Life-Threatening Arrhythmias in the Intensive Care Unit

Scott Reising, MD*
Fred Kusumoto, MD†
Nora Goldschlager, MD‡

Symptomatic arrhythmias are frequently observed in the intensive care unit and often lead to significant hemodynamic compromise because of the presence of multisystem disease. In particular, prompt evaluation of patients with tachycardia is critical because treatment depends on the accurate diagnosis of the arrhythmia mechanism. The electrocardiogram remains the most important diagnostic tool for the evaluation of both wide complex and narrow complex tachycardia. For wide complex tachycardia, evaluation of the atrioventricular relationship and QRS morphology are critical, and for narrow QRS complex tachycardias, evaluation focuses on identification of the location and morphology of P waves. Bradycardia can arise from sinus node dysfunction or atrioventricular conduction block.

Key words: *tachycardia; bradycardia; wide complex tachycardia*

Symptomatic or sustained arrhythmias are observed in approximately 20% of patients in the intensive care unit (ICU) [1]. Tachycardias are responsible for more than 90% of abnormal rhythms in the ICU, with the greatest proportion in most series due to atrial fibrillation (30%-60%) [1,2]. Surgical procedures, hypoxia, high cardiac filling pressures, greater degrees of physiologic derangement, sepsis, and the systemic inflammatory response syndrome appear to be significant independent risk factors for the development of abnormal tachycardias [1,2]. This review will discuss the diagnosis and treatment strategies of abnormal rhythms encountered in the ICU with an emphasis on electrocardiographic evaluation.

Tachycardias

Clinicians generally classify tachycardias by their electrocardiogram (ECG) characteristics: wide QRS complex versus narrow QRS complex and regular versus irregular. Although the broad classification scheme is very useful and will be used in this review, it is also important to keep pathophysiologic and anatomic mechanisms of tachycardias in mind. Tachycardias can arise from 2 basic pathophysiologic mechanisms. First, in the presence of 2 electrophysiologically separate paths, perfectly timed premature beats can initiate a reentrant circuit that continually reactivates itself. Reentry is the most commonly encountered arrhythmia mechanism and is the basis for most forms of ventricular tachycardia (VT), atrial flutter, and tachycardias involving accessory pathways. Abnormal automaticity due to rapid repetitive depolarization from a single cell or group of cells can also lead to rapid rates. Although not abnormal, sinus tachycardia due to concomitant disease or β agonists is an example of increased automaticity leading to rate heart rates. Abnormal automaticity is less common than reentry but is the basic mechanism for multifocal atrial tachycardia, focal (ectopic) atrial tachycardia, and torsade de pointes VT.

As schematized in Figure 1, tachycardias can be classified by the location of the tachycardia site. Using this concept, the atrial tachyarrhythmias would include atrial fibrillation, atrial flutter, multifocal atrial tachycardia, and focal (ectopic) atrial tachycardia. In tachycardias involving the atrioventricular (AV) node or AV junction, a reentrant circuit or, more rarely, an automatic focus is located in the region of the AV node. Reentry within the AV nodal region (AV node reentrant tachycardia) is the most common tachycardia involving the AV junction in adults. In VT, the

From the *Department of Community Internal Medicine, Mayo Clinic, Jacksonville, Florida; †Electrophysiology and Pacing Service, Division of Cardiovascular Disease, Department of Medicine, Mayo Clinic, Jacksonville, Florida; and the ‡Cardiology Division, Department of Medicine, San Francisco General Hospital, San Francisco, California, and the Department of Medicine, University of California, San Francisco.

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Address correspondence to Fred Kusumoto, MD, Electrophysiology and Pacing Service, Division of Cardiovascular Disease, Mayo Clinic, 4500 San Pablo Avenue, Jacksonville, FL 32224, or e-mail: kusumoto.fred@mayo.edu.

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reentrant circuit or site of automaticity is located within ventricular tissue. Finally, in accessory pathway-mediated tachycardias, the accessory pathway provides a pathway parallel to the AV node that forms the substrate for reentry. The most common form of accessory pathway-mediated tachycardia is orthodromic AV reentry, where the ventricles are activated via the normal AV node–His–Purkinje system and retrograde activation of the atria occurs over the accessory pathway. Two less common types of tachycardia observed in patients with accessory pathways are antidromic AV reentry, in which the ventricles are activated via the accessory pathway and retrograde activation occurs via the AV node, and atrial tachycardia, with rapid ventricular activation via the accessory pathway. Atrial tachycardias, AV node or junctional tachycardias, and orthodromic AV reentry tachycardias cause narrow QRS complex tachycardias (supraventricular tachycardias). Abnormal wide QRS complex tachycardias are most commonly due to VTs, although supraventricular tachycardias from any mechanism with abnormal ventricular activation due to an accessory pathway or conduction delay in the left or right bundle branch will lead to a wide QRS complex tachycardia; the latter is referred to as "intraventricular aberration."

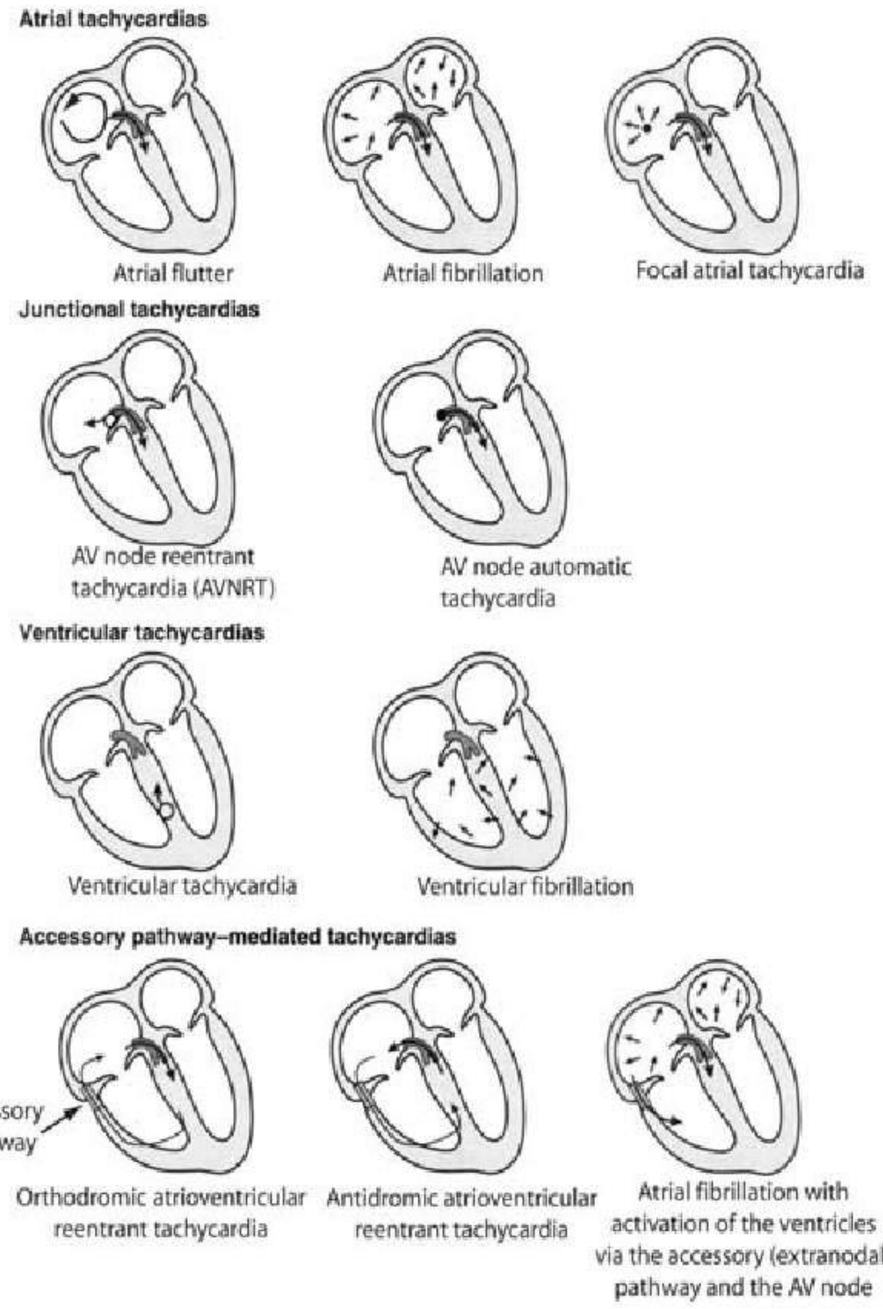

Fig 1. Schematic drawing illustrating an anatomic classification for tachycardias. Atrial tachycardias, tachycardias involving the atrioventricular (AV) node (junction), and orthodromic reciprocating tachycardias produce narrow QRS complexes unless prior bundle branch block exists. VTs, atrial tachycardias with ventricular activation over an accessory pathway, and antidromic reciprocating tachycardias produce wide QRS complex tachycardias. Orthodromic = ventricular depolarization over the normal AV node–His–Purkinje pathways; antidromic = ventricular depolarization via an extra-AV nodal accessory pathway with retrograde atrial activation occurring over the normal conducting pathways.

Regular Wide Complex Tachycardias

Pathophysiology

VT is the most common rhythm causing wide complex tachycardia. In studies of patients with wide complex tachycardia referred for electrophysiologic study, approximately 80% were found to have VT [3-6]. Because of referral bias, however, the true incidence of VT in ICU patients is probably lower. Other causes of wide complex rhythms that the intensivist must consider include bundle branch block, tricyclic antidepressant overdose, hyperkalemia, and ventricular pacing, all of which cause an increase in QRS duration, regardless of the atrial rhythm.

Electrocardiography

Two centuries ago, Galvani and Volta demonstrated that electrical phenomena were involved in the spontaneous contractions of the heart [7]. Recording cardiac electrical activity from surface electrodes (the ECG) has become the essential tool for evaluating cardiac rhythms. Once a patient's clinical status has been assessed, the ECG should be evaluated in an attempt to differentiate VT from supraventricular tachycardia with intraventricular aberrancy (Figure 2). It is important to acknowledge the limitations of ECG criteria for evaluating wide complex tachycardia. Using a variety of published criteria from 6 different studies, Drew and Scheinman found that approximately 10% of ECGs "defy differentiation," and that "tachycardias >190 beats/minute often do not exhibit unequivocal criteria with which to make a certain diagnosis" [8].

Clinical Clues to Rhythm Diagnosis

The clinical history can be useful for identifying patients with VT. VT is the most likely diagnosis in

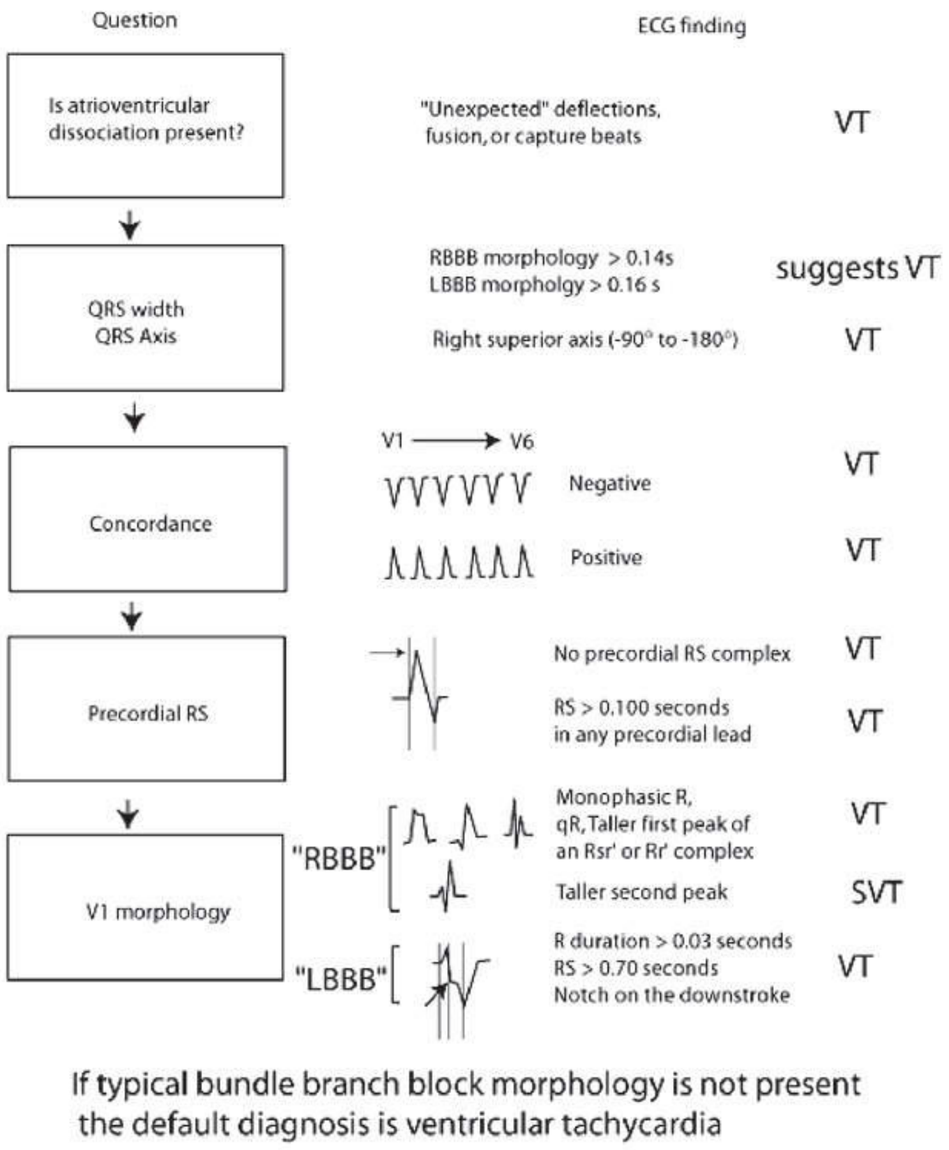

Fig 2. Electrocardiogram flow diagram for evaluation of wide complex tachycardia. VT = ventricular tachycardia; SVT = supraventricular tachycardia; RBBB = right bundle branch block morphology; LBBB = left bundle branch block morphology.

patients with cardiac disease. In one study, histories of myocardial infarction, congestive heart failure, or recent angina pectoris all had positive predictive values >95% for a wide QRS complex tachycardia's being VT [5]. In another study of conscious patients with wide complex tachycardia, if the patients answered "yes" to 2 questions ("Have you had a heart attack in the past?" and "Did symptoms start after your heart attack?"), VT was the diagnosis in 28 of 29 cases [9].

On physical examination, variable intensity of the first heart sound and identification of cannon A waves in the venous pulsations in the neck indicate AV dissociation and are highly specific and sensitive findings in VT [10]. It is most important to note that hemodynamic stability is less useful. In one study of 20 conscious patients with wide complex tachycardia of mean duration of 4.8 hours, mean heart rate of 186 beats per minute, and mean systolic blood pressure of 111 mm Hg, VT was established as the cause of the arrhythmia in 85% of the cases [4]. Thus, hemodynamic stability does not rule out VT, nor does hemodynamic instability confirm VT, as supraventricular arrhythmias can themselves cause hemodynamic collapse.

Atrioventricular Relationships

Electrocardiographic evaluation for VT should begin with identifying the AV relationship. In VT, if the atrial rhythm is sinus, P waves that occur independent of the QRS complexes (AV dissociation) can be discerned. AV dissociation can be identified by irregular deflections in the QRS complex, ST segment, or T waves that occur at a regular rate but do not have a relationship to the QRS rate. AV dissociation is most easily identified in the inferior leads (II, III, aVF) and V1. In some cases, P waves can be seen using a Lewis lead, in which the right arm electrode is placed in the right parasternal area at the second or third interspace, the left arm lead is placed in the left parasternal area in the fourth or fifth interspace, and ECG from lead I is recorded. Evidence of AV dissociation can also be identified by the presence of capture and fusion beats (Figure 3): if the tachycardia rate is relatively slow, a random but properly timed atrial impulse can be conducted to the ventricles, leading to normal QRS complexes (capture beats) or near-normal QRS complex morphology in which the complexes are intermediate between a normal QRS complex and the VT complex (fusion beats). Although highly specific for VT, AV dissociation and its manifestations (fusion and capture beats) can be identified in only 25% to 40% of cases [11].

QRS Duration

The QRS duration can provide some clues to the mechanism of wide complex tachycardia. If the QRS complex is predominantly negative in lead V1, it is classified as having left bundle branch block (LBBB) morphology. If the QRS complex is predominantly positive in lead V1 (eg, RsR′, RR′, Rs, qR) and without a terminal conduction delay, it is classified as having right bundle branch block (RBBB) morphology. A QRS duration >0.14 seconds in RBBB tachycardias and >0.16 seconds in LBBB tachycardias favor VT [11]. This is due in part to the correlation between QRS duration and ventricular size and function. The duration of the QRS complex in VT is dependent on the distance between the tachycardia site and the His-Purkinje system, with wider QRS complexes resulting if the site is at the lateral wall and narrower complexes resulting if the site is in the basal septum. The value of QRS width as a diagnostic clue to tachycardia origin decreases significantly if the patient is taking antiarrhythmic medications that slow intraventricular conduction (eg, flecainide, propafenone), if hyperkalemia is present, and if ventricular pacing, particularly from the right ventricular apex, is occurring.

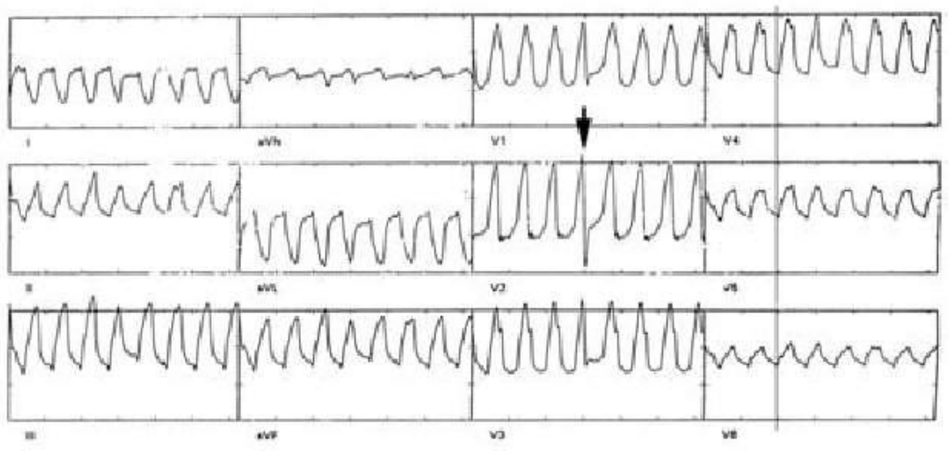

Fig 3. Wide QRS tachycardia with right bundle branch block morphology morphology. The presence of a fusion beat (arrow) indicates that artrioventricular dissociation is present. Also, the tachycardia has positive concordance in the precordial leads, which is suggestive for ventricular tachycardia. Since modern electrocardiogram machines acquire tracings simultaneously, it is useful to draw a plumb line (dashed line), which helps identify the initial portion of the QRS complex.

Frontal Plane QRS Axis

A right superior axis (–90° to –180°) is highly specific (0.97) for VT. An upright complex in aVR suggests a tachycardia arising from the right ventricular apex [11]. Left axis deviation (–60° to –90°) in the presence of a RBBB-type wide complex tachycardia is another specific (0.97) finding for VT. Other axis and QRS morphology combinations are not as useful for differentiating VT from supraventricular tachycardia with aberrant intraventricular conduction [11].

Precordial QRS Complex Morphology

Concordance. Concordance is present if all precordial QRS complexes are predominantly negative (negative concordance) or positive (positive concordance). In negative concordance, ventricular activation must be initiated from the apex, generally ruling out any type of aberrant intraventricular conduction [11]. Negative concordance associated with supraventricular tachycardia and aberrant conduction has been reported in a patient with pectus excavatum [12]. In positive concordance, ventricular activation must be arising from the posterior base of the left ventricle. Aberrant ventricular activation due to bundle branch block will not result in positive concordance since RBBB is associated with a deep wide S wave in V6 and LBBB produces a predominantly negative QRS complex in V1. Positive concordance will be present if ventricular activation occurs solely through a left-sided accessory pathway in a patient with Wolff-Parkinson-White conduction.

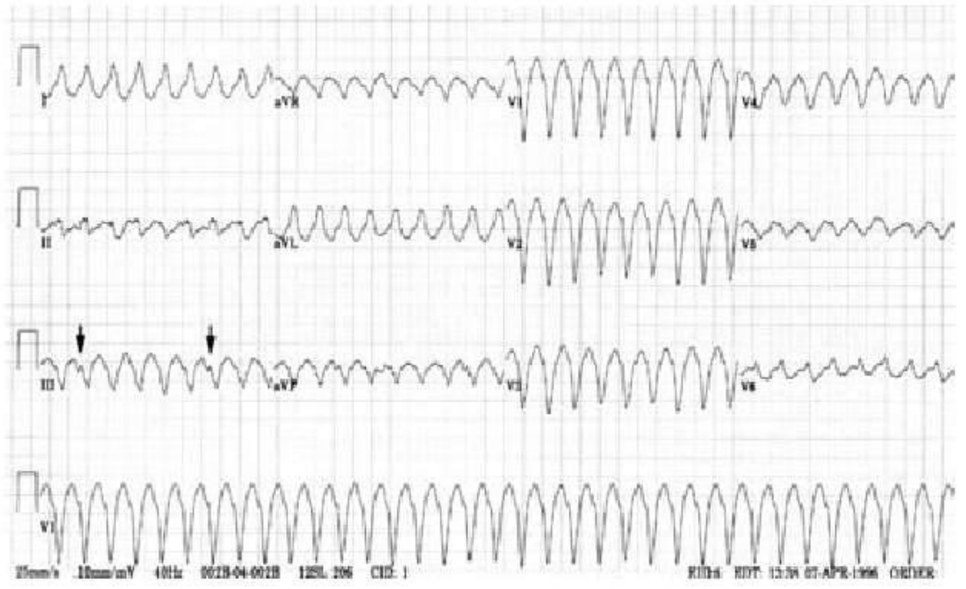

Fig 4. Wide QRS complex tachycardia with left bundle branch block morphology. The only morphologic clue to its origin is the absence of an RS complex in the precordial leads; the morphology of V6 is not definitive, illustrating the difficulty of using morphologic criteria for the diagnosis of ventricular tachycardia. The deflections due to P waves with artrioventricular dissociation (arrows) and the different QRS morphologies in lead V6 are diagnostic of ventricular tachycardia.

Absent RS and RS >100 seconds. In a prospective analysis of 554 wide complex tachycardias (384 ventricular and 170 supraventricular), Brugada and coworkers found that an RS complex was present in at least 1 precordial lead in all patients with supraventricular tachycardia; in contrast, however, 26% of VTs did not have an RS complex in any precordial lead (Figure 4) [13]. In tachycardias with an RS complex, the interval between the onset of the R wave and the nadir of the S wave in any precordial lead was >100 milliseconds in patients with VT (Figure 2).

Negative QRS complex in lead V1 (LBBB morphology). In general, the tachycardia site of VT with LBBB morphology is the interventricular septum in patients with heart disease and the right ventricle for patients with normal left ventricular function. An initial positive wave of greater than 0.03 seconds, slurring of the downstroke, or a greater than 0.07-second duration between the onset of the QRS complex and the nadir of the S wave are highly specific (1.00) findings for VT [14]. Unfortunately, these criteria for VT are dependent more on the presence of structural cardiac disease than specific electrocardiographic criteria. In a study of 36 patients with VT, including 20 patients with structural heart disease and 16 with normal hearts, the sensitivity of V1 criteria was 100% in patients with heart disease but only 50% in patients with normal hearts [15]. In addition, morphology criteria for VT

are less useful in patients with preexisting intraventricular conduction delays [16].

The QRS complex morphology in lead V6 can also be useful in patients with tachycardia with LBBB morphology. The presence of a Q wave in lead V6 is associated with VT [11].

Positive QRS complex in V1 (RBBB morphology). For patients with a predominantly positive QRS complex in V1, the presence of a qR, pure R, or RR′ complex makes VT more likely [11]. If the QRS complex has more than 1 peak, a higher first peak favors VT (Figure 3) while a higher second peak does not; if the second peak is broad, however, supraventricular tachycardia with RBBB is favored [17].

The absence of a deep S wave in V6 (R:S <1) makes VT more likely since RBBB is usually characterized by a deep S wave in the lateral precordial leads [11]. As emphasized by Griffith et al, if a typical pattern of aberrant conduction is not observed, the wide complex tachycardia should be considered to be VT [18].

Initiation and Termination of Tachycardia

The initiation of any tachycardia should be carefully evaluated. VT is almost always initiated by a premature ventricular depolarization. Although initiation of tachycardia with a premature atrial depolarization does not completely rule out VT, it certainly makes supraventricular tachycardia with aberrant intraventricular conduction more likely. The T wave of the preceding QRS complex should be evaluated and compared to that in sinus rhythm for any deformities (eg, notches) that might represent premature atrial activity.

The QRS complexes in tachycardia should be compared to those during sinus rhythm. A frontal plane QRS axis change of greater than 40° suggests VT [19]. In a patient with a baseline bundle branch block, a change in the QRS morphology or an increase in the QRS duration suggests VT [20], although in the latter instance, a rate-dependent increase in QRS duration must be considered.

Irregular Wide QRS Complex Tachycardias

Irregular wide QRS complex tachycardias generally have beat-to-beat changes in QRS morphology (polymorphic). Polymorphic VT can be associated with ischemia and, if sustained, almost always leads to hemodynamic collapse and ventricular fibrillation (Figure 5). A specific type of polymorphic VT is

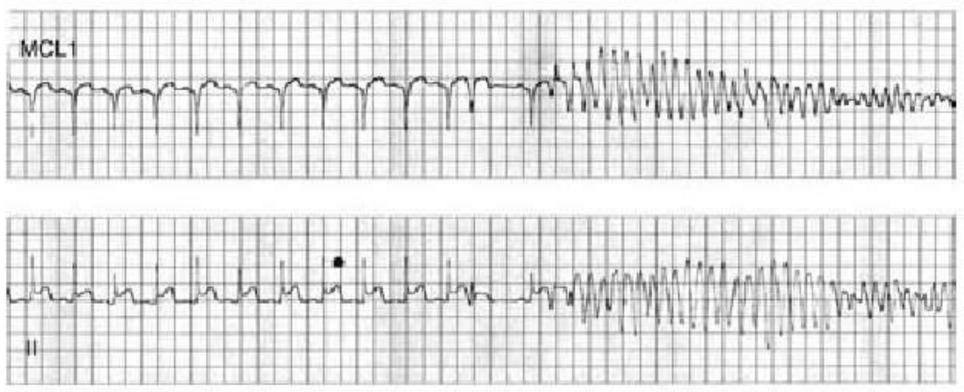

Fig 5. Polymorphic ventricular tachycardia due to ischemia, with ST segment elevation in lead II (bottom strip). Since the QT interval is normal, this rhythm is not considered to be torsade de pointes.
Courtesy of Tom Evans, MD.

torsade de pointes ("twisting of points"), associated with positive and negative QRS morphologies that appear to rotate around a fixed baseline. For the diagnosis of torsade de pointes, the baseline ECG must have a prolonged QT interval. Torsade de pointes can result from use of drugs that prolong the QT interval, such as antiarrhythmic drugs, and psychotropic drugs such as phenothiazines, thoridazine, and lithium; electrolyte disturbances such as hypokalemia and hypomagnesemia; or congenital long QT syndrome. Another cause of an irregular polymorphic tachycardia is atrial fibrillation with rapid activation of the ventricles via an accessory pathway, but in this case, the QT interval is normal and the pattern of torsade de pointes is not classical.

Irregular monomorphic wide complex tachycardias are generally atrial fibrillation with aberrant intraventricular conduction; very rarely, however, VT can also be associated with an irregular rate.

An important rhythm to be considered is atrial fibrillation with ventricular activation occurring over an accessory pathway in a patient with Wolff-Parkinson-White syndrome (Figure 6). Since the accessory pathway is extra-AV nodal and does not delay AV conduction, rapid atrial rhythms can be transmitted directly to ventricular tissue (Figures 1 and 6). This leads to a characteristic triad of ECG findings: irregular, very rapid, and wide QRS complex tachycardias, with delta waves on the QRS complexes signifying conduction over the accessory pathway (Figure 6).

Management of Wide Complex Tachycardias

Comprehensive review of the management of wide QRS complex tachycardias is beyond the scope of this article, but several points pertinent to ICU treatment are worth emphasizing (Table 1) [21-23]. First,

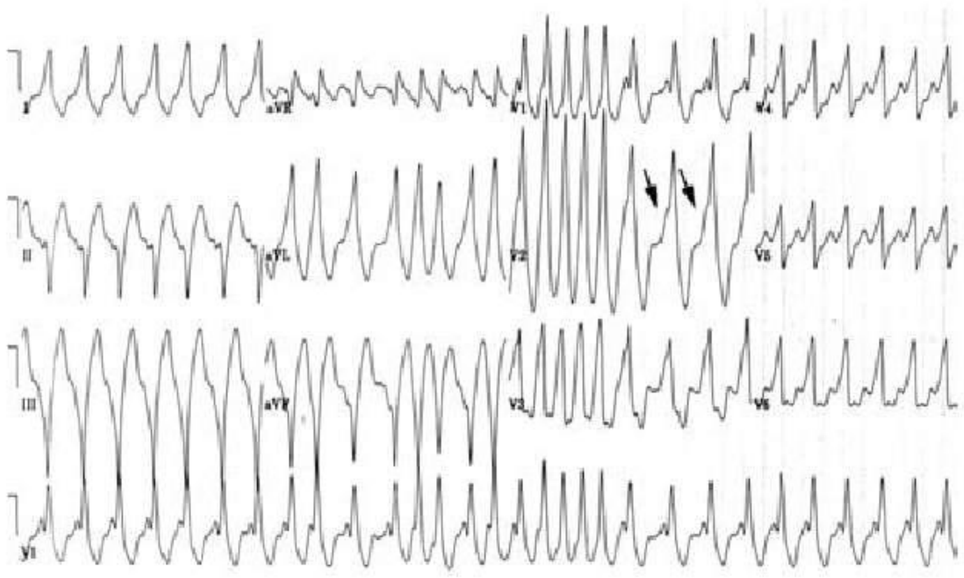

Fig 6. Atrial fibrillation with rapid activation of the ventricles via an accessory (extra-AV nodal) pathway, designated by the delta waves easily discerned in some, but not all, leads (arrows). Wide, irregular QRS complexes with a very rapid rate are present; some of the R-R intervals are <200 milliseconds (heart rate >300 beats/min), indicating a high risk for ventricular fibrillation.

the hemodynamic status of the patient should be considered. The hemodynamically unstable patient requires immediate electrical cardioversion. In the hemodynamically stable patient, intravenous adenosine can be considered if it is believed that the patient has supraventricular tachycardia with intraventricular aberrancy or if the specific type of arrhythmia is unclear [24]. Several case series have evaluated the use of intravenous adenosine in regular wide QRS complex tachycardias. Although generally safe, there are reported cases of ventricular fibrillation after adenosine infusion possibly due, at least in part, to the relative ventricular bradycardia after its use [24-26]. Adenosine will terminate supraventricular tachycardias that are dependent on conduction within the AV node (AV node reentrant tachycardia, accessory pathway-mediated orthodromic reciprocating tachycardia) and some atrial and VTs due to abnormal automaticity. In general, adenosine should not be used for irregular wide complex tachycardias. Its half-life is too short for clinically effective ventricular rate control, and its tendency to accelerate conduction within accessory pathways in patients with atrial fibrillation can lead to ventricular fibrillation (Table 1) [27].

If the patient with wide QRS complex tachycardia is stable, drug therapy with intravenous amiodarone or procainamide can be used (Table 1). Intravenous amiodarone is generally the most effective medication for the treatment of wide QRS complex tachycardias. Amiodarone will slow the ventricular rate or terminate most supraventricular tachycardias and also some VTs. Procainamide is effective in terminating 80% to 90% of monomorphic VTs, especially if associated with acute myocardial ischemia or infarction, but it requires a 20-minute infusion and may be associated with profound hypotension [21-23,28]. Lidocaine is generally ineffective for treating wide QRS complex tachycardia [28].

For patients with torsade de pointes VT, several specific treatment strategies should be considered. Once electrolyte abnormalities and any reversible cause for the arrhythmia have been excluded or treated, intravenous lidocaine and magnesium may be effective (Table 1) [29]. In cases refractory to pharmacologic therapy, temporary transvenous cardiac pacing with ventricular rates of up to 100 beats/min to shorten the QT interval is generally effective.

Regular Narrow Complex Tachycardias

It is clinically useful to distinguish between regular and irregular supraventricular tachycardias (Figure 7). The electrocardiographic approach to the diagnosis of wide QRS complex tachycardias is generally based on evaluation of the QRS complex. In contrast, because ventricular activation is normal in narrow QRS complex supraventricular tachycardias, ECG analysis of these arrhythmias focuses on identification of atrial activation (P waves) and analyzing the rate and morphology. In addition, it is useful to ascertain whether the tachycardia persists in the presence of AV block (AV node independent) or terminates, indicating reliance on conduction over the AV node for its perpetuation (AV node dependent).

P Wave Location and Morphology

Identification of P waves can be difficult. Electrocardiograms recorded during tachycardia and at baseline in normal rhythm should be compared and evaluated for any changes in the QRS complex, ST segment and T waves that might represent atrial activity (Figures 8 and 9).

Each type of tachycardia has a general location for P waves relative to the QRS complexes (Figure 7). For focal (ectopic) atrial tachycardias, the P wave is located in the usual position and has a normal or near-normal PR interval, although in the presence of rapid atrial rates, AV block can occur (Figures 7 and 9). In tachycardias involving the AV node or junction, the QRS complexes frequently obscure the P waves since the atria and ventricles are activated simultaneously (Figures 1 and 7); the P waves can also be seen in the terminal portions of the QRS complexes, particularly in lead V1. In accessory pathway-mediated tachycardias, the P waves are usually located within the ST segment

Table 1. Antiarrhythmics

Medication	Dosing	Adverse Effects
Procainamide Use: stable wide complex tachycardia	Loading: 15-17 mg/kg IV at 20-30 mg/min Maintenance: 1-4 mg/min	Increased antinuclear antibodies (50%), SLE, wide PR or QRS, hypotension, asthenia, apraxia, depression, chills, abdominal pain, diarrhea, nausea, vomiting, bitter taste, myopathy, rash Warning: May produce life-threatening hematologic disorders (leukopenia, agranulocytosis (0.5%)
Amiodarone Use: wide complex tachycardia; slows ventricular rate in selected patients with narrow complex tachycardia	Initial: 5 mg/kg IV over 20-30 min Maintenance: 1 g infusion over 24 h (1 mg/min for 6 h then 0.5 mg/min for 18 h)	Elevated liver enzymes (3% to 20%) Nausea, vomiting, constipation, anorexia (25%) Dermatologic reactions (15%) Malaise, fatigue, tremor, peripheral neuropathy, abnormal gait, paresthesias (20%-40%) Pulmonary fibrosis (10%-70%) Hypotension with IV (16%) Arrhythmias (2%-5%) Hypo-/hyperthyroidism (2%) Flushing, edema (1%-3%) CHF (3%) Corneal micro deposits
Lidocaine Use: torsade de pointes; ventricular arrhythmias associated with ischemia	1-1.5 mg/kg IV repeated in 5 min Max: 3 mg/kg	Dizziness, nausea, drowsiness, speech disturbance, perioral numbness, muscle twitching, psychosis, seizures, sinus arrest, complete AV block Cautions: toxicity more common in elderly patients, liver failure, and heart failure
Magnesium Use: torsades de pointes	2 g IV bolus followed by an additional 2 g if arrhythmia persists	Cardiovascular collapse, respiratory paralysis, hypothermia, pulmonary edema, depressed reflexes, hypotension, flushing, drowsiness, diaphoresis, hypocalcemia, hypophosphatemia, hyperkalemia, visual changes
Adenosine Use: terminate AV node dependent tachycardias; diagnostic maneuver for evaluating tachycardia	6-12 mg peripherally and 3-6 mg centrally given as IV rapid push	Flushing (18%-44%) Chest pain (7%-40%) Dyspnea (12%-20%) Headache (2%-18%) Light-headedness (2%-12%) Throat or jaw discomfort (15%) Nausea (3%) Bradycardia, bronchoconstriction, arrhythmia, AV block
Diltiazem Use: decrease ventricular rate in patients with atrial tachycardia (atrial fibrillation, atrial flutter, focal atrial tachycardia, MAT)	Initial: 15-20 mg bolus Optional second bolus of 20-25 mg Maintenance drip: 5-15 mg/h	Hypotension (3%-4%) Dizziness, headache (1%-5%) Nausea (3%) AV block, bradycardia, syncope, gingival hyperplasia, and peripheral edema
Ibutilide Use: terminate atrial fibrillation/flutter	Greater than 60 kg: 1 mg IV push; repeat after 10 min as needed Less than 60 kg: 0.01 mg per kilogram IV push; repeat after 10 min as needed	Headache (4%) Bradycardia, hypertension, hypotension, palpitations (2%) QT prolongation, nausea (<2%) Heart failure, heart block, torsade de pointes, potentially lethal arrhythmias

SLE = systemic lupus erythematosus; CHF = congestive heart failure; AV = atrioventricular; MAT = multifocal atrial tachycardia.

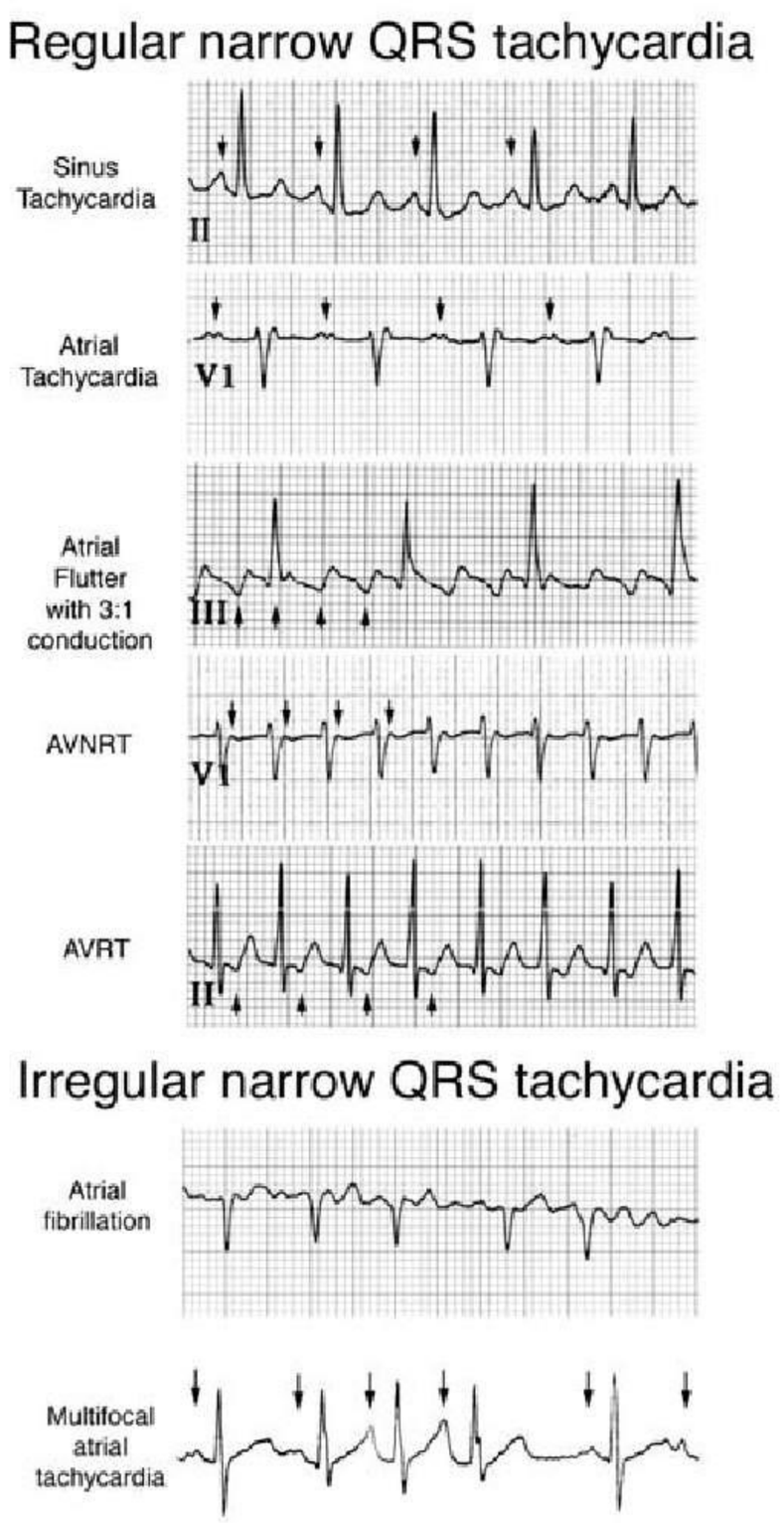

Fig 7. Electrocardiographic evaluation of narrow complex tachycardias focuses on identification of atrial activity (arrows). (Top) For regular QRS tachycardias, atrial activity can be identified in specific leads depending on the type of tachycardia. (Bottom) For irregular QRS tachycardias, multifocal atrial tachycardia is characterized by discrete P waves of varying morphology with intervening isoelectric periods, while atrial fibrillation is characterized by more constant, nondiscrete atrial activity and absence of an isoelectric baseline. AVNRT = atrioventricular nodal reentry tachycardia; AVRT = atrioventricular reentry tachycardia.

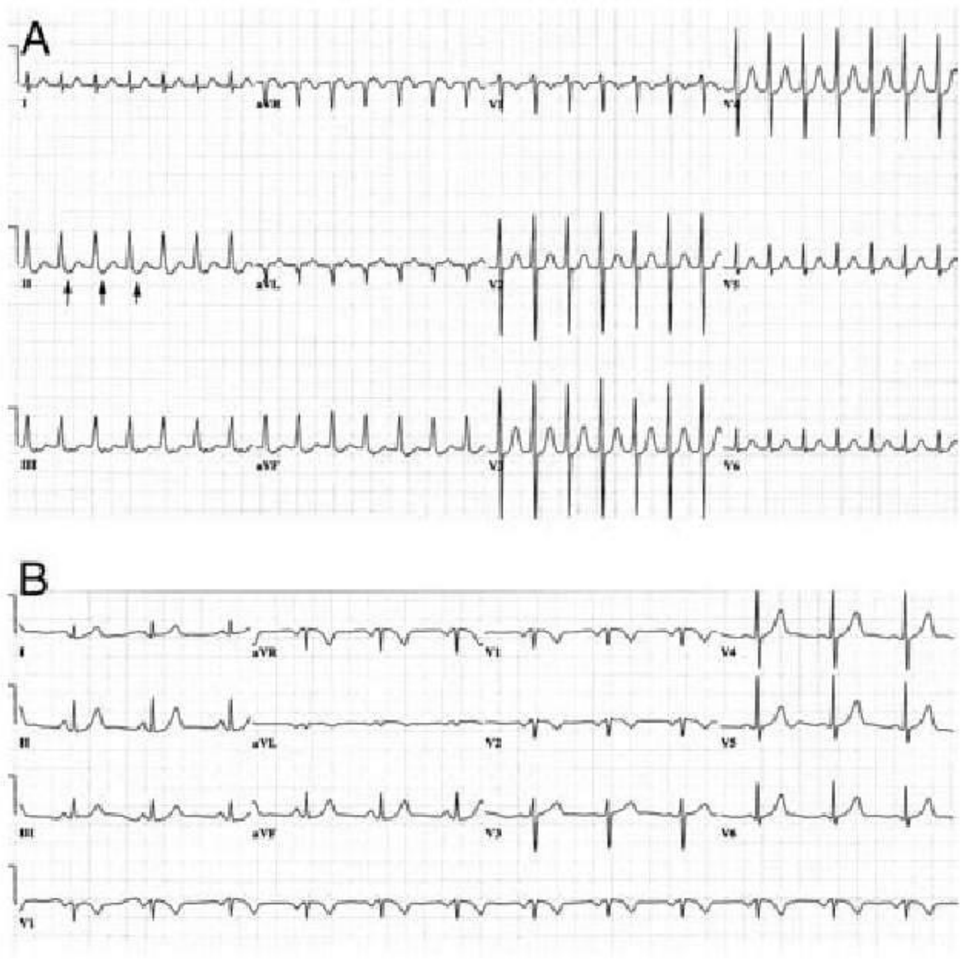

Fig 8. Electrocardiogram (ECG) during tachycardia and at baseline in a patient with orthodromic atrioventricular (AV) reentrant tachycardia (A), in which the ventricles are activated over the normal AV node–His–Purkinje system. Inverted P waves indicating retrograde atrial activation are located in the ST segments of leads II, III, and aVF, best seen in lead II. Such QRST deformations by superimposed P waves can mimic ST segment abnormalities: depression or elevation, or intraventricular conduction delays. Evaluation of the baseline ECG (B) confirms that the deflections observed in the ST segment represented atrial depolarization.

since retrograde activation over the accessory pathway is more rapid than ventricular activation via the AV node (Figures 1, 7, and 8). Electrophysiologists often describe supraventricular tachycardias as being short R-P (most commonly AV nodal atrial tachycardias and orthodromic AV reentry tachycardias) or long R-P (most commonly atrial tachycardias) based on the location of the P waves relative to the preceding QRS complexes (Figure 10).

Once identified, the P wave morphology can be evaluated. Atrial activation is generally classified as high-low if the P waves are upright in the inferior leads or low-high if the P waves are inverted in the inferior leads. Inverted P waves suggest that atrial activation is initiated from the junction or the mitral and tricuspid annulus, while upright P waves suggest that atrial activation is initiated from a superior location (Figures 8 and 9).

AV Node–Dependent Versus AV Node–Independent Supraventricular Tachycardias

Atrial tachycardias are not dependent on AV node conduction for their perpetuation, and in atrial flutter and rapid atrial tachycardias, more P waves than QRS complexes are usually observed. Although the ventricular rate will decrease if AV block develops or is induced (by, eg, vagal maneuvers or AV blocking drugs), the atrial rate and rhythm will continue unchanged. In contrast, AV nodal reentrant and accessory pathway-mediated tachycardias are AV node dependent; thus, the tachycardia terminates if AV block develops.

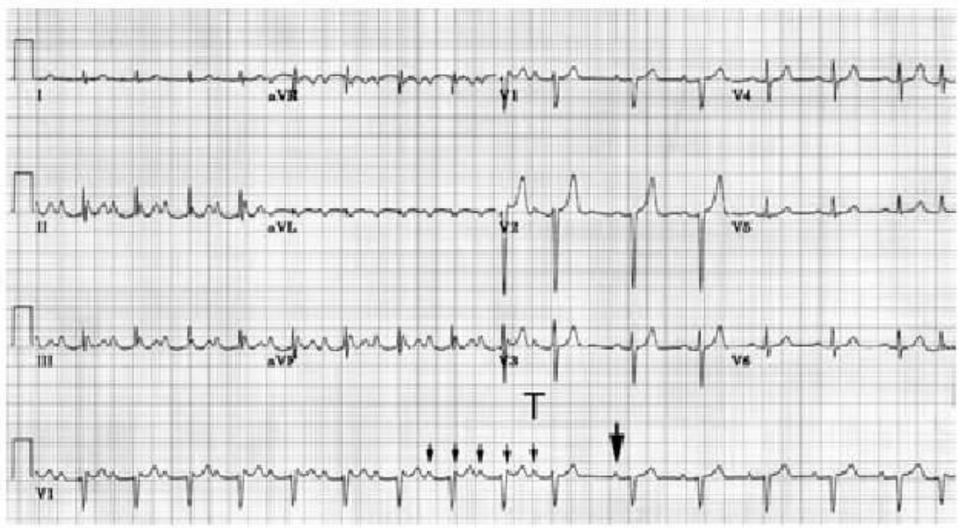

Fig 9. Electrocardiogram (ECG) illustrates intermittent atrial tachycardia and its termination (T). Comparison of the ECG during tachycardia and during sinus rhythm shows the atrial activation in the terminal portion of the QRS complexes (arrows). The patient has an automatic atrial tachycardia with 2:1 atrioventricular block. The atrial tachycardia site is near the sinus node; this is the reason that the P wave morphologies are similar during tachycardia (small arrows) and sinus rhythm (large arrow).

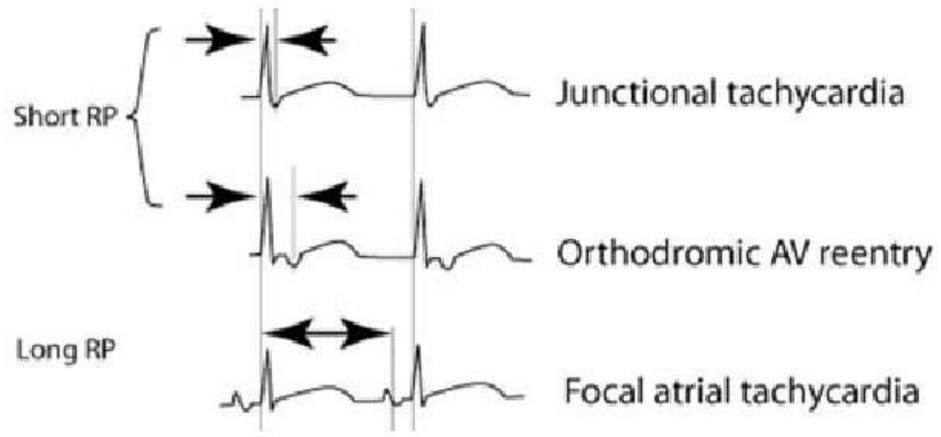

Fig 10. Schematic diagram illustrating the usual location of P waves for specific tachycardias. In tachycardias involving the atrioventricular (AV) node or junction, the P waves are buried within and obscured by the QRS complexes or are visible in the terminal portions of the QRS complexes (see Figure 7). For accessory pathway-mediated orthodromic AV reentrant tachycardias, the P waves are located in the ST segments. For atrial tachycardias, the P waves are usually located in the normal position (longer RP interval compared to the PR interval) unless AV block is present.

Irregular Narrow Complex Tachycardia

Atrial Fibrillation

Although the precise mechanisms for atrial fibrillation are complex and patient specific, the atrial rhythm is rapid and irregular. Since some portion of the atria is always being activated, the ECG is characterized by low-amplitude fibrillatory waves without an isoelectric period. Atrial fibrillation can develop in 10% to 65% of patients after cardiac surgery, usually on the second or third postoperative day, unless specific therapy such as amiodarone or β-blocking agents is provided in the perioperative period [30-32]. Atrial fibrillation is commonly observed after valve surgery, particularly in older patients. In noncardiac intensive care units, the incidence of atrial fibrillation is lower (2%-10%) and appears to be associated with blunt thoracic trauma and septic shock [31].

Multifocal Atrial Tachycardia

In multifocal atrial tachycardia, several different foci activate the atria. The specific definition of multifocal atrial tachycardia is an atrial rate >100 beats/min, at least 3 morphologically distinct P waves, and irregular P-P (and generally also P-R) intervals (Figure 7) [32]. The different rates of atrial activation lead to irregular ventricular rates, which can be quite rapid. In contrast to atrial fibrillation, there are isoelectric periods in which no atrial activation occurs. Multifocal atrial tachycardia is usually observed in older patients with chronic obstructive lung disease [33]. Multifocal atrial tachycardia is also associated with hypoxemia, hypokalemia, hypomagnesemia, congestive heart failure, and pulmonary embolus [32,33]. It is often confused with atrial fibrillation; the distinction is important since management strategies differ considerably.

Management of Narrow QRS Complex Tachycardias

Rapid administration of adenosine (6-12 mg peripherally and 3-6 mg centrally) is the standard treatment of regular narrow QRS complex tachycardia in which the diagnosis is in doubt or if an AV node–dependent tachycardia is suspected (Table 1) [21-23]. AV node–dependent tachycardias such as AV nodal reentry tachycardia and orthodromic AV reentrant tachycardia will terminate with adenosine due to transient AV block. The atrial rhythm in AV node–independent tachycardias will continue despite a transient period of slowing of ventricular rate. However, it should also be emphasized that most focal atrial tachycardias will terminate with adenosine because of direct effects on atrial tissue and potentially mislead the clinician into believing that an AV node–dependent tachycardia is present [34].

In the patient with hemodynamic compromise, synchronized direct current cardioversion is effective for terminating all forms of supraventricular tachycardia. However, cardioversion appears to have less clinical utility in the critically ill patient but hemodynamically stable patient. In a study of 37 patients who underwent a strategy of attempted

immediate cardioversion for new-onset supraventricular arrhythmias, only 16% remained in sinus rhythm after 24 hours, due both to poor initial efficacy and also to the development of recurrent arrhythmias [35]. For hemodynamically stable ICU patients with supraventricular tachycardias, it is generally more useful to employ pharmacologic therapy to slow the ventricular rate by slowing AV node conduction. Intravenous diltiazem is frequently used since it is easy to administer and can be titrated (Table 1). In patients with hemodynamic compromise and rapid ventricular rates, intravenous amiodarone is also effective for controlling heart rate [36]. A recently completed randomized prospective study has found that magnesium sulfate may be comparable to intravenous diltiazem for the treatment of paroxysmal atrial fibrillation (Table 1) [37].

Once the ventricular rate has been slowed with AV node blocking agents, if clinically appropriate, the patient can be given treatment to terminate the atrial arrhythmia. Intravenous amiodarone and ibutilide can be used to terminate atrial fibrillation and atrial flutter; ibutilide is, however, not recommended due to the usual clinical instability of the ICU patient. Intravenous amiodarone can be useful for suppressing recurrent episodes of atrial arrhythmias.

Bradycardias

Slow heart rates can occur because of abnormally slow automaticity or AV block. Significant bradycardia other than brief episodes is uncommon in ICU patients, accounting for approximately 10% of arrhythmias observed in this setting [1]. One specific cause of bradycardia that is frequently seen in the intensive care unit is a slow wide QRS rhythm due to hyperkalemia. The classic arrhythmia in severe hyperkalemia is the presence of sino-ventricular rhythm. In this situation, the sinus node continues to lead to ventricular activation, but P waves are not seen because of loss of atrial depolarization (Figure 11).

Sinus pauses (sinus arrest and sinoatrial block) and sinus bradycardia may be physiologic. Atrial bradycardia is more common and pronounced during sleep; heart rates of 30 to 35 beats/min and pauses exceeding 2.5 seconds can be observed. Sinus bradycardia and pauses can also be observed with medications that suppress automaticity, such as β-blockers, calcium channel blockers, and clonidine. Specific causes of sinus bradycardia observed in the intensive care unit also include sleep apnea during apneic periods, increased intracranial pressure, and enhanced vagal tone from endotracheal suctioning or emesis.

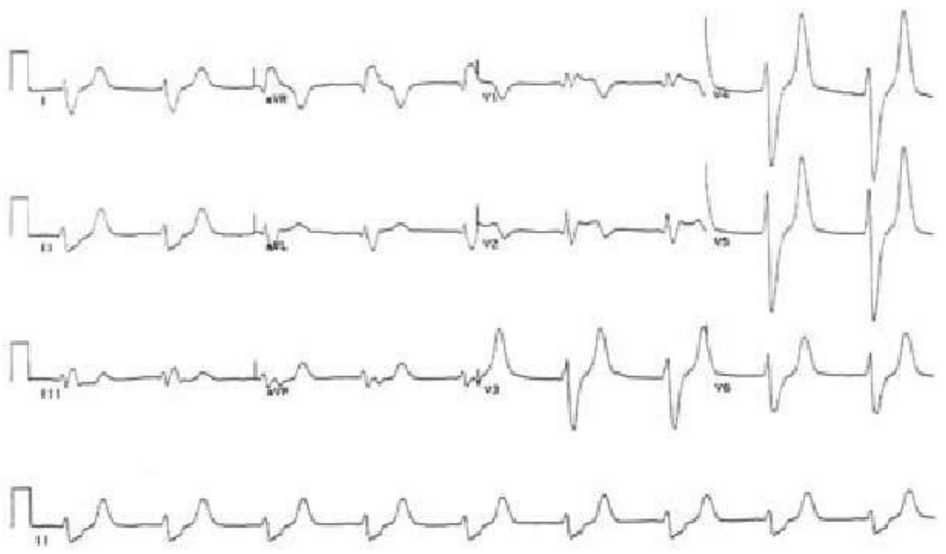

Fig 11. Electrocardiogram in a patient with hyperkalemia. A slow wide QRS complex rhythm without P waves is called sino-ventricular rhythm and is characteristic of severe hyperkalemia.

AV block can be observed in the ICU setting after cardiac surgery or if the patient is receiving medications such as β-blockers, calcium channel blockers, amiodarone, or lithium. AV block due to these agents can be due to block within the AV node or His bundle; bundle branch block causing AV block is decidedly unusual. AV block is commonly observed in hypervagotonic states such as emesis, endotracheal sunctioning, and endoscopy; cardiac pacing is not indicated.

Treatment of Bradycardia

Only symptomatic bradycardia should be treated. If the patient is hemodynamically unstable, intravenous atropine can be used but is often ineffective in the ICU setting since increased sympathetic tone is so frequently present. Patients with persistent and hemodynamically significant bradycardia may require temporary pacing. Temporary pacing is most easily accomplished by the transcutaneous route and is accomplished by using large surface area skin electrodes placed in the anterior-posterior position (cathode over the cardiac apex and the anode placed between the right scapula and spine) or the anterior-anterior position (cathode over the apex and anode on the right chest). Transcutaneous pacing is effective (>90%) but cannot be used for long periods of time because of pain, muscle stimulation, and loss of capture due to impedance changes. Transvenous pacing is generally required if continuous pacing for more than 20 to 30 minutes is required [38].

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