Cardiac Arrhythmia Diagnosis

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Cardiac dysfunction is often manifested as arrhythmia, with disruption of the normal periodicity and regularity of electromechanical activity. Cardiac arrhythmias, or abnormalities of cardiac rhythm, are associated with a diverse group of conditions, including congenital, metabolic, structural, physiological, and immunological, and infectious abnormalities. Dysarrhythmia may also be classified as primary because of endogenous electrical abnormalities, or secondary, because of exogenous influences such as ischemia or adrenergic stimuli. Clinical arrhythmia syndromes begin with a single asymptomatic abnormal complex that is benign, progressing to grouped, sustained complexes associated with worsened symptoms and outcome. Proper diagnosis of arrhythmia reflecting symptomology and outcome is essential in acute cardiac care. (Am J Emerg Med 1995; 13:204-210. Copyright © 1995 by W.B. Saunders Company)

The importance of rapid diagnosis and therapy of cardiac arrhythmia is self-evident. Survival from prehospital cardiac arrest is generally poor, with 15% of patients with ventricular tachycardia or fibrillation and 3% with asystole or idioventricular rhythm reaching the emergency department (ED) and surviving to discharge.[1] The outcome of hospitalized patients is slightly better, with 20% surviving tachycardiac or bradycardiac events, 19% surviving ventricular tachycardia-fibrillation, and only 1% surviving episodes of asystole.[2] The pharmacological and mechanical interventions used in resuscitation have a wide range of effectiveness. Antiarrhythmic agents, specifically the class I, type A agents (quinidine, procainamide, disopyramide) are used extensively, in approximately 10 million patients annually.[3] This becomes important when one considers that 54% of patients suffering cardiac arrest while hospitalized are maintained on digoxin or an antiarrhythmic agent.[4] Considerations include failing to diagnose, an error of omission, or overtreating selected arrhythmias, an error of commission, both of which may be detrimental to patients in light of the variable effects or outcomes of the arrhythmias balanced against the adverse effects of therapy.

PHYSIOLOGY

The cardiac cycle is based on the action potential generated in myocardial cells with specific properties of contractility, automaticity, and conduction[5] (Figure 1). Contractile tissue in the atria and ventricles is governed by the fast sodium channel and maintains a stable rest membrane potential. Automatic or conduction cells, including the sinoatrial (SA) and atrioventricular (AV) nodes and, to some extent, the bundle of His and Purkinje's fibers, depend on the slow calcium channel activity and do not maintain a stable rest membrane potential. Thus, cells at rest maintain a negative potential with depolarization, resulting in sodium and calcium influx approaching a more positive baseline. Repolarization occurs with chloride influx and potassium efflux, resulting in a more negative membrane rest potential (Table 1). The Hodgkin-Goldman-Katz equation potassium · hydrogen · magnesium/sodium · calcium quantifies this relation to determine membrane potential and subsequent cardiac membrane irritability.[6]

Arrhythmia generation is dependent on several intrinsic factors. The refractory period, in which sodium channels are unavailable, may be absolute (phase 0, 1, or 2) or relative (phase 3); the rate of conduction depends on the rest membrane potential, slope, and amplitude. The normal action potential has a rest membrane potential of −90 mV and an amplitude of 100 mV and undergoes "all or none" depolarization at a rate of 2.5 m/s. Arrhythmias may occur with a less negative rest potential, a decreased amplitude, or a "decremental" depolarization at a slower rate.[7]

The propensity to develop arrhythmias is also dependent on extrinsic factors. The stimulatory effort is mediated through the stellate ganglion as sympathetic discharge, releasing norepinephrine targeting the beta-1 receptors. The inhibitory influence is mediated through the parasympathetic system, where the vagal nerve, releasing acetylcholine, stimulates the muscarinic receptors and inhibits the beta-1 receptors, resulting in a net inhibitory effect exerted on the heart.

MECHANISM

Several defined correlates determine arrhythmia generation. First, increased diastolic depolarization (phase 4) results in a less negative rest potential (−60 mV) because of increased sodium influx and decreased potassium efflux associated with ischemia, alkalemia, and hyperkalemia. Second, increased conduction and slope or rate of rise (phase 0) caused by increased sodium influx may reach maximum depolarization more rapidly, causing heterogeneous depolarization, usually as a result of an adrenergic exogenous stimulus. Third, the action potential amplitude may be increased, depending on more rapid calcium influx, again resulting in heterogeneous repolarization that is often associated with

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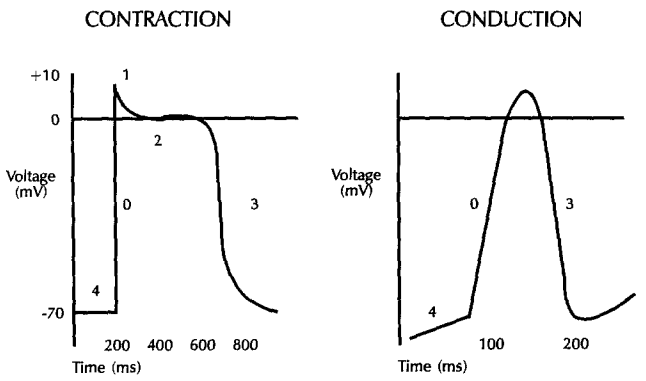

FIGURE 1. Cardiac action potential.

the hyperadrenergic state. Fourth, decreased duration of the action potential occurs as a result of more rapid repolarization, decreasing the effective refractory period with increased potassium efflux caused by hypokalemia or acidemia. These mechanisms manifest in diverse ways as a balance between altered refractoriness and conduction in myocardial tissue. Therapy consists of drugs selected for rapidly binding sodium channels found in ischemic tissue, the phenomenon of "use dependence."[5,7,8]

APPROACH

Diagnosis of arrhythmia begins with consideration of anatomic and physiological variables. Cardiac cells have distinct properties, including automaticity (SA, AV nodes), conduction (bundle of His, Purkinje's fibers), and contractility (monocytes). This conduction system is supplied predominantly by the right coronary artery (55% to 90%), with some left circumflex (10% to 45%) influence. Contractility depends on depolarization of individual monocytes, which is regulated by a number of factors.

The approach to the diagnosis of arrhythmia is to analyze the rhythm rate, regularity (PP, RR), discrete components (P, QRS, T, U), and intervals (PR, QT) to determine their significance for prognosis and therapy (Table 2). Arrhythmias may be classified by origin (atrial or ventricular), regularity (organized or disorganized), rate (bradycardia or tachycardia), QRS complex (narrow or wide), and outcome (benign or malignant) (Table 3).

Atrial Arrhythmias

Commonly encountered arrhythmias may be classified as atrial, ventricular, or conduction delay in origin and regular or irregular in morphology. *Sinus bradycardia* occurs with decreased SA node discharge (40 to 60 BPM) because of excess parasympathetic influence, with maintenance of normal AV conduction (PR interval). This condition is associated with athletic conditioning; vagal hyperactivity; pain; sleep; pharmacological intervention such as narcotic analgesics, calcium channel antagonists, quinidine, procainamide, and digoxin; hypothyroidism; intracranial pressure elevation; and the systolic dysfunction of inferior myocardial infarction.[9-13]

Sinus tachycardia occurs with increased SA node discharge (100 to 150 BPM), with normal AV node conduction. This arrhythmia can occur in the young as a result of anxiety, activity, fever, hypovolemia, pharmacological intervention such as anticholinergic or sympathomimetic medications (eg, theophylline), hypothyroidism, and the diastolic dysfunction of the anterior wall myocardial infarction. This adaptive mechanism may increase cardiac output, compensating for a decreased stroke volume (cardiac output equals stroke volume times heart rate), but it is costly because it increases myocardial oxygen consumption.[9-13]

Atrial tachycardia is a rapid (140 to 220 BPM), regular rhythm that is often initiated by a premature atrial contraction followed by a sustained non-SA node focus. The P wave is present but can be retrograde in nature. The rhythm may be paroxysmal, occurring intermittently, or nonparoxysmal, occurring persistently. The variable rate of conduction ranges from 1:1 (300 BPM), for those with healthy hearts, to 2:1 (150 BPM), often associated with digoxin therapy, to higher grades of block (3, 4, or 5:1), found with intrinsic heart disease. Atrial tachycardia may compromise cardiac output because stroke volume decreases at more rapid rates. The rhythm may be associated with exogenous stimulants such as caffeine, nicotine, cocaine, or amphetamines; medical intervention such as digoxin or theophylline overuse; and endogenous stimulation typified by hyperthyroidism.[14] Therefore, atrial tachycardia is a rapid, regular rhythm with periodicity that is based on the integrity of AV node conduction.[9-11,15]

Supraventricular tachycardia, in its broadest sense, is a heterogeneous group of regular arrhythmias that includes atrial tachycardia, atrial flutter, nodal tachycardia, and accessory pathway syndromes. These arrhythmias originate predominantly from a reentry mechanism (90%), but they may occur via an ectopic or bypass tract (10%).[16] The classic reentry mechanism occurs with fast, anterograde conduction and slow, retrograde movements that then encounter a unidirectional block because of a partial refractory period, establishing a "circus pathway" within the AV node. This rapid rhythm occurs at a rate of between 150 and 200 BPM in an absolutely regular fashion, with anterograde, retrograde, or hidden P waves. Supraventricular tachycardia is found in patients with normal hearts or those with structural or functional damage and can result from pulmonary (infection, chronic obstructive pulmonary disease) or cardiac (ischemia, myocarditis) pathology.[9-11,16]

Atrial flutter is a rapid (260 to 360 BPM) discharge of an autonomic focus. This focus, a counterclockwise macro reentry circuit, is located in the right atrium.[15] This arrhythmia lacks an isoelectric baseline and has a characteristic "sawtooth flutter wave" pattern and an absent P wave. This rapid atrial discharge is conducted with no delay (1:1) in ventric-

TABLE 1. Cardiac Action Potential Phases

Phase	Activity	Voltage	Ion Mechanism	Complex
4	Rest	−90	Na/K Balance	
0	Depolarization	+20	Na in	QRS
1	Equilibration	+15	Cl in	
2	Plateau	0	Ca in, K out	ST
3	Repolarization	−40	K out	T

ABBREVIATIONS: Na, sodium; K, potassium; Cl, chloride; Ca, calcium.

TABLE 2. Electrocardiogram Components

Component	Duration(s)	Mechanism	Association
P Wave	0.1	Atrial depolarization	Cardiac, pulmonary disease
PR Interval	0.12 to .20	AV node delay	Pericarditis
QRS Complex	0.08 to .12	Ventricular & depolarization and atrial repolarization	
ST Segment		Isoelectric plateau	Ischemia
T Wave		Ventricular repolarization	Metabolic (decreased K) autonomic
QT Interval	0.44	Ventricular repolarization	Metabolic (decreased Ca, Mg)
U Wave		After potential	Metabolic (decreased K, increased Ca)

ABBREVIATIONS: Ca, calcium; Mg, magnesium; K, potassium.

ular response through a bypass tract, a slight delay (2:1) through a normal AV node, and a significant delay (4:1) through a diseased AV node. This arrhythmia is usually found in patients with abnormal heart function caused by ischemia, myocardial infarction, or myocarditis and in those with coronary artery disease. The recurrence rate is 50%.[9-11,17]

The next subgroup of atrial arrhythmias includes those that are predominately regular in appearance. *Sinus arrhythmia* is the result of reflex variation of vagal tone with respiration. The atrial rate is between 60 and 100 BPM, increasing with inspiration and decreasing with expiration. Although AV conduction is normal, the RR interval undergoes phasic variation (0.16 second). The rhythm is physiological and is found in healthy adolescents or young adults. This finding has been confirmed in a canine model, implicating age, respiratory rate, and state of wakefulness.[9-11,18]

Premature atrial contraction often is the initiating event for supraventricular tachyarrhythmias. The single atrial atopic focus generates a QRS complex, usually with normal AV conduction but alternate P wave morphology, appearing earlier than expected in the cardiac cycle. Similarly, the atrial depolarization may not be conducted, noted as an isolated P wave. This premature beat then resets the SA node, resulting in a noncompensatory pause and delay in the next conducted complex. Morphological grouping of premature atrial contractions may include atrial bigeminy with paired complexes and atrial tachycardia where the premature atrial contractions are sustained (greater than 3 seconds). This rhythm may be found with sympathomimetic or vasopressor use and in healthy patients or those with pulmonary disease, cardiac ischemia, myocardial wall distention caused by congestive heart failure, or irritability-pericarditis.[9-11,19]

The *wandering atrial pacemaker* is characterized by a mobile atrial impulse with a controlled rate (100 BPM) and variable PR, PP, and RR intervals, found in normal hearts. This abnormality assumes clinical significance because *multifocal atrial tachycardia* is a distinct entity caused by vagal suppression of the SA node, usually found in those with pulmo-

TABLE 3. Arrhythmia Classification

Atrial			Ventricular	
Regular	Irregular	Conduction	Regular	Irregular
Sinus bradycardia	Sinus arrhythmia	SA Node Sick-Sinus syndrome	Parasystole Idioventricular rhythm	Premature ventricular contraction
Sinus tachycardia	Premature atrial contraction	AV Node 1° AV block	Ventricular tachycardia Torsades de Pointes	Ventricular fibrillation
Atrial tachycardia Paroxysmal Nonparoxysmal	Wandering atrial pacemaker	2° AV block Mobitz I (Wenckebach)		
Supraventricular tachycardia	Multifocal atrial tachycardia	Mobitz II (Non-Wenckebach)		
	Atrial flutter	3° AV block		
	Atrial fibrillation	AV dissociation		
		Premature junctional contraction		
		His Bundle—Purkinje		
		Intraventricular conduction delay		
		Hemi-block Left anterior Left posterior		
		Bundle branch block Left Right		
		Accessory pathway Wolff-Parkinson-White Lown-Ganong-Levine		

nary disease. This rhythm is rapid (100 to 160 BPM), originating from the SA node and two ectopic atrial foci, which results in three different P wave morphologies. Therefore, the rhythm appears with variable PR, RR, or PP intervals, but often with normal AV conduction. The rhythm is often associated with chronic pulmonary disease, but may be caused by a wide variety of conditions such as infection, heart disease, or supratherapeutic levels of theophylline.[9-11,20]

Atrial fibrillation is a relatively disorganized rhythm caused by a rapid (400 to 800 BPM), chaotic discharge that is impossible for the AV node to conduct effectively, resulting in an irregular (100 to 200 BPM) rate.[21] The atrial contribution (P wave) to venous return is dyssynchronous, which is more significant in the diseased than in the healthy heart, when cardiac output decreases by 30%, or when mean atrial pressure decreases. The arrhythmia is common especially in patients with heart disease with increased atrial size or mass, and it is often indicated by a deficient radial pulse. Atrial fibrillation is often associated with cardiomyopathy, chronic obstructive pulmonary disease, or hyperthyroidism. Patients may be symptomatic as the heart rate becomes uncontrolled and ventricular filling is compromised. An embolic event may occur in 10% to 25% of patients, and is usually related to spontaneous or electrical conversion.[9-11,21,22]

Atrial fibrillation is part of a continuum, originating with atrial flutter and progressing to fibrillation as disorganization increases. Most such rhythms are in fact a combination, atrial fibrillo-flutter, and are often associated with the use of class I antiarrhythmic agents or operative intervention.[23] Another variation of atrial fibrillation is Ashman's phenomenon, when aberrancy occurs during a rapid ventricular condition. The aberrant beat will follow a "long-short" interval sequence, coinciding with the refractory period.[24]

Ventricular Arrhythmias

Ventricular arrhythmias, generally denoted by a wide (greater than 0.12 second) QRS complex, are markers for more significant cardiac pathology. Regular arrhythmias include *parasystole,* where an isolated, protected Purkinje's fiber with an ectopic focus discharges and competes with the SA node. Parasystole is characterized by a fixed interval between ectopic beats that is reproducible. This arrhythmia is associated with a wide variety of endogenous heart diseases.[9-11]

Idioventricular rhythm, often called "slow ventricular tachycardia," is caused by a ventricular ectopic focus occurring during a slowing period in the SA node. The rhythm consists of a grouped sequence of wide complex beats that may be transient or recurrent. The rate may vary from 50 to 120 BPM, and symptoms may depend on the ability to maintain adequate perfusion. This arrhythmia is often associated with significant myocardial ischemia and may be an end-stage phenomenon.[9-11,25]

Ventricular tachycardia is the most frequently encountered regular ventricular arrhythmia. This rhythm abnormality occurs with a sequence of three or more wide complex beats occurring at a rate of 140 to 150 BPM, almost never greater than 220 BPM. Atrial activity is often associated with an absent or obscure P wave. This rhythm is associated with a wide variety of heart conditions, including ischemia, congestive heart failure, electrolyte abnormalities, and exogenous causes, such as the use of therapeutic or toxic drugs.[9-11,26,27]

A common clinical dilemma is the distinction between a ventricular or supraventricular etiology for wide complex tachycardia.[28,29] Some morphological characteristics are helpful in distinguishing cause and subsequent therapy (Table 4). The distinction between monomorphic ventricular tachycardia, characterized by a single uniform focus and wide complex appearance, and polymorphic ventricular tachycardia, characterized by multiple foci and complexes of variable amplitude, is also clinically important in regard to therapy.

Torsades de pointes is a form of polymorphic ventricular tachycardia that has a characteristic "twisting about a point" appearance. The rhythm is paroxysmal, occurring in short runs, or it may be sustained, at a rate of 200 to 250 BPM. The arrhythmia is caused by atrial refractoriness and is associated with a prolonged QT interval often initiated by a "long on short" sequence. Torsades de pointes is associated with myocardial ischemia, electrolyte abnormalities such as hypokalemia or hypomagnesemia, and the use of drugs such as class I antiarrhythmic agents or antipsychotic agents.[30]

The irregular ventricular arrhythmias typify the continuum of benign to malignant outcome. The *premature ventricular contraction* originates from an ectopic ventricular focus manifested as a wide (greater than 0.14 second) complex. Atrial contraction is disorganized, so the P wave is absent, and the QRS complex is discordant or opposite in polarity from the subsequent P wave. The premature ventricular contraction is followed by a compensatory pause, because the SA node is unaffected by the distant ventricular discharge as opposed to the premature atrial contraction with its noncompensatory pause. Thus, the occurrence of premature ventricular contractions results in an irregular rhythm.[31]

Although premature ventricular contractions usually indicate acute or chronic heart disease, they may also occur in the healthy heart as a result of stress, stimulants, drug effect, or the presence of electrolyte abnormalities such as hypokalemia and hypomagnesemia. The association of prema-

TABLE 4 Differentiation of Wide Complex Tachycardia

	SVT/Aberrancy	Ventricular
Rage		
Regularity	Regular	Irregular
QRS	<0.14	>0.14
AXIS		Left
Pause	Noncompensatory	Compensatory
Coupling Interval	Irregular	Constant
Atrioventricular Dissociation		Present
Fusion		Present
Complex	RBBB	LBBB
V1	rSR	qR
V6	qRS	rS
Polarity	Concordant	Discordant
Cannon A Waves		Present

ABBREVIATIONS: RBBB, right bundle branch block; LBBB, left bundle branch block.

ture ventricular contractions with subsequent outcome was addressed by Lown and Wolf,[32] who studied so-called "marker arrhythmias." The mortality rate progressively increases as rhythm complexity increases from unifocal premature ventricular contractions occurring either more or less frequently than 30/h, multiformed premature ventricular contractions, bigeminy, couplets, and "R-on-T" phenomenon. The last of these marker arrhythmias is significantly associated with the presence of ventricular tachycardia (37% versus 3%).[32]

Ventricular fibrillation is the common endpoint of ventricular arrhythmia degeneration. The rhythm is characterized by a disorganized, irregular appearance, is devoid of organized QRS complexes, and is manifested as loss of consciousness in the patient. The mechanism may be caused by a rapidly discharging ventricular focus without adequate time for repolarization. The arrhythmia is a primary phenomenon in acute myocardial infarction or a secondary event in advanced left ventricular failure. Interestingly, the prognosis and outcome may be better for the former than for the latter condition.[33]

Conduction Delay Arrhythmias

Alteration of myocardial conduction properties, in addition to abnormalities of automaticity, may result in significant arrhythmias. Conduction disease is first localized to the SA node. The *Sick-Sinus Syndrome* appears as a combination of diverse bradyarrhythmias and tachyarrhythmias caused by SA node dysfunction. This condition is manifested as a slowing in SA discharge followed by an erratic escape mechanism. This results in a highly variable P wave rate (40 to 100 BPM), appearance, or regularity, and patients are often symptomatic. The condition may result from disorders caused by idiopathic degeneration (Lenegre's disease) or calcification (Lev's disease) of the conduction system.[34] Patients are predisposed to cardiorespiratory dysfunction resulting from ischemia, hypertrophy, or idiopathic causes.[9-11,35]

Sinus arrest occurs with failure of SA node discharge, which is often caused by an exit block so that no complex is generated. Asystole may occur if no endogenous escape rhythm assumes control of pacemaking. Primary asystole occurs with a normal P wave and absent QRS complex and is associated with previous bifascicular block and sudden death. Secondary asystole occurs in a terminal cardiogenic state with anoxic myocardium that usually results in death. A regular RR interval suggests a SA node exit block, whereas an irregular RR interval indicates primary SA pacemaker failure. This condition may be caused by the effects of drugs such as class I, type A antiarrhythmic agents, digoxin, and narcotics, ischemia, or infectious myocardial conditions.[9-11,35]

The AV node is commonly affected, causing AV delay. *First-degree AV block* is characterized by a PR interval of 0.20 seconds or greater and is found in healthy patients, those treated with digoxin, or in the setting of myocardial ischemia accompanied by an excessive vagal effect.[9-11,36]

Second-degree AV block occurs with progressive nodal delay. *Mobitz Type I (Wenckebach) block* occurs with progressive prolongation of the PR interval, until a subsequent QRS complex is dropped. Proximal nodal involvement results in an otherwise normal ventricular depolarization (QRS complex). Conditions associated with this arrhythmia include digoxin use, inferior wall myocardial infarction, or excessive vagal stimulation of a healthy heart. The rhythm occasionally progresses to complete heart block and, rarely, sudden death.[9-11,37]

Mobitz Type II (non-Wenckebach) block occurs when conduction delay is localized inferior to the AV node. Spontaneous nonconduction occurs without PR prolongation, a condition associated with worsened prognosis. This arrhythmia is associated with heart disease such as inferior or anterior myocardial infarction and often progresses to more advanced conduction delay and sudden cardiac death.[9-11,38]

Third-degree AV (complete) block occurs with conduction delay usually inferior to the AV node. Complete AV dissociation occurs with independent conduction of P wave and QRS complexes. Ventricular conduction may be relatively normal (narrow QRS complex rate of 60 BPM) or abnormal (wide QRS complex rate of 40 BPM) depending on whether the site of delay is the inferior AV node or bundle of HIS, respectively. Patients may become symptomatic with Stokes-Adams syncope as attacks occur. This arrhythmia implies structural heart damage with significant ischemia, especially with delay below the bundle of His.[9-11,39,40]

Atrioventricular dissociation, or atrial and ventricular dysynchrony may be accompanied by a normal or rapid ventricular rate, making diagnosis more difficult. However, AV dissociation is associated with fusion beats as a distinguishing feature. Thus, AV dissociation is necessary for the diagnosis of complete heart block, whereas other specific rhythm abnormalities may be incorporated into this diverse grouping.[9-11,41]

The *premature junctional contraction* occurs with the early discharge of an AV nodal ectopic focus. Bidirectional, retrograde, or anterograde, conduction occurs with a P wave that is absent, accompanied by a normal QRS complex. This rhythm is usually found in abnormal hearts affected by chamber dilation, ischemia, or drug toxicity. The premature junctional contraction is analogous to the premature atrial contraction, which respectively degenerates to junctional tachycardia and atrial fibrillation. Junctional bradycardia occurs when the normal SA node impulse fails, resulting in autonomous discharge from the AV node. This secondary pacemaker function is characterized by an absent P wave (40 to 60 BPM) and a QRS complex that may be wide or narrow depending on the site of AV node automaticity–low or high, respectively. This rhythm is associated with myocardial ischemia, as well as digoxin or quinidine use.[9-11,42]

Junctional tachycardia is classified into two distinct groups. Proximal junctional tachycardia has a rapid (150 to 250 BPM) response, with abrupt onset and termination, and may occur in otherwise healthy patients or in those with metabolic abnormalities or excessive levels of catecholamine. The accelerated junctional rhythm has a slower (70 to 150 BPM) response, with slower onset and termination, and occurs in patients with heart disease such as junctional ischemia, advanced congestive heart failure, and cardiogenic shock.[9-11,43]

Conduction delay may be localized to the bundle of His or Purkinje's fibers. *Nonspecific Intraventricular Conduction Delay* has a prolonged QRS complex (greater than 0.12 sec-

TABLE 5. Ventricular Conduction Delay

Hemiblock	LAH	LPH
QRS	0.12	0.12
Axis	Left >40°	Right >120°
I	qR	rS
III	RS	qR
Bundle Branch Block	**RBBB**	**LBBB**
QRS	>0.12	>0.12
Axis	Right	Left
Intrinsicoid Deflection	0.15	0.15
I		Absent septal Q, notched
V1	rSR	QS
V3	T wave inversion	rS
V6	Deep S	T wave inversion
Repolarization	V1-3	I

ABBREVIATIONS: LAH, left anterior hemiblock; LPH, left posterior hemiblock; RBBB, right bundle branch block; LBBB, left bundle branch block.

ond) caused by diffuse or functional involvement of the bundle of His as a result of hyperkalemia or quinidine or procainamide toxicity.[35] Specific conduction delay may be localized to the left anterior fascicle with inferior ischemia or the left posterior fascicle with anterolateral ischemia.[44] *Incomplete Bundle Branch Block* is manifested as a borderline QRS prolongation (0.10 to 0.12 second). *Right Bundle Branch Block* is a unifascicular delay that occurs commonly because of a longer duration of repolarization and a thin ventricular chamber that is prone to dilation, has a single blood supply, and thus is susceptible to adjacent valve damage.[45] *Left Bundle Branch Block* by definition is a bifascicular block and is indicative of heart damage (Table 5).[46,47]

The final form of conduction disturbance is the *preexcitation syndrome,* which bypasses the normal conduction pathway occurring at an incidence of 0.25% of patients.[48] Accessory tracts include the bundle of Kent connecting with the atrium and ventricle by bypassing the AV node. This disturbance is characterized by upright P waves, a decreased PR interval (less than 0.12 second), QRS complex prolongation, and upstroke slurring (delta wave), designated the *Wolff-Parkinson-White* (WPW) syndrome. WPW syndrome occurs at a rate of 220 to 360 BPM, a differential point compared with ventricular tachycardia, which occurs at rates below 250 BPM.[49]

Type A WPW syndrome, characterized by a left-sided bundle of Kent, features an initial QRS depolarization originating in the posterior left ventricle, manifested as a positive delta wave in leads V_1 and V_2, sometimes appearing as an anterior wall MI or a right bundle branch block pattern. Morphology can be predicted by equating rapid accessory antegrade conduction with a delay in the opposing fascicle. Type B WPW syndrome is characterized by an initial QRS depolarization originating in the inferoposterior right ventricle manifested as a negative delta wave in leads V_1 and V2, sometimes appearing as an inferior wall MI or left bundle branch block pattern. Type C WPW syndrome is characterized by a left lateral accessory bundle with initial QRS depolarization originating in the posterior lateral left ventricle manifested as a negative delta wave in leads V_5 and V_6, sometimes appearing as a posterior wall MI pattern with R:S ratio >1 in leads V_1 and V_2 (Table 6).[9-11,50,51]

Two additional forms of conduction disturbance can be seen in some patients. The first occurs when the Mahaim fibers connect the AV node or His bundle and the ventricle, so that the PR interval is not decreased, but a QRS alteration (delta wave) is present.[51] The second occurs when the James fibers bypass the atrium to the bundle of His, so that the PR interval is decreased (0.08 second), but QRS is normal. This conduction disturbance is known as the *Lown-Ganong-Levine syndrome.*[52]

CONCLUSION

The diagnosis of cardiac arrhythmias is facilitated by an appropriate interpretative scheme that is based on rate, regularity, and morphology of complexes. This information is then used to make a treatment decision that is based on arrhythmia incidence, disease association, and outcome. Accuracy is clearly an important factor, because inappropriate therapeutic intervention may have significant associated sequelae.

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TABLE 6. Preexcitation Syndrome: Accessory Bypass Tracts

Syndrome	Bypass Tract	PR	Depolarization	QRS (Delta)	Pattern
Wolff-Parkinson-White	Kent Atria to Ventricle				
A		↓	Posterior LV	+ V_1, V_2	RBBB AWMI
B		↓	Inferoposterior RV	− V_1, V_2	LBBB IWMI
C		↓	Posterolateral LV	− V_5, V_6	PWMI
	Mahaim AV node to Ventricle	−		+	
Lown-Ganong-Levine	James Atria to His Bundle	↓		−	

ABBREVIATIONS: LV, left ventricle; RV, right ventricle; RBBB, right bundle branch block; LBBB, left bundle branch block; AWMI, anterior wall MI; IWMI, inferior wall MI; PWMI, posterior wall MI.

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