Patients with an isolated finding of short PR interval may be characterized as having accelerated atrioventricular nodal conduction.

The term enhanced atrioventricular nodal conduction (EAVNC) refers to a set of functional criteria which includes an AH interval less than or equal to 60 ms, 1-to-1 AV nodal conduction at rates as high as 200 beats per minute, and an abnormally small increase in AH interval as atrial pacing rate is increased.

EAVNC represents a functional characterization of the AV node, whereas LGL refers to a syndrome of supraventricular tachycardia in association with a short PR interval. The short PR interval in LGL may be related to the presence of EAVNC. LGL and EAVNC may coexist, or either may exist alone in a given patient.

**Frequency**

Lown and associates described tachyarrhythmias in 17% of patients with a short PR interval. Some 2-4% of the adult population has a PR interval less than or equal to 0.12 second. Taken together, these data provide an estimate of the frequency of LGL as 0.5% of the adult population.

Frequency mirrors that in the United States.

Paroxysms of tachycardia represent the primary morbidity of LGL. Few data are available regarding the frequency of these paroxysms. Data regarding mortality from LGL are scant. Numbers in published studies are too small to estimate mortality rate with significant accuracy or confidence. In the absence of significant structural heart disease, the mortality rate appears to be very low.

**Causes**

No environmental factors that contribute to occurrence of LGL have been identified. Some evidence suggests that both WPW and LGL may be hereditary in certain families.

**Pathophysiology**

No single structural anomaly has been implicated directly as the cause of LGL. Indeed, most authors believe that LGL does not exist as a phenomenon separate from other known conditions. Several structural anomalies have been proposed as the possible basis for LGL, including the presence of James fibers, Mahaim fibers, Brechenmacher-type fibers, and an anatomically underdeveloped or small AV node. James fibers run from the upper portion of the AV node and insert in the lower portion or in the bundle of His. Mahaim fibers may originate in the lower portion of the AV node, the bundle of His, or the bundle branches, and they terminate in the interventricular septum or in a bundle branch. Each of these fibers has been identified histologically. However, none of these anomalous communications has been linked causally to the presence of LGL. The histologic presence of fibers does not speak to whether these fibers are functional, with conductive properties.

EAVNC has been investigated as a possible functional basis for LGL. The criteria for EAVNC were established arbitrarily on the basis of observations of some patients with what seemed to be abnormally rapid AV nodal conduction times. In 1983, however, Jackman et al provided convincing evidence that EAVNC does not exist as a phenomenon separate from normal AV nodal physiology, but that AV nodal conduction physiology comprises a spectrum of AH intervals. In their series of 160 consecutive patients, they failed to identify a distinct group of patients with abnormally rapid AV nodal conduction. Rather, they found a broad spectrum of AH intervals in a unimodal, continuous distribution.

The modern view of LGL is that no convincing evidence suggests that this is a syndrome separate from other known phenomena. LGL was identified as a syndrome prior to the advent of catheter-based electrophysiologic (EP) studies. EP studies have led to several realizations. The short PR interval of LGL likely represents one end of the spectrum of normal PR intervals. Most patients with putative LGL are found at EP study to have another basis for paroxysmal tachycardia. Most have AV nodal reentrant tachycardia. Others have concealed accessory pathways, usually near the septum.

Thus, unless further studies demonstrate definitive structural or functional anomalies, the diagnosis of LGL remains a clinical diagnosis of the era before EP study.

**CLINICAL**

**History**

Symptoms of paroxysmal tachycardia may be elicited. The manifestations of these paroxysms include palpitations, lightheadedness, and shortness of breath. In cases of underlying structural heart disease or coronary artery disease, episodes of tachycardia may induce cardiac stress and produce symptoms of chest pain or possibly of hypotension or other hemodynamic instability.

**Physical**

Findings are normal except during tachycardic episodes; cardiovascular examination may then reveal a rapid heart rate. However, absence of this finding does not exclude LGL as a possible diagnosis, as the tachycardia of LGL is paroxysmal.

**Lab Studies**

Workup is directed at determining the cause of tachycardia. LGL is an outdated diagnosis, and as such no workup is directed at making this diagnosis. However, identification of a short PR interval during sinus rhythm in a patient with paroxysmal supraventricular tachycardia (PSVT) should raise suspicion of a possible underlying bypass tract (ie, WPW). In the case of isolated short PR interval with no history of tachycardia or symptoms suggestive of paroxysms of tachycardia, no further workup is indicated.

Patients may present in an acute episode of tachycardia or with a history of symptoms suggestive of paroxysms of tachycardia.

In the acute setting, institute a standard workup for tachycardia, including an ECG to document the rhythm, serum electrolytes, calcium, and magnesium levels.

For a history suggestive of recurrent paroxysms of tachycardia, a Holter monitor or event recorder may prove useful for documenting the rhythm during acute symptomatic episodes. In rare instances, an implantable rhythm monitor may prove helpful.

**Imaging Studies**

To meet criteria for LGL, the 12-lead ECG must demonstrate a PR interval less than 0.12 second and a normal QRS upstroke and duration. If a paroxysm of supraventricular tachycardia, not atrial fibrillation or atrial flutter, is recorded on ECG, the third criterion for LGL is met.

A delta wave on the QRS complex precludes the diagnosis of LGL, because one of the criteria for LGL is a normal QRS complex. A delta wave indicates WPW, another preexcitation syndrome.

In the case of shortness of breath, posteroanterior and lateral chest films are indicated.

**Procedures**

If tachycardia is present, diagnostic workup to determine the cause may include Valsalva maneuvers. If blood pressure is stable, the patient has no angina and is not presyncopal, and no carotid bruits are present, carotid massage may be instituted to break the rhythm and provide a ventricular pause long enough to reveal the underlying atrial rhythm. If these maneuvers fail to break the tachycardic rhythm, a trial of adenosine administration with simultaneous rhythm strip recording may reveal the rhythm.

**TREATMENT**

Medical Care

Because LGL is an outdated diagnosis, no specific therapy is indicated. In the acute setting of tachycardia, the goals of medical care include identifying the cause of tachycardia and, in symptomatic cases, controlling the ventricular rate. Treatment should be based on the cause of tachycardia. As with any tachycardia, hospitalization is warranted in cases of hemodynamic instability.

In the outpatient setting, empiric therapies for recurrent PSVT may be instituted. These therapies may include beta-blockers, calcium channel blockers, and digoxin.

Surgical Care

Rare patients for whom the criteria of LGL are met may have no inducibility of tachyarrhythmias by EP study. Rarely, medical therapy fails in these patients, who continue to have recurrent, intolerable symptoms. In such extreme cases, radiofrequency (RF) ablation of the AV node or bundle of His may be considered, followed by implantation of a pacemaker.

**SINUS NODE DYSFUNCTION**

The term sick sinus syndrome first appeared in the literature in 1967 to describe a rapid atrial rhythm interspersed with varying periods of bradycardia that followed cardioversion. Today, the term is applied to a broad range of electrophysiological abnormalities, including inappropriate sinus bradycardia, sinus arrest, sinus node exit block, chronic atrial fibrillation, and bradycardia-tachycardia syndrome. Although the term remains popular, the more comprehensive title sinus nodal dysfunction (SND) is gaining favor.

The disease commonly is observed in older patients with a history of concomitant heart disease. Its natural history is largely unknown, but it frequently runs an erratic and progressively malignant course. For this reason, cardiac pacing has become the cornerstone of therapy for symptomatic patients.

**Frequency**

In the US: The exact incidence rate of SND is unknown because it never has been evaluated adequately in asymptomatic patients. One study estimates the incidence to be about 3 cases in 5000 patients older than 50 years.

Internationally: SND is more prevalent in countries where citizens have a longer life expectancy, indicating that the disease is more common in elderly people.

Duration from the onset of symptoms to death is not well defined; untreated, a patient may live a couple of weeks to more than 10 years. SND almost always is progressive, and patients usually become increasingly symptomatic if left untreated. Survival appears to depend primarily on the severity of the underlying cardiac disease. Thromboembolic complications are a frequent cause of morbidity and mortality. Chronic atrial fibrillation and tachycardia-bradycardia syndrome are the variants with the most significant stroke risk. Sudden cardiac death is possible at any point during the disease course.

SND tends to be a disease of elderly people, with a peak incidence in the sixth and seventh decades of life.

The disease may be observed in any age group, including adolescents and children.

Pediatric patients are most susceptible during the postoperative period of major cardiac surgery, especially transposition of the great vessels.

**Causes**

Although the exact etiology usually is not identified, most cases are believed to be attributable to a combination of intrinsic and extrinsic influences. The most common intrinsic causes are idiopathic degenerative disease and coronary artery disease. The most common extrinsic causes are medications and autonomic hyperactivity. Remember that SND is always an acquired condition.

Intrinsic sinus nodal dysfunction

Idiopathic degenerative disease is the most common intrinsic cause. With aging, the myocardium surrounding the sinus node gradually becomes replaced by fibrous stroma. As this fibrosis progresses, automaticity and sinoatrial conduction can become impaired. In a patient with SND, fibrosis of the sinus node is virtually complete. Why certain patients with diffuse fibrosis develop SND while others do not remains unclear.

Coronary artery disease may cause SND either by chronic hypoperfusion or as a complication of an acute ischemic event. An acute inferior or lateral myocardial infarction sometimes is complicated by significant bradycardia or sinus arrest; these arrhythmias are due to local neural effects and usually are transient. Temporary SND has been reported in 5-10% of acute myocardial infarctions.

The following also may cause intrinsic SND:

* Infiltrative diseases (amyloid, hemochromatosis, neoplasms)
* Cardiomyopathy
* Hypertension
* Collagen vascular diseases (systemic lupus erythematosus [SLE], scleroderma)
* Congenital heart disease
* Surgical trauma/heart transplant
* Musculoskeletal disorders (myotonic dystrophy, Friedreich ataxia)
* Myocarditis/pericarditis
* Extrinsic sinus nodal dysfunction
* Medications that depress sinus nodal function often are implicated as the cause of SND. Common offending agents include the following:

Beta-blockers

Nondihydropyridine calcium channel blockers (diltiazem, verapamil)

Cardiac glycosides (digoxin)

Sympatholytic antihypertensives (clonidine, methyldopa, and reserpine)

Membrane-active antiarrhythmics (amiodarone, sotalol, bretylium)

Less commonly, phenytoin, amitriptyline, lithium, and phenothiazine

Autonomic dysfunction may be caused by vagal stimulation slowing the sinus rate and lengthening the refractory period of the sinus node. Conditions associated with marked hypervagotonia can result in SND. Symptomatic bradycardias secondary to excess vagal tone have been observed in well-trained athletes. Vasovagal syncope, specifically in elderly people, can present with SND. Finally, carotid sinus syndrome has been associated with increased vagal tone that infrequently leads to symptomatic bradyarrhythmias.

The following also can cause extrinsic SND: electrolyte imbalance (eg, hypokalemia or hypocarbia), hypothyroidism or hyperthyroidism, hypothermia, and sepsis.

**Pathophysiology**

SND is characterized by delayed or failed conduction between the sinus node and the atria, either due to inadequate sinus node pacemaking or because of intrinsic or extrinsic conduction disturbance. If the degree of dysfunction is minimal, the patient usually is asymptomatic. Advanced SND is unpredictably malignant, however, and it often is characterized by significant cardiovascular impairment. Clinical manifestations are due to hypoperfusion of the vital organs, specifically the brain and heart, usually as a result of an inadequate ventricular rate. Patients commonly present with complaints related to cerebral or cardiac dysfunction.

**CLINICAL**

**History**

Because SND is a heterogenous condition, its clinical manifestations may vary widely. In the early stages of SND, most patients are asymptomatic. As the disease advances, however, patients often seek medical attention for bradycardia-related symptoms. Syncope, near-syncope, and dizziness are the most frequently reported complaints, followed by palpitations, angina, or shortness of breath. Because these symptoms are relatively nonspecific, a high index of suspicion is necessary to make the correct diagnosis.

* Cerebral symptoms

Patients with mild symptoms present with vague complaints of fatigue, irritability, labile mood swings, or forgetfulness.

As the disease progresses and blood flow is further compromised, the cerebral manifestations become more profound. They can include lightheadedness, slurred speech, near-syncope, and, finally, syncope. Syncope almost always implies marked bradycardia. It is believed to occur in most patients with SND.

* Cardiac symptoms

Early in the course of the disease, patients may note an abnormally slow or irregular pulse.

As the disease progresses, the 3 most common cardiac manifestations are palpitations, angina, and shortness of breath, which is due to congestive heart failure (CHF).

Palpitations may be attributed to runs of supraventricular tachycardia (SVT) or to a mixture of tachycardia and bradycardia.

Angina and CHF symptoms usually are due to cardiac hypoperfusion. Cases of flash pulmonary edema have been reported.

In late stages, the incidence of ventricular tachycardia or fibrillation increases, thereby augmenting the risk for sudden cardiac death.

* Other symptoms

Other symptoms include hypoperfusion of the kidney with resultant oliguria.

Some patients have a history of vague gastrointestinal complaints, probably also related to inadequate oxygenation.

**Physical**

Certain physical findings are suggestive, but the results of the physical examination alone never are diagnostic. The most persistent finding is long periods of bradycardia, appearing in as many as 75% of patients. Any person with unexplained, marked bradycardia probably requires further workup. Occasionally, arrhythmia may be detected with careful assessment of the pulse. Several basic maneuvers can help establish the diagnosis.

Valsalva maneuver

In a healthy individual, the Valsalva maneuver will increase heart rate.

A patient with SND, however, usually will not respond appropriately (heart rate will not change).

Carotid sinus massage: Carotid sinus massage may be helpful. If carotid massage provokes a sinus pause of longer than 3 seconds, SND must be considered carefully.

**Lab Studies**

Laboratory blood tests rarely are helpful in the workup of SND.

Because hypokalemia, hyponatremia, and hypocarbia sometimes can aggravate underlying susceptibility for SND, a renal panel should be ordered routinely.

Thyroid dysfunction also has been reported to cause SND, so ordering a thyroid screening test should be considered in appropriate patients.

**Imaging Studies**

* Electrocardiogram

The best diagnostic test for SND is electrocardiographic documentation during a symptomatic episode. Therefore, a 12-lead ECG should be obtained in any patient presenting with symptoms suggestive for SND. The ECG rarely is helpful in milder cases, but it can be diagnostic in more severe disease. SND may manifest as any of the following:

Inappropriate sinus bradycardia: Refractory sinus bradycardia, defined as bradycardia that does not increase with exertion, usually is the earliest manifestation of SND. **Therefore, the diagnosis should be considered strongly in any patient with unexplained chronic bradycardia.**

Sinus arrest/sinus pause: A sinus arrest will occur whenever the sinus node becomes so diseased that it ceases to fire impulses appropriately. A pause greater than 3 seconds is strongly suggestive for SND. A well-trained athlete occasionally can produce a pause greater than 2 seconds, but a 3-second pause is very unusual in a healthy individual.

Sinoatrial exit block: Sinoatrial exit block occurs whenever a conduction barrier within the sinoatrial node blocks exiting impulses directed to the atria. Because the atria are not depolarized, the asystolic pause will not be terminated by a P wave, but instead by a sinus beat. A way to distinguish sinoatrial block from sinus arrest is to examine the period of asystole. The pause that occurs in sinoatrial block typically is an exact multiple of the preceding P-P interval, whereas the pause observed in sinus arrest usually is not.

Chronic atrial fibrillation: Chronic atrial fibrillation with slow ventricular response often implies SND. Attempts at cardioversion typically produce a long sinus pause, followed by a slow unstable rhythm.

Tachycardia-bradycardia syndrome: This syndrome affects about 50% of the patients with SND. The term refers to alternating periods of sinus bradycardia and SVT. A long pause often follows spontaneous termination of the tachycardia. During this pause, patients frequently experience lightheadedness or syncope.

Holter monitoring: A 24- to 48-hour Holter monitor may be useful in patients who report frequent symptoms. Most often, its true benefit is to exclude SND as the cause of symptoms. If only normal sinus rhythm is documented, SND is unlikely to be the diagnosis.

Event recorder: A loop monitor is a helpful tool for those patients with infrequent complaints. It can be triggered to record an ECG with the onset of symptoms. This test requires a certain degree of patient compliance; they must tolerate carrying the device, and they must remember to turn on the recorder at the appropriate moment.

* Electrophysiological study

Although electrophysiological studies are performed frequently on patients with suspected SND, their usefulness remains controversial.

Most commonly, the test is used to measure sinus nodal recovery time via overdrive suppression of the sinus node. Patients are diagnosed with SND if they have a longer sinus node recovery time than healthy individuals. The primary criticism of the test is that the laboratory method usually employed yields an indirect measurement of sinus nodal recovery time. Even under the best circumstances, the sensitivity of this test for SND is only about 70%.

The second commonly used electrophysiological test measures sinoatrial conduction time using premature atrial stimuli. Once again, the results often are not conclusive because the measurements are obtained indirectly. Because of these limitations, most experts agree that an invasive electrophysiological study should be reserved only for those patients who have a history consistent with SND but who have failed to have the disease documented via noninvasive means.

Exercise testing

Exercise testing has limited value in the diagnosis of SND. It can be helpful in differentiating patients with resting sinus bradycardia with normal exercise response from those patients with chronotropic incompetence.

* An echocardiogram should be considered because it can document the presence of underlying valvular or ischemic heart disease.

**TREATMENT**

* Medical Care

Once the diagnosis of SND has been established, the treatment plan should be individualized to address the specific arrhythmias and symptoms of each patient.

Asymptomatic SND requires no treatment. Individuals with asymptomatic bradycardia or sinus pauses simply can be observed.

Medications that depress the sinus node should be discontinued whenever possible in patients with symptomatic bradycardia.

Patients with chronic atrial fibrillation have a significant risk for thromboembolic complications. They should undergo prophylactic anticoagulation with warfarin. If strong contraindications exist for the use of warfarin or if a patient has lone atrial fibrillation, then an aspirin a day will suffice.

Tachycardia-bradycardia syndrome usually needs a treatment plan for both components. The tachycardia typically is treated with medications that control ventricular rate. Unfortunately, these drugs may exacerbate bradycardia by further depressing sinus nodal function. In these instances, pacemaker insertion is indicated to prevent the recurrence of symptoms related to bradycardia or sinus arrest.

* Surgical Care

Pacemaker placement is the cornerstone of treatment for symptomatic SND. Various studies estimate that 25-50% of all pacemaker implantations in Western countries are performed for some form of SND.

Most often, pacing is recommended only for those patients who experience symptomatic bradycardia. According to recent American College of Cardiology guidelines, a pacemaker is not indicated for individuals with asymptomatic bradycardia or sinus pauses.

Mounting evidence suggests that atrial-based pacing is the preferred pacing mode for SND in the absence of atrioventricular conduction disease. Although dual-chamber and ventricular-based pacing are used more commonly, several recent retrospective studies suggest that single-chamber atrial pacing incurs a lower risk of atrial fibrillation, thromboembolic events, CHF, and mortality. In one trial, these benefits were maintained over an 8-year follow-up period. A frequent criticism of atrial pacing has been the possibility that a pacemaker revision might become necessary if atrioventricular block develops later in the course of the disease. Any such risk is minimal, however, and does not warrant routine dual-chamber pacemaker placement, especially when cost is factored into the equation.

Ventricular pacing remains an appropriate choice for chronic atrial fibrillation with slow ventricular response.

**ATRIOVENTRICULAR BLOCK**

Atrioventricular (AV) block occurs when atrial impulses fail to reach the ventricles or when atrial impulses are conducted with a delay. Three degrees of AV block are recognized.

First-degree AV block consists of prolongation of the PR interval on the electrocardiogram (ECG) (>0.20 s in adults and >0.16 s in young children). The upper limit of the reference range for the PR interval is age-dependent in children. All atrial impulses reach the ventricles in first-degree AV block; however, conduction is delayed within the AV node.

Second-degree AV block is characterized by atrial impulses failing to conduct to the ventricles in either of 2 different ways.

Mobitz I second-degree AV block (Wenckebach block) consists of progressive prolongation of the PR interval with the subsequent occurrence of a nonconducted P wave that creates a pause. The pause is shorter than the sum of any 2 consecutive conducted beats (R-R interval). An episode of Mobitz I AV block usually consists of 3-5 beats, with a ratio of nonconducted to conducted beats of 4:3, 3:2, and so on.

Mobitz II second-degree AV block is characterized by a constant PR interval followed by sudden failure of a P wave to be conducted to ventricles in a systematic fashion, eg, either an occasional dropped P wave or a regular conduction pattern of 2:1, 3:1, and so on.

Third-degree AV block is diagnosed when no supraventricular impulses are conducted to the ventricles. P waves on ECG reflect an independent sinus node rhythm from QRS electrocardiographic wave complexes that represents an escape rhythm, either junctional or ventricular. The escape rhythm originating from the junctional or high septal region is characterized by narrow QRS complexes at a rate of 40-50 beats per minute, whereas escape rhythm from low ventricular sites is characterized by broad QRS complexes at a rate of 30-40 beats per minute. No relationship exists between the rhythm of P waves and the rhythm of QRS complexes. The frequency of P waves (atrial rate) is higher than the frequency of QRS complexes (ventricular rate).

**Frequency**

First-degree AV block in the US can be found in healthy adults, and incidence increases with age. At age 20 years, the PR interval may exceed 0.20 seconds in 0.5-2% of healthy people. At age 60 years, more than 5% of healthy individuals have PR intervals exceeding 0.20 seconds.

Type II second-degree AV block (Mobitz II) is very rare in healthy individuals, whereas type I second-degree AV block (Wenckebach) is observed in 1-2% of healthy young people, especially during sleep.

Congenital third-degree AV block is very rare—1 case per 20,000 births.

AV blocks occur more frequently in older people, especially in people who have organic heart disease. Approximately 5% of patients with heart disease have first-degree AV block, and about 2% have second-degree AV block. The incidence of third-degree AV block peaks after age 70 years (approximately 5-10%).

The international incidence is the same as incidence in the United States.

Progressive degrees of AV block carry increasing morbidity and potential related mortality.

Third-degree AV block and subsequent asystole has been estimated to cause 20% of cases of sudden cardiac death. This high contribution is increasing with improved survival of patients with advanced congestive heart failure. In most cases of sudden death due to complete AV block, the conduction block appears without warning. In people diagnosed with third-degree AV block and treated with permanent pacemakers, the subsequent mortality rate is very low. Mobitz I AV blocks are not associated with increased mortality rates. Second-degree Mobitz II block may progress to complete AV block.

AV blocks generally are not associated with major morbidity. However, the low heart rate observed in third-degree or Mobitz II AV block may lead to syncopal episodes with major related injuries (eg, head trauma, hip fracture), exacerbation of congestive heart failure, or exacerbation of ischemic heart disease symptoms due to low cardiac output.

A 60% female preponderance exists in congenital third-degree AV block.

For acquired third-degree AV block, a 60% male preponderance exists.

The incidence of AV block increases with age. The incidence of third-degree AV block is highest in people older than 70 years (approximately 5-10% of patients with heart disease).

**Causes**

Delay or lack of conduction through the AV node has multiple causes.

First- and second-degree Mobitz I (Wenckebach) AV blocks may occur in healthy, well-conditioned people as a physiologic manifestation of high vagal tone. Mobitz I (Wenckebach) block also may occur physiologically at high heart rates (especially with pacing) due to increased refractoriness of the AV node, which protects against conducting a fast rhythm to the ventricles.

AV block may be caused by acute myocardial ischemia or infarction. Inferior myocardial infarction may lead to third-degree block, usually at the AV node level. Anterior myocardial infarction usually is associated with third-degree block due to ischemia or infarction of bundle branches.

Degenerative change in the AV node or bundle branches (eg, fibrosis, calcification, infiltration) is the most common cause of nonischemic AV block. Lev disease, usually occurring in older people, consists of fibrosis and loss of conduction fibers in the proximal bundle branches. Lenegre disease, affecting younger individuals, consists of degenerative changes in more peripheral branches of the conduction system. Both diseases may lead to third-degree AV block. In 1999, degenerative changes in the AV conduction system were linked to mutations of the SCN5A sodium channel gene (mutations of the same gene may lead to congenital long QT syndrome type 3 and to Brugada syndrome).

Infiltrative myocardial diseases resulting in AV block include sarcoidosis, myxedema, hemochromatosis, and progressive calcification related to mitral or aortic valve annular calcification.

Endocarditis and other infections of the myocardium, such as Lyme disease with active infiltration of the AV conduction system, may lead to varying degrees of AV block.

Systemic diseases, such as ankylosing spondylitis and Reiter syndrome, may affect the AV nodal conducting tissue.

Surgical (eg, aortic valve replacement, congenital defect repair) or other therapeutic procedures (eg, AV ablation in patients with supraventricular arrhythmias, alcohol septal ablation in patients with obstructive hypertrophic cardiomyopathy) may cause AV block. Patients with corrected transposition of the great vessels have anterior displacement of the AV node and are prone to develop complete heart block during right heart catheterization or surgical manipulation.

A variety of drugs may affect AV conduction. The most common drugs include digitalis glycosides, beta-blockers, calcium channel blockers, and other antiarrhythmic agents.

**Pathophysiology**

The atrioventricular node (AVN) is a part of the conduction system of the heart that allows electrical impulses to be transmitted from the sinus node via atrial tissue (intra-atrial fascicles) to the ventricles. The AV node consists of 3 parts—atrionodal (transitional zone), nodal (compact portion), and nodal-His (penetrating His bundle). The nodal part demonstrates the slowest conduction. The AV node is supplied by the right coronary artery (90%) or by the circumflex artery (10%) and is innervated by both sympathetic and parasympathetic fibers.

First- and second-degree Mobitz I (Wenckebach) AV blocks usually are caused by a delay at the AV node level, whereas second-degree Mobitz II and third-degree AV blocks are caused by conduction disturbances at the His bundle level or below. In most cases of complete AV block, an escape rhythm originates from the ventricles, with wide QRS complexes at a low regular rate of 30-40 beats per minute. A higher anatomic location of the block results in a higher location of the escape rhythm pacemaker, a faster escape rhythm (40-60 beats per min in the region of His bundle), and a narrower QRS duration.

**CLINICAL**

**History**

First-degree AV block is asymptomatic and usually is an incidental finding on ECG. People with newly diagnosed first-degree AV block may be healthy individuals with high vagal tone (eg, well-conditioned athletes), or they may have a history of myocardial infarction or myocarditis. First-degree AV block also may represent the first sign of a degenerative process of the AV conduction system.

Second-degree AV block usually is asymptomatic. However, in some patients, sensed irregularities of the heartbeat, presyncope, or syncope may occur. The latter usually is observed in more advanced conduction disturbances such as Mobitz II AV block. A history of medications that affect AV node function (eg, digitalis, beta-blockers, calcium channel blockers) may be contributory and should be obtained diligently.

Third-degree AV block frequently is symptomatic, with fatigue, dizziness, lightheadedness, presyncope, and syncope being reported most frequently. Syncopal episodes due to slow heart rates are called Morgagni-Adams-Stokes (MAS) episodes in recognition of the pioneer work on syncope by these researchers in the 19th century. Patients with third-degree AV block may have associated symptoms of acute myocardial infarction either causing the block or related to reduced cardiac output from bradycardia in the setting of advanced atherosclerotic coronary artery disease.

**Physical**

Routine physical examination does not lead to the diagnosis of first-degree AV block.

Second-degree AV block may manifest on physical examination as bradycardia (especially Mobitz type II) and/or irregularity of heart rate (especially type I, Wenckebach).

Third-degree AV block is associated with profound bradycardia unless the site of the block is located in the proximal portion of the AV node. Exacerbation of ischemic heart disease or congestive heart failure caused by AV block–related bradycardia and reduced cardiac output may lead to specific clinically recognizable symptoms (eg, chest pain, dyspnea, confusion, pulmonary edema).

Cannon a waves may be observed intermittently in the jugular venous pulsation when the right atrium contracts against a closed tricuspid valve due to atrioventricular dissociation.

**Lab Studies**

Laboratory testing usually is not indicated in patients with AV block.

Levels of electrolytes and drugs (eg, digitalis) can be checked in the case of second- or third-degree AV block when suspicion of increased potassium level or drug toxicity exists.

In cases when second- and third-degree AV block might be a manifestation of acute myocardial infarction, cardiac enzymes should be measured.

If clinical evaluation suggests systemic illness, appropriate directed laboratory studies for infection, myxedema, or connective tissue disease should be performed.

**Imaging Studies**

Routine ECG recording and cardiac monitoring with careful evaluation of the relationship between P waves and QRS complexes are the standard tests leading to proper diagnosis of AV blocks.

Identifying episodes of transient AV block with sudden pauses and/or low heart rate causing syncopal episodes may require 24-hour Holter monitoring, multiple ECG recordings, event (loop) ECG recordings, or, in selected cases, monitoring with implantable loop recorders (Reveal, Medtronic, Inc).

Electrophysiologic testing is indicated in a patient with suspected AV block as the cause of syncope. The invasive recording of AH (atrium-His) and HV (His-ventricle) intervals may determine the degree of conduction abnormality and may guide decisions for pacemaker therapy.

Routine imaging studies are not helpful in diagnosing AV blocks.

Imaging studies (eg, echocardiography) might be useful in diagnosing underlying comorbidity (eg, aortic valve stenosis with calcification, wall motion abnormalities in acute ischemia, cardiomyopathy, congenital heart disease such as congenitally corrected transposition of the great vessels).

As described, in selected cases, invasive diagnostic procedures may include implantation of loop recorders and electrophysiology testing.

**TREATMENT**

Medical Care

Pacing is the treatment of choice in patients with advanced or symptomatic heart block. No effective long-term medical therapy exists.

First- and second-degree Mobitz I AV blocks do not require treatment. If a drug overdose is a possible cause, the drug needs to be withheld (and its future use or dosage subsequently should be decreased or reconsidered).

Second-degree Mobitz II or third-degree AV block usually requires temporary and/or permanent cardiac pacing. In the setting of acute anterior myocardial infarction, transcutaneous pacing initially and transvenous pacing subsequently are warranted. With inferior myocardial infarction, the block usually resolves spontaneously within several days, and only a small percentage of patients require temporary or permanent pacing. Patients with persistent bundle branch block and transient third-degree AV block may benefit from permanent pacing therapy, especially after anterior myocardial infarction.

Indications for permanent pacing

Class I (generally accepted indications) include the following:

* Symptomatic third-degree AV block
* Asystole 3 seconds or more or escape rhythm 40 beats per minute or less in asymptomatic patients with complete heart block
* Symptomatic second-degree Mobitz II AV block
* Atrial tachycardia with associated symptomatic high-degree AV block

Class II (considered as secondary indications) include the following:

* Asymptomatic complete heart block with ventricular escape rhythm greater than 40 beats per minute
* Asymptomatic second-degree Mobitz II AV block

Asymptomatic second-degree Mobitz I (Wenckebach) AV block at intra-His or infra-His levels

Types of cardiac pacemakers implanted in patients with heart block may include ventricular (usually VVI) or dual chamber (usually DDD) modes of pacing. The electrophysiologist-cardiologist or cardiologist-electrophysiologist should make the decision regarding the most optimal mode of pacing.

Long-term medical therapy is not indicated in AV block. Permanent pacing is the therapy of choice in advanced AV block, and it does not require concomitant medical therapy. Sometimes AV nodal blocking medications that contribute to heart block can be discontinued if not necessary. Temporary transcutaneous or transvenous pacing is the treatment of choice for an emergency involving a slow heart rate (and for asystole) caused by AV blocks. In emergencies where bradycardia is caused by a proximal AV block (located in the AV node), atropine administration may improve AV conduction.

Surgical Care

Pacemaker implantation is a routine surgical procedure, generally performed with local anesthesia in standard surgical facilities. Often, a same-day procedure or 1-day hospitalization occurs.

Activity

Advanced heart block, such as Mobitz II or third-degree AV block, may become more symptomatic with increased activity, where an actual increase in block and decrease in effective heart rate may occur.

Exercise may be used to evaluate 2:1 heart block and differentiate Mobitz I (where the conducted rate increases) from second-degree Mobitz type II heart block (where the block becomes more significant and often symptomatic).

Restrictions after permanent pacemaker implantation include restricted weight lifting with the ipsilateral hand/arm to the pacemaker until healing occurs (approximately 6 wk). Contact sports are restricted unless a protective shield is worn over the implanted pacemaker.

**Further Inpatient Care**

Patients with first-degree and benign second-degree Mobitz I AV block do not require hospitalization.

Patients with symptomatic second- or third-degree AV block require hospitalization with telemetry monitoring. Transcutaneous or transvenous pacing should be utilized, and indications for permanent pacing need to be determined.

**Further Outpatient Care**

Patients with first- and second-degree Mobitz I AV block may require follow-up ECGs or Holter monitoring to determine the likelihood and rate of progression of the AV conduction disorder.

Patients with implanted pacemakers require routine follow-up to monitor pacemaker function.

**Complications**

Sudden death due to asystole or secondary ventricular tachyarrhythmias

Cardiovascular collapse with syncope, aggravation of ischemic heart disease, congestive heart failure, exacerbation of renal disease

Head and musculoskeletal injuries during syncopal episodes

Pacing therapy (temporary or permanent) may be complicated acutely by tamponade, hemothorax, or pneumothorax. Dysfunction of the pacemaker, lead fracture, and malfunction (eg, inappropriate capture or sensing) are infrequent complications of pacing therapy. Infection of the pacemaker or lead wires is a rare, but important, short-term and long-term complication of pacemaker implantation.

**Prognosis**

Patients treated with permanent pacing due to AV blocks have an excellent prognosis. Patients with advanced AV blocks who are not treated with permanent pacing remain at high risk of sudden cardiac death.

**BUNDLE BRANCH BLOCK**

The term *bundle branch block* refers to a conduction block in either the left or right bundle branches. The figure below reviews the anatomy of the ventricular bundle branches.The normal sequence of ventricular activation should be familiar to you by now. The wave of depolarization sweeps out of the AV node and bundle of His into the bundle branch system. The right and left bundle branches deliver the current to the right and left ventricles, respectively. This is the most efficient means of dispersing the electrical current, and the resultant QRS complex, representing ventricular depolarization from start to finish, is narrow—less than 0.1 seconds in duration. Also, because the muscle mass of the left ventricle is so much larger than that of the right ventricle, left ventricular electrical forces dominate those of the right ventricle, and the resultant electrical axis is leftward, lying between 0° and +90°.

Thus, with normal ventricular depolarization, the QRS complex is narrow and the electrical axis is between 0° and 90°. **All of this changes with** bundle **branch block.**

**Bundle branch block is diagnosed by looking at the width and configuration of the QRS complexes.**

*Right Bundle Branch Block*

In *right bundle branch block,* conduction through the right bundle is obstructed. As a result, right ventricular depolarization is delayed; it does not begin until the left ventricle is almost fully depolarized. This causes two things to happen on the EKG:

1. The delay in right ventricular depolarization prolongs the total time for ven­tricular depolarization. As a result, the QRS complex widens beyond 0.12 seconds.
2. The wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle, V]5 and V2. As you know, the normal QRS complex in these leads consists of a small positive R wave and a deep negative S wave, reflecting the electrical dominance of the left ventricle. With right bundle branch block, you can still see the initial R and S waves as the left ventricle depolarizes, but as the right ventricle then begins its delayed depolarization, unopposed by the now fully depolarized and electrically silent left ventricle, the electrical axis of current flow swings sharply back toward the right. This inscribes a *second* R wave, called R', in leads V1 and V2. The whole complex is called RSR', and its appearance has been likened to rabbit ears. Meanwhile, in the left lateral leads overlying the left ventricle (I, AVL, V5, and V6), late right ventricular depolarization causes reciprocal late deep S waves to be inscribed.

*Left Bundle Branch Block*

In *left bundle branch block,* it is *left* ventricular depolarization that is delayed. Again, there are two things to look for on the EKG:

1. The delay in left ventricular depolarization causes the QRS complex to widen beyond 0.12 seconds in duration.
2. The QRS complex in the leads overlying the left ventricle (I, AVL, V5, and V6) will show a characteristic change in shape. The QRS complexes in these leads already have tall R waves. Delayed left ventricular depolarization causes a marked prolongation in the rise of those tall R waves, which will either be broad on top or notched. True rabbit ears are less common than in right bundle branch block. Those leads overlying the right ventricle will show reciprocal, broad, deep S waves. The left ventricle is so dominant in left bundle branch block that left axis deviation may also be present, but this is variable.

*Who Gets Bundle Branch Blocks?*

Although right bundle branch block can be caused by diseases of the conducting system, it is also a fairly common phenomenon in otherwise normal hearts.

Left bundle branch block, on the other hand, rarely occurs in normal hearts and almost always reflects significant underlying cardiac disease, such as degen­erative disease of the conduction system or ischemic coronary artery disease.

*Critical Rate*

Both right and left bundle branch block can be intermittent or fixed. In some individuals, bundle branch block only appears when a particular heart rate, called the *critical rate,* is achieved. In other words, the ventricles conduct nor­mally at slow heart rates, but, above a certain rate, bundle branch block develops.

The development of a rate-related bundle branch block is directly related to the time it takes a particular bundle branch to repolarize and thus prepare itself for the next electrical impulse to come down the pass. If the heart rate is so rapid that a particular bundle branch cannot repolarize in time, there will be a temporary block to conduction, resulting in the classic EKG appearance of a rate related bundle branch block.