Rhythm Disturbances

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KEYWORDS

• Dysrhythmia • Bradycardia • Supraventricular tachycardia
• Ventricular tachycardia

Patients with cardiac rhythm disturbances may present with a myriad of complaints ranging in severity from palpitations and weakness to chest pain and syncope. Patients may be unstable, requiring immediate interventions, or stable, allowing for a more deliberated approach. Rapid assessment of the patient's stability, underlying rhythm, and determination of appropriate interventions guides timely therapy.

Presenting rhythm disturbances may be primary dysrhythmias or may be secondary to an underlying medical problem. A critical step for the provider is to make this determination early, as this evaluation directly affects treatment. For example, a hypoxemic patient in respiratory failure with significant bradycardia requires attention to oxygenation and ventilation and will not benefit from pacing; a septic patient with sinus tachycardia will not benefit from cardioversion. Conversely, a patient with symptomatic bradycardia requires pharmacologic or electrical therapy to improve heart rate, and an unstable patient in atrial fibrillation with rapid ventricular response requires cardioversion. This article reviews the differential diagnosis and treatment of adult patients presenting with primary bradydysrhythmias and tachydysrhythmias, with the exception of atrial fibrillation and atrial flutter, which are covered in article by Bontempo and Goralnick elsewhere in this issue. A concise approach to diagnosis and determination of appropriate therapy is presented. Challenging scenarios are reviewed, for example, when the etiology of a dysrhythmia is unclear or complicated, as are safe and reliable interventions in such cases. Lastly, critical considerations for specific dysrhythmias are discussed, highlighting high-risk scenarios.

GENERAL APPROACH TO THE PATIENT WITH A CARDIAC RHYTHM DISTURBANCE

Identifying the rhythm disturbance as a primary problem rather than being secondary to a reversible underlying etiology is of critical importance. The use of the word

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"primary" by the authors refers to those pathologies that are intrinsic to the heart, for example, acute myocardial infarction or infiltrative diseases resulting in bradycardia or accessory conduction pathways resulting in reentrant tachycardia. Primary rhythm disturbances may manifest as a result of abnormal impulse formation, ectopic electrical activity, or aberrant conduction. Dysrhythmias secondary to a reversible cause may manifest in the same way as primary rhythm disturbances. However, these patients will require interventions specific to the underlying etiology. The history and physical examination are invaluable for identifying the causes of primary or secondary arrhythmias, as outlined in **Table 1**. For example, patients with a known history of sinus node disease presenting with symptomatic bradycardia are likely to be in heart block, and those on rate-controlling medications are at risk for an adverse drug-related event resulting in bradycardia. Conversely, a patient with a history of supraventricular tachycardia (SVT) presenting with palpitations is likely to have had a recurrence. Physical examination findings consistent with a sepsis syndrome, frank pulmonary edema, or a patient with a history of missed dialysis might direct one away from a diagnosis of primary arrhythmia and toward a treatable underlying etiology. In the unstable patient the reversible causes outlined in **Table 2** must be considered before embarking on purely rhythm-based therapy.

A major determinant of stability in patients with bradycardic and tachycardic rhythm disturbances is heart rate. Patients with extremes of bradycardia and tachycardia will more likely present in an unstable condition, with such symptoms as altered mental status, syncope, dyspnea, chest pain, or cardiovascular collapse, and will require immediate intervention. Patients with impaired cardiovascular function at baseline will be less tolerant of such rhythms. Those with moderate bradycardias or tachycardias are more likely to have limited symptoms, to be hemodynamically normal, and allow time for deliberation and a focused approach to their care. This point is the first juncture in the approach to management of the patient with a primary dysrhythmia: is the patient stable or unstable (**Fig. 1**)? The stable patient allows time for a thorough evaluation and carefully tailored treatment; the unstable patient, defined as having altered mental status, hemodynamic instability, or chest pain considered to be of cardiac origin, requires emergent intervention.

Regardless of whether or not they are stable, all patients with symptomatic dysrhythmias require standard emergency evaluation and treatment. Such assessment includes evaluation of airway, breathing, and circulation with appropriate

Table 1
Potential causes of primary and secondary arrhythmias

	Primary Arrhythmia	Secondary Arrhythmia
Bradycardic	Idiopathic Infiltrative disease Collagen vascular disease Ischemia or infarction Cardiac surgery Heritable arrhythmias Infectious disease (Lyme)	Autonomic syndromes Cardioactive drugs Hypothyroid Known comorbidities Reversible causes (see **Table 2**)
Tachycardic	Known prior dysrhythmia Structural heart disease Prior myocardial infarction	Autonomic syndromes Cardioactive drugs Hyperthyroid Known comorbidities Reversible causes (see **Table 2**)

Table 2
Reversible causes of arrhythmias

Six H's	Five T's
Hypovolemia	Toxins
Hypoxia	Tamponade, cardiac
Hydrogen ion	Tension pneumothorax
Hypo-/hyperkalemia	Thrombosis
Hypoglycemia	Trauma
Hypothermia	

interventions as needed, supplementary oxygen, serial blood pressure monitoring, continuous cardiac rhythm monitoring, continuous pulse oximetry, intravenous access, and a 12-lead electrocardiogram (ECG), with additional testing and interventions as deemed necessary by the treating provider and as guided by the history and physical examination.

APPROACH TO THE PATIENT WITH A BRADYDYSRHYTHMIA

Evaluation

Bradycardia is defined as a heart rate less than 60 beats per minute (bpm). Many patients, such as highly trained athletes, tolerate bradycardia well and require no intervention. Pathologic bradycardias, on the other hand, result from either abnormal impulse formation or abnormal impulse conduction. The sinus node is dysfunctional in disorders of impulse formation, resulting in sinus bradycardia, sinus pause, or sinus arrest. With sinus pause of sufficient duration, and in sinus arrest, junctional or ventricular automaticity may result in slow escape rhythms with rates of roughly 40 to 60 or 30 to 40 bpm, respectively. In the absence of adequately conducted junctional or ventricular automaticity, asystole occurs. Abnormal impulse conduction may result in a variety of heart block conditions: intraventricular conduction delay, first-degree, second-degree type I, second-degree type II, and third-degree heart block. Intraventricular conduction delay and first-degree heart block are not expected to result in symptomatic bradycardia; however, they can confound rhythm interpretation, which is particularly true with bundle-branch blocks.

Interpretation of bradycardic dysrhythmias is relatively straightforward compared with interpretation of tachycardic dysrhythmias, due to the limited range of possibilities. **Box 1** provides a conceptual organization for basic bradycardic dysrhythmias. **Table 3** provides the ECG characteristics for sinus bradycardia and heart blocks

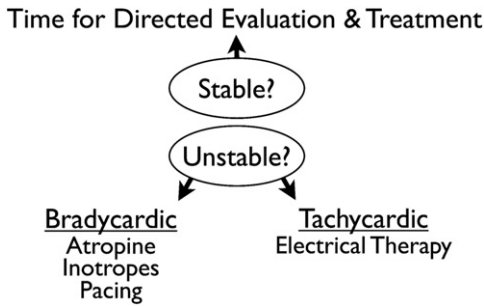

Fig. 1. Algorithm for stable versus unstable presentations.

Box 1
Conceptual approach to bradycardias

Regular, Narrow

Sinus bradycardia

Second degree type I

Slow atrial flutter

Junctional rhythms

Irregular, Narrow

Slow atrial fibrillation

Slow atrial flutter

Slow multifocal atrial tachycardia

Second degree type I or II

Third degree

Regular, Wide

Sinus bradycardia aberrancy

Irregular, Wide

Idioventricular rhythm

Atrial fibrillation/flutter with aberrancy

Heart block with aberrancy

without aberrant conduction as well as important causes to consider during acute presentation of these primary dysrhythmias. Note that the differential diagnosis is not limited to these important causes. Acute coronary syndrome must be considered in all patients presenting with a bradyarrhythmia. Brady and colleagues[1] described

Table 3
Characteristics of bradycardia and heart block

	Sinus Bradycardia	**First-Degree Block**	**Second-Degree Type I Block**	**Second-Degree Type II Block**	**Third-Degree Block**
Atrial rate	**≤60**	Any	Any	Any	Any
Ventricular rate	Matches atrial rate	Matches atrial rate	Slower than atrial rate	Slower than atrial rate	Slower than atrial rate
Rhythm	Regular, P-QRS ratio 1:1	Regular, P-QRS ratio 1:1	Irregular	Regular or Irregular	Irregular
PR	<200 ms	**≥200 ms**	**Prolongs before dropped QRS**	**Fixed before dropped QRS**	**No association**
Acute causes to consider	Normal drugs Vagal tone ACS	Drugs Vasovagal ACS	Drugs Vagal tone ACS	ACS	ACS

Key characteristics are in boldface.
Abbreviation: ACS, acute coronary syndrome.

a case series of 131 patients presenting with unstable bradycardia and heart block, all of whom received atropine and some of whom were paced. Of these, 45 (34%) had a discharge diagnosis of myocardial infarction and 12 (9%) had a discharge diagnosis of ischemia. Of the 45 presenting with atrioventricular (AV) block, 25 (56%) had a discharge diagnosis of myocardial infarction.

Treatment

Stable bradydysrhythmias

The stable patient with asymptomatic bradycardia may be observed, without any specific emergent intervention. For example, a patient with bradycardia due to third-degree heart block may present in stable condition and without symptoms while at rest, though he or she may complain of exertional dyspnea, weakness, or dizziness while ambulating. Such a patient requires hospitalization for evaluation and management of the heart block; however, emergency interventions such as atropine, inotrope infusion, or pacing are not indicated.

Unstable bradydysrhythmias

Atropine is the first-line treatment for symptomatic bradycardia, a recommendation that is supported by consensus opinion as well as research performed in the prehospital setting, emergency department, and operating room.[1–5] The initial dose is 0.5 mg as an intravenous bolus, and may be repeated every 3 to 5 minutes to a maximum of 3 mg. Atropine may prove adequate as a sole intervention, with the patient only requiring further monitoring.

However, if it is ineffective or if recurrent doses of atropine are required, transcutaneous pacing or inotrope infusion should be considered. As with the recommendation for atropine as a first-line agent, consensus and limited scientific evidence support the use of pacing or inotrope infusion as equally effective second-line interventions after atropine.[2–5] The recommended inotropes are dopamine, 2 to 10 μg/kg/min or epinephrine, 2 to 10 μg/min.[3] **Fig. 2** provides a framework for treating the patient with symptomatic bradycardia. Transcutaneous pacing requires sedation and analgesia in the conscious patient, whereas inotrope infusion does not. In all patients, but especially those who do not respond promptly and adequately to atropine, preparations for transvenous pacing should be initiated. Once the patient is stabilized the provider should again review the potential etiology, with particular attention to identifying reversible causes of the abnormal presenting rhythm.

APPROACH TO THE PATIENT WITH A TACHYDYSRHYTHMIA

Evaluation

Tachycardia is defined as a heart rate greater than 100 bpm. Tachycardia is more likely to be a primary arrhythmia when the heart rate exceeds 150 bpm. The upper rate of sinus tachycardia can be approximated by subtracting the patient's age in years from 220. Because a rapid heart rate is often an appropriate response to a physiologic stress such as fever, dehydration, or anemia, such factors should be identified early to determine whether the rate is a primary dysrhythmia or secondary to an underlying condition, with treatment aimed at the underlying cause if present. For example, when cardiac output and systemic perfusion are dependent on heart rate, as in the septic patient, slowing the heart rate may be detrimental. However, when the etiology of sinus tachycardia is not correctable with intravenous volume, analgesics, or anxiolytics, limiting the tachycardic response and, therefore, myocardial oxygen consumption, may be desirable in select critically ill patients.[6]

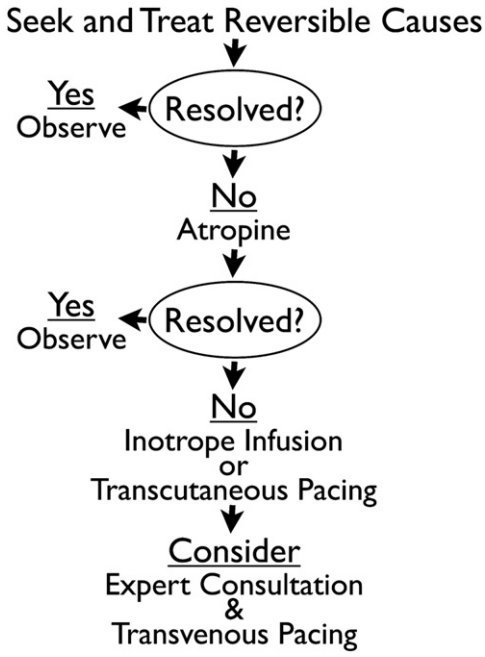

Fig. 2. Approach to treatment of symptomatic bradycardias.

Tachydysrhythmias may be classified based on the regularity of the rhythm and the width of the QRS complex (**Box 2**). Those with a QRS duration less than 120 milliseconds are considered narrow complex tachycardias, with the origin above the bundle of His. When the duration of the QRS complex is greater than 120 milliseconds, the rhythm is considered a wide complex tachycardia. Irregular narrow complex tachydysrhythmias include atrial fibrillation, atrial tachycardia, atrial flutter with variable AV conduction, and multifocal atrial tachycardia. Regular tachydysrhythmias include sinus tachycardia, atrial flutter, atrial tachycardia, AV nodal reentry tachycardia, and AV reentrant tachycardia. Note that some tachydysrhythmias may be regular or irregular, such as atrial flutter. Regular wide complex tachydysrhythmias are generally considered to be ventricular tachycardia or SVT with aberrancy; however, antidromic AV reentrant tachycardia (AVRT) may present as a wide complex tachycardia. Irregular wide complex tachycardias may be polymorphic ventricular tachycardia or an irregular supraventricular rhythm with an abnormal conduction path. Examples of this latter phenomenon include atrial fibrillation, atrial flutter with variable conduction, or atrial tachycardia with variable conduction, in the setting of bundle-branch block or conduction via an accessory pathway such as with Wolfe-Parkinson-White syndrome. Care should be taken to evaluate such rhythms on a 12-lead ECG when possible, as a wide complex tachycardia may appear narrow on a single-lead rhythm strip.[7]

Narrow complex tachydysrhythmias

Narrow complex tachydysrhythmias include sinus tachycardia, atrial flutter, atrial fibrillation, AV nodal reentrant tachycardia (AVNRT), orthodromic AVRT, atrial tachycardia, multifocal atrial tachycardia, and junctional tachycardia. Though atrial fibrillation and flutter are types of SVT, they are reviewed elsewhere in this issue and are not discussed further here.

Box 2
Conceptual approach to tachycardias

Regular, Narrow

1. Sinus tachycardia
2. Atrial flutter
3. Atrial tachycardia
4. AV nodal reentrant tachycardia
5. AV reentrant tachycardia
6. Junctional tachycardia

Irregular, Narrow

1. Atrial fibrillation
2. Atrial tachycardia—variable conduction
3. Atrial flutter—variable conduction
4. Multifocal atrial tachycardia

Regular, Wide

1. Supraventricular tachycardia with aberrancy
2. Ventricular tachycardia

Irregular, Wide

1. Above rhythms with either

a. Aberrancy

b. Accessory pathway

2. Polymorphic ventricular tachycardia

AVNRT is the most common type of nonatrial fibrillation/flutter SVT in adults, with AVRT being the second most common.[8] The cause of AVNRT is a reentrant pathway or tract within the AV node, which appears as a regular, narrow SVT on the electrocardiogram. AVRT is caused by the presence of an abnormal accessory pathway that serves as a conduit for impulses that originate from the sinoatrial node and allows more rapid conduction, bypassing the AV node either on its way to the ventricles (antidromic) or on its return to the atria (orthodromic). The result is a reentrant circuit such as that seen in Wolff-Parkinson-White syndrome.[9] **Fig. 3** provides a visual summary of this concept. In patients with orthodromic AVRT, the tachydysrhythmia may appear as a regular narrow complex SVT indistinguishable from AVNRT. Antidromic AVRT appears as a regular wide complex tachycardia on ECG and may be indistinguishable from ventricular tachycardia, especially if the clinical history of a bypass tract is not known.

Atrial tachycardia is an SVT that can originate from a single focus of atrial tissue or be multifocal in origin, and is the result of increased automaticity.[10,11] Atrial tachycardia can be either focal or macro-reentrant. In focal atrial tachycardia, atrial activation starts rhythmically in a small area from which it spreads out centrifugally. In macro-reentry, reentrant activation occurs around a central obstacle; this would include, for example, typical atrial flutter.[11] Multifocal atrial tachycardia is defined as a rhythm with an atrial rate greater than 100 bpm, at least 3 morphologically distinct P waves, irregular P-P intervals, and an isoelectric baseline between P waves.[12]

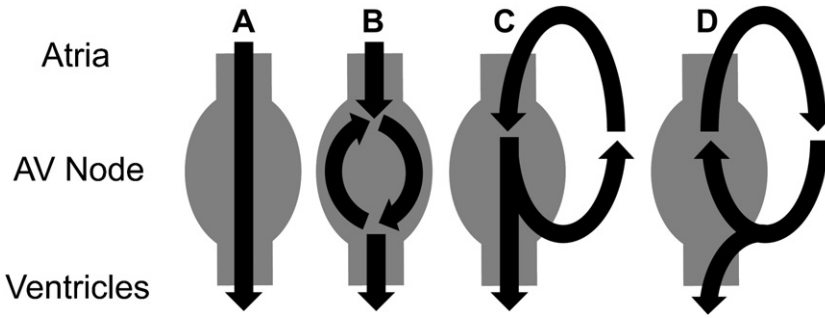

Fig. 3. Conduction through the atrioventricular (AV) node in sinus rhythm and paroxysmal supraventricular tachycardia. (*A*) Sinus rhythm; (*B*) atrioventricular nodal reentry tachycardia; (*C*) orthodromic atrioventricular reentry tachycardia; (*D*) antidromic atrioventricular reentry tachycardia.

Junctional tachycardia is also a disorder of increased automaticity, although it is relatively rare. It is approached in a manner similar to the atrial tachycardias.

Wide complex tachydysrhythmias

Differentiating between ventricular tachycardia and SVT with aberrant conduction can be challenging, and the rhythm should be assumed to be ventricular tachycardia in the absence of clear-cut evidence to the contrary. Clinical findings that support the diagnosis of ventricular tachycardia include symptoms consistent with myocardial infarction or ischemia, prior myocardial infarction or coronary artery disease, and symptoms occurring in older patients.[13] Hemodynamic stability should not be used to discriminate between ventricular tachycardia and SVT with aberrancy.[14] Factors associated with the diagnosis of SVT with aberrancy include young patients, prior bundle-branch block or similar appearance on prior electrocardiogram, and a history of SVT with aberrancy. Several decision rules have been proposed in attempts to identify features on the ECG to differentiate ventricular tachycardia from SVT with aberrancy. Brugada and colleagues[15] proposed a simplified algorithm based on 4 criteria applied in a stepwise manner: absence of RS in all precordial leads; R to S interval greater than 100 milliseconds in one precordial lead; AV dissociation; and morphologic criteria for ventricular tachycardia present in V1, V2, and V6. The criteria are designed to be applied in a stepwise manner, and if at any step criteria are present the diagnosis is ventricular tachycardia. If none are present the rhythm is determined to be SVT with aberrancy. These investigators prospectively applied their criteria and demonstrated a sensitivity and specificity of 98.7% and 96.5%, respectively, for correctly identifying ventricular tachycardia. Other investigators have found the Brugada criteria to be useful, though not as reliable as reported by Brugada and colleagues, and have proposed alterative approaches.[16–18] Overall, although algorithmic approaches to identifying the etiology of wide complex tachycardias are helpful, there are a substantial number of cases where even expert electrocardiogram analysis is indeterminate.[17]

Treatment

Unstable narrow complex tachydysrhythmias

Management of the adult patient presenting with an unstable tachydysrhythmia can be relatively straightforward if certain fundamental decision points are considered and acted on appropriately, as outlined in **Fig. 4**. The critical actions are the following: assess patient stability, identify and treat underlying causes if present, identify the

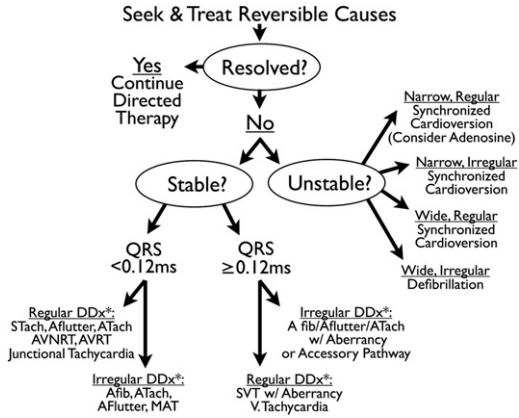

Fig. 4. Approach the patient with tachycardia. Note the emergent interventions for unstable rhythms and the differential diagnosis for stable rhythms. * Readers are referred to the text for discussion of interventions for stable tachydysrhythmias. Afib, atrial fibrillation; Aflutter, atrial flutter; ATach, atrial tachycardia; AVNRT, atrioventricular nodal reentry tachycardia; AVRT, atrioventricular reentry tachycardia; DDx, differential diagnosis; MAT, multifocal atrial tachycardia; STach, sinus tachycardia; SVT, supraventricular tachycardia; w/, with.

heart rate, rhythm, and QRS morphology, and select electrical and/or pharmacologic interventions. Unstable patients, for example, those with a systolic blood pressure less than 90 mm Hg, chest pain, or altered mental status, with a narrow complex SVT that is not a compensatory sinus tachycardia, should be immediately cardioverted. Cardioversion of nonatrial fibrillation SVT can be successful with low energies, from 50 to 100 J when using a biphasic defibrillator, and 200 J when using monophasic defibrillator.[3,19] If the initial cardioversion fails, then the dose should be increased in a stepwise fashion.[20] Although the current American Heart Association guidelines generally recommend synchronized cardioversion as the treatment of choice for all unstable tachydysrhythmias, a trial of adenosine may be considered in patients with mildly unstable narrow complex regular SVT before cardioversion.[3] This recommendation is based on retrospective evidence showing that adenosine may convert unstable narrow complex SVT promptly and resolve hemodynamic instability.[21–25]

Stable narrow complex tachydysrhythmias

The differential diagnosis for stable narrow complex supraventricular rhythms is discussed above. For the purposes of this article, discussion of specific interventions is limited to those for AVNRT, AVRT, and atrial tachycardias, excluding atrial fibrillation and atrial flutter. The goal in treatment of these tachydysrhythmias is rate and rhythm control. For AVNRT and AVRT, as opposed to disorders of atrial automaticity, the distinguishing feature is a reentry circuit involving the AV node. Both AVNRT and orthodromic AVRT (jointly referred to as SVT for the remainder of the article) will present as narrow complex regular tachycardias. Regular, stable, monomorphic, wide complex tachycardias in the setting of known preexisting bundle-branch block that do not meet criteria for ventricular tachycardia may also be supraventricular in origin. Similarly, antidromic AVRT will present as a regular wide complex tachydysrhythmia. Discussion of these more complicated wide complex tachyarrhythmias is expanded in the section on stable wide complex tachycardia.

The approach to treating SVT is relatively straightforward and may involve either vagal maneuvers, adenosine, or nodal blocking agents (calcium-channel blockers or β-blockers). Vagal maneuvers and adenosine are the preferred initial interventions. The most widely used vagal maneuver is the Valsalva maneuver, with a success rate of approximately 15% to 20%.[26] However, success rates of up to 30% have been reported using a modified Valsalva maneuver whereby patients expired into a section of suction tubing and pressure gauge for at least 15 seconds at 40 mm Hg while in a Trendelenburg position.[27] Carotid massage may be considered. As a diagnostic tool it may slow the ventricular rate to assist in rhythm analysis, and as a therapeutic tool it is equivalent to Valsalva in terminating SVT.[28] However, concerns have been raised regarding the safety of carotid massage especially in the elderly, with neurologic complications being reported in 0.1% to 1%.[29,30]

Adenosine is the second-line agent if SVT does not respond to vagal maneuvers, and has several advantages over other agents. For example, it demonstrates similar conversion rates to calcium-channel blockers with the advantage of a more rapid onset and fewer side effects. It has a short half-life, which allows for use of other AV nodal blocking agents if necessary.[31–33] Adenosine is administered as a rapid intravenous bolus, with an initial dose of 6 mg. If the rhythm does not convert, the dose should be increased to 12 mg and repeated. Common side effects are flushing, dyspnea, and chest discomfort.[34] Bronchospasm has been reported following adenosine administration in patients with underlying asthma or chronic obstructive pulmonary disease; however, it has also been reported to be given safely in such cases, and as such is a relative contraindication.[26,35] Patients treated with adenosine may convert to a different arrhythmia, including prolonged AV block, paroxysmal atrial fibrillation, nonventricular tachycardia, transient premature ventricular complexes, torsades de pointes, atrial fibrillation, or atrial flutter, with such conversions being noted to occur in 13% of patients.[34] Most of these arrhythmias are transient and recover spontaneously. When an underlying preexcitation syndrome such as Wolff-Parkinson-White is suspected despite a presentation with a narrow complex regular tachycardia, a defibrillator should be available out of concern for the possibility of inducing atrial fibrillation with rapid ventricular response.

When SVT fails to convert to sinus rhythm, recurs, or when treatment with vagal maneuvers or adenosine reveal atrial fibrillation or atrial flutter, treatment with AV nodal blocking agents is indicated. These agents include the nondihydropyridine calcium-channel blockers verapamil and diltiazem, and β-blockers. Verapamil and diltiazem have similar efficacy in the treatment of narrow complex SVT, though they have a more concerning side effect profile when compared with adenosine.[31,33,36–39] Verapamil and diltiazem should only be used in cases known to be supraventricular in origin, and they should avoided in patients with reduced left ventricular function. All nodal blocking agents should be avoided in patients with wide complex tachycardias, preexcited atrial fibrillation, or atrial flutter. The initial dose of verapamil is 5 mg, administered intravenously. If there is no response and no drug-related side effects, the dose can be repeated every 15 minutes up to a total of 20 to 30 mg. The initial dose for diltiazem is 20 mg, and an additional 20 mg may be administered as needed after 15 minutes. A diltiazem maintenance infusion at 5 to 15 mg/h may be used if continuous rate control is required.[3] Pretreatment with 1 g calcium gluconate or 333 mg calcium chloride has been suggested as an intervention to act against hypotension during calcium-channel blocker use.[40]

β-Blockers slow conduction through the AV node and reduce sympathetic tone. Though suggested as second-line agent for SVT, the evidence supporting their use is more limited.[41–45] Like calcium-channel blockers they have a significant

side-effect profile in this setting, including bradycardia, conduction delays, and hypotension. Furthermore, they are less effective when compared with calcium-channel blockers.[46,47] Serial use of these long acting AV nodal blocking drugs should be avoided, as hypotension and bradycardia are a significant risk.[3] Alternative drug therapy includes antiarrhythmic agents such as amiodarone, procainamide, or sotalol. However, these drugs have potential for toxicity and proarrhythmic effects, and as such their use in SVT is limited.

Lastly, atrial tachycardia and multifocal atrial tachycardia are considered. These disorders are of increased automaticity at a single or multiple atrial sites, respectively. These conditions differ from SVT in that the ectopic electrical activity resides in the atria, and there is no reentrant circuit through the AV node. Multifocal atrial tachycardia is typically an epiphenomenon of an underlying disorder such as hypoxemia, chronic obstructive pulmonary disease, congestive heart failure, and electrolyte disorders.[10] Reversal of precipitating causes remains the cornerstone of treatment for multifocal atrial tachycardia, as this may be all that is required to control the arrhythmia and avoid the potentially harmful effects of antiarrhythmic agents. If the arrhythmia persists, the clinical significance of the tachycardia must be evaluated before the use of antiarrhythmics is considered. Because they are disorders of automaticity, electrical cardioversion is not effective and pharmacologic therapy is required. Agents to consider are β-blockers, calcium-channel blockers, and amiodarone. Metoprolol has been shown to be effective for rate control in a double-blind, placebo-controlled trial, and may be considered a first line in the treatment of multifocal atrial tachycardia in the absence of contraindications.[48,49] However, in patients with impaired left ventricular function, drugs with negative inotropic properties should be avoided. In such cases amiodarone may be preferred.[50,51]

Unstable wide complex tachydysrhythmias

Synchronized cardioversion is recommended for unstable monomorphic ventricular tachycardia when a pulse is present, with initial energies of 100 J whether using a biphasic or monophasic defibrillator. Pulseless ventricular tachycardia is treated the same as ventricular fibrillation, and is defibrillated. The initial energy is 120 to 200 J for biphasic defibrillators based on the manufacturer's recommendation, or 360 J for monophasic defibrillators. Unstable wide irregular tachycardias should also be treated with defibrillation using the same energy setting.

Polymorphic ventricular tachycardia presents as an irregular wide complex tachycardia with varying QRS morphology. It is typically associated with hemodynamic instability, and often degenerates to ventricular fibrillation. As outlined earlier, the treatment of choice for such unstable patients is defibrillation with the same strategy as for ventricular fibrillation, and attention to identifying and treating the underlying cause of the dysrhythmia.[3] Efforts should be directed to identifying and treating ischemia, electrolyte abnormalities, or drug toxicities that may have precipitated the arrhythmia. For patients who have experienced polymorphic ventricular tachycardia without a prolonged QT interval, the most common cause is myocardial ischemia. In such cases, when the dysrhythmia has resolved, β-blockers are appropriate, amiodarone may be effective in preventing arrhythmia recurrence, and prompt percutaneous coronary intervention should be considered.[3] Less common causes of polymorphic ventricular tachycardia include Brugada syndrome and catecholaminergic ventricular tachycardia, which may be responsive to isoproterenol and β-blockers, respectively. However, expert consultation is advised.

Polymorphic ventricular tachycardia may or may not be torsades de pointes, and this cannot be distinguished on the ECG during a period of ventricular tachycardia.

Torsades de pointes is diagnosed when a prolonged QT interval is known to be present or is observed during a period of sinus rhythm. It is often preceded by a period of bradycardia, and is more likely to occur in the setting of electrolyte abnormalities or when preceded by a QT-prolonging antiarrhythmic infusion such as procainamide, quinidine, or sotolol. Whether torsades de pointes is due to a congenital long-QT syndrome or if it is acquired, the management approach is similar. Magnesium infusion of 1 to 2 g over 15 minutes may be effective.[52–54] In patients with recurrent polymorphic ventricular tachycardia accompanied by bradycardia or precipitated by pauses in rhythm, temporary overdrive pacing is appropriate.[55] Similarly, isoproterenol may be an effective treatment in drug-induced QT prolongation by increasing heart rate and shortening the QT interval.[56] However, it should be avoided in patients with suspected ischemia because it increases myocardial oxygen demand, as well as in patients with congenital long-QT syndromes. QT-prolonging drugs should be recognized and discontinued, and electrolyte abnormalities should be corrected.

Stable wide complex tachydysrhythmias

As in stable narrow complex tachycardia, the first step in management is to determine whether the rhythm is regular or irregular. A regular wide complex tachycardia is either ventricular tachycardia, SVT with aberrant conduction (AVNRT with aberrancy or orthodromic AVRT with aberrancy), or antidromic AVRT. Irregular wide complex tachycardias may be atrial fibrillation with aberrancy, preexcited atrial fibrillation, or polymorphic ventricular tachycardia (torsades de pointes). A very rapid (>220 bpm) irregular wide complex tachycardia is virtually pathognomonic of preexcited atrial fibrillation.[57]

If the origin of a stable, regular, monomorphic wide complex tachycardia cannot be determined, adenosine has been demonstrated to be safe and effective for diagnostic and therapeutic purposes.[58] Furthermore, the likelihood of making a correct diagnosis of SVT or ventricular tachycardia increases. If the underlying rhythm is SVT with aberrant conduction, administration of adenosine will likely result in conversion to sinus rhythm.[58] If the rhythm is ventricular tachycardia there will likely be no effect, although there is a subset of patients with ventricular tachycardia responsive to adenosine.[59] Adenosine should not be used in irregular or polymorphic ventricular tachycardia. The treating provider should be prepared for defibrillation under such circumstances.

Given the diagnostic uncertainty often seen in cases of wide complex tachycardia, the default should be to treat such rhythms as ventricular tachycardia unless there is clear evidence to the contrary.[60] Cardioversion or antiarrhythmic drugs are the recommended treatment strategy. Cardioversion is a safe, rapidly effective intervention with relatively few side effects even in the setting of stable ventricular dysrhythmias. When a pharmacologic approach is desired, the recommended drugs include procainamide, amiodarone, and sotolol. Procainamide and sotolol should be avoided in cases of prolonged QT interval. In cases where one pharmacologic agent has not been effective, cardioversion should be attempted or expert consultation obtained before administration of a second pharmacologic agent.[3]

Procainamide is the preferred pharmacologic agent for treatment of hemodynamically stable ventricular tachycardia when cardioversion is not selected as the first-line intervention. Procainamide has a relatively fast onset of action and terminates ventricular tachycardia in to 80% to 90% of patients.[61] Procainamide is recommended as an infusion of 20 to 50 mg/min to a total dose of 17 mg/kg. The other end points are arrhythmia suppression, hypotension, or QRS duration increase of greater than 50%. Hypotension is more common with higher infusion rates.[62] Procainamide should be avoided in patients with reduced left ventricular function, congestive heart failure,

or prolonged QT interval. Although procainamide may be more effective than amiodarone in the short term, it should be noted that no difference has been demonstrated in treating sustained ventricular tachycardia, and that amiodarone is the preferred agent in patients with reduced left ventricular function.[50,63]

Amiodarone has proved useful in hemodynamically unstable and recurrent ventricular tachycardia.[64,65] Some studies have demonstrated it to be effective in hemodynamically unstable patients with monomorphic ventricular tachycardia refractory to other medications, whereas others have shown that it is no more effective than other antiarrhythmics.[66–68] Care should be taken to monitor for hypotension, although it is thought that this adverse effect was attributable to the solvent used in older formulations of the drug.[69] The dose of amiodarone is 150 mg over 10 minutes repeated to a maximum of 2.2 g over 24 hours.

Sotalol is much less commonly used in emergency medicine practice, although it has been used with success to treat stable sustained monomorphic ventricular tachycardia, and has been demonstrated to be more effective than lidocaine.[70,71] The dose of sotalol is 100 mg (1.5 mg/kg) given over 5 minutes.

Although management of atrial fibrillation is discussed elsewhere, the special case of atrial fibrillation with AVRT (Wolff-Parkinson-White syndrome) is mentioned here, as it is in the differential for wide complex tachycardias and has specific management considerations. AV nodal blocking agents used for most supraventricular rhythms (adenosine, calcium-channel blockers, β-blockers) should be avoided in this circumstance. These agents slow conduction through the AV node, but do not affect conduction through the accessory pathway, which can allow very rapid conduction of up to 300 to 400 bpm.[72] In such cases AV nodal blocking agents may accelerate conduction over the accessory pathway, resulting in cardiovascular decompensation and ventricular fibrillation.[73,74] In general, patients with preexcited atrial fibrillation are unstable, with very rapid heart rates, and require emergent cardioversion. Pharmacologic interventions should be approached with great caution, and only in cases where cardioversion is not appropriate or effective.[3] Agents to consider include procainamide and amiodarone. Procainamide increases the antegrade effective refractory period and the intra-atrial conduction time.[75] Schatz and colleagues[76] support procainamide as the favored pharmacologic treatment for patients with preexcited atrial fibrillation, due to its ability to safely lengthen the effective refractory period. Amiodarone may be effective, but recent studies have shown a small yet serious risk of ventricular fibrillation in this setting.[77–81] The use of either amiodarone or procainamide may also result in the termination of the atrial rhythm, as such thromboembolic complications must be considered.

SUMMARY

Patients with cardiac rhythm disturbances can be challenging to manage, especially when they present under emergent conditions. High-quality, safe emergency care involves rapid assessment focusing on identification of the unstable patient, recognition of comorbidities, dysrhythmia diagnosis, and prompt intervention. A useful cognitive strategy is to categorize patients as stable or unstable, and the rhythm as fast or slow, regular or irregular, and wide or narrow. The differential diagnosis is hence narrowed, and focused interventions can be selected in a rapid, informed, and organized manner.

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