**Chapter 14
Cardiac dysrhythmias**

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**Introduction**

Emergency medical services physicians often use the same approach in the field and the hospital to provide patient care, even though the goals in each area differ. The care of patients with dysrhythmias before hospital arrival focuses on treating all life-threatening or imminently life-threatening rhythm changes within minutes. In the emergency department (ED) and in the hospital, the same need exists but more time is available to identify other non-lethal rhythms and deliver definitive long-term treatment.

This chapter discusses a pragmatic method of providing medical oversight for non-arrest dysrhythmias. The most important field observations and actions will be highlighted to help simplify the approach when giving direct medical oversight, creating written protocols, or providing direct patient care. We offer a “low-tech” approach to the problems, emphasizing simple tools including a brief history, physical examination, and standard 3- or 12-lead field ECG. Similarly, we focus on interventions that are effective and easily provided in the out-of-hospital setting. In general, the approach offered is consistent with the 2010 American Heart Association (AHA) Advanced Cardiac Life Support (ACLS) guidelines, although we highlight areas where simplified or alternative approaches exist.

**Evaluation**

Three basic sources of information are available during the assessment of field dysrhythmias: patient history, physical examination, and the ECG. Rarely will any one of these suffice in guiding treatment. Rather, all three considered together guide care [1,2]. Four steps can be used to manage patients with dysrhythmias in the field. Treatment decisions often can be made before completing all steps, allowing an economy of effort.

**Step 1: identify symptoms and how they relate to the rhythm**

Two groups of patients present with dysrhythmias: asymptomatic patients with incidental rhythm changes and patients with symptomatic rhythm changes. Incidental dysrhythmias may relate to the symptoms, but are the result and not the cause of another problem, and they do not worsen the immediate outcome. Patients with incidental dysrhythmias or who are asymptomatic rarely require field rhythm-directed treatment. Those with incidental dysrhythmias typically require treatment of any underlying acute condition (e.g. analgesia for pain or fluids for hypovolemia).

A 67-year-old male patient with a history of “extra heart beats” transported for an isolated ankle injury displays a sinus tachycardia (from pain) and occasional premature ventricular complexes, but no other symptoms or abnormalities on physical examination. He requires splinting and analgesia, not antidysrhythmics. This should not be confused with dysrhythmias with symptoms, such as tachycardia or bradycardia associated with chest pain, weakness, breathing difficulties, or syncope.

**Step 2: identify stable and unstable patients**

Because asymptomatic or incidental dysrhythmias usually require no direct treatment, the prehospital focus shifts to those dysrhythmias associated with symptoms. These patients are classified based on the severity of symptoms as either stable or unstable. Although many patients have symptoms attributable to the change from a “normal” rhythm, most tolerate these well and are stable. However, unstable patients are likely to suffer harm or deteriorate. Providers and EMS physicians must identify these unstable patients and rapidly intervene.

Unstable patients have signs and symptoms of inadequate end-organ perfusion due to the rhythm disturbance [2]. A few brief historical questions and physical examination steps must be rapidly completed to identify these patients early in their evaluation.

* Hypotension – often arbitrarily defined as a systolic blood pressure below 90 mmHg, though any departure of more than 15% from a known baseline may be functional hypotension.
* Cardiac dysfunction – seen as chest pain, shortness of breath, or rales (signifying inadequate myocardial perfusion or function).
* Altered consciousness – from mild agitation or somnolence to obtundation or coma (signifying central nervous system [CNS] hypoperfusion).

Delayed capillary refill and lowered skin temperature can indicate poor perfusion; the subjective nature of these observations and multiple other potential causes limit their use in the field.

Assessing instability is usually a continuum, not an “all-or-nothing” phenomenon. Either a single severe sign or symptom or multiple mild findings is diagnostic of an unstable rhythm. A single mildly abnormal finding suggests “borderline” stability. The blood pressure is the simplest method of assessing circulatory adequacy, but it alone may be insufficient in accurately classifying patients. A patient with a systolic blood pressure of 60 mmHg is always unstable. Another patient with a blood pressure of 90 mmHg systolic, rales, and a depressed sensorium is also unstable. If awake and with no rales, chest pain, or other symptoms, the patient with a systolic blood pressure of 90 mmHg occupies a borderline position due to the singular mild finding. Similarly, agitation suggests mild CNS hypoperfusion and borderline stability, whereas coma is associated with more profound derangement and instability.

In the absence of clear evidence of instability, each patient can receive a more complete evaluation, although the total prehospital time interval should not be prolonged. Unstable patients need rapid therapy, usually with electrical interventions such as external countershock or pacing. Symptomatic but stable or borderline unstable patients can be initially treated with pharmacological agents, with electrical devices nearby in case of deterioration. The more extreme the sign or symptom of instability (e.g. coma versus mild anxiety), the more intensive the initial treatment should be.

**Step 3: classify the electrocardiogram findings**

After assessing stability, the field providers need to categorize the ECG. Using a traditional approach of separating dysrhythmias into dozens of categories is tempting. In the field evaluation, a simpler scheme should be used based on the assessment of stability and three ECG features: QRS complex rate, regularity, and duration.

Electrocardiogram interpretation is performed in two ways: by medical oversight physicians receiving transmitted tracings, or independently by the field personnel. Transmitted tracings are occasionally hampered by technical problems which can obscure salient features. Field providers can learn the basics of ECG interpretation to identify common and lethal rhythms. However, some errors are common. For example, misclassification of QRS duration and rate occurs in up to 20–30% of tachycardias [3]. Protocols and medical oversight decisions must assume that the potential for misclassification exists and attempt to minimize attendant adverse outcomes. The strategies outlined herein apply to both field and transmitted interpretation. In all steps, ECG interpretation must be done from a printed strip and not “guesstimated” from the monitor screen.

**Rate**

Initially, the rate should be classified as fast (>120/minute), slow (<60/minute), or normal/near normal (60–120/minute) based on the frequency of QRS complexes over 6 seconds multiplied by 10. After the estimation of rate, sinus P-waves should be sought in those patients with normal or fast rates. Sinus P-waves always precede the QRS complexes and have a consistent appearance and relationship (i.e. distance) to the QRS complexes.

As a simple rule, all unstable patients with non-sinus fast rhythms (no discernible P-waves and QRS rate >120/ minute) deserve immediate synchronized countershock with 100 J. Often, lower energy levels can convert specific rhythms, such as supraventricular tachycardia (SVT) or atrial flutter, but little benefit is gained by attempting to make fine distinctions in these unstable patients. Although changes in heart rate that fall into the normal range can cause symptoms, these are usually of little importance in the field management.

Biphasic waveform defibrillators are increasingly common among EMS services. In general, lower energy biphasic waveform shocks are equally or more effective than monophasic shocks [4]. However, no outcome benefit to biphasic waveforms has yet been demonstrated [5]. In addition, the ideal energy for first-shock biphasic waveform defibrillation is uncertain [4]. The defibrillator manufacturer’s recommended energy levels for cardioversion and defibrillation should be used.

Patients with slow dysrhythmias only require classification of their stability. All other details (e.g. P-wave characteristics, type I or II second-degree block, junctional versus ventricular escape) add little value in prehospital management. Slow stable dysrhythmias need no intervention besides continued monitoring for deterioration. Slow unstable dysrhythmias require external pacing (preferred) or atropine (0.5–1 mg IV in adults, repeated up to 2–3 mg total). Transcutaneous pacing is best started as early as possible to maximize the potential for mechanical or clinical capture and restoration of perfusion [6,7]. Also, do not delay pacing in unstable patients to administer atropine. Conversely, concerns of clinical deterioration after atropine are unwarranted when the correct dose is given to those with symptomatic bradycardia, though there may be no response.

Internal, implanted pacemakers should prevent bradycardias, but they may malfunction. When a patient has pacer spikes on the ECG and is still bradycardic, the pacemaker is not working properly and the patient should be treated in the same fashion previously described with atropine or external pacing. The pacer pads should be kept 10 cm or more away from the internal pacemaker pouch. Trying to evaluate the pacemaker in the field is impossible and should await hospital evaluation (see Volume 1, [Chapter 15](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c15.xhtml)).

Bradycardias resulting from beta-blocker or calcium channel blocker overdoses may be refractory to atropine. In these cases, glucagon (1–3 mg IV) may improve the heart rate. Again, drug administration should not delay transcutaneous pacing.

Previously, isoproterenol was a second-line therapy for atropine-resistant bradycardias. With the availability of external pacemakers in the field and the poor clinical effectiveness of isoproterenol, this treatment is not currently recommended. In adults, a pressor medication (e.g. dopamine) infusion and a fluid bolus should be administered if transcutaneous pacing has normalized the heart rate but hypotension persists.

**Regularity and duration**

In contrast to bradycardia, if the ventricular rate is fast, the regularity and duration of the QRS complexes should be assessed. Regularity is divided into two categories: mostly or completely regular, and chaotic (i.e. “irregularly irregular” without any pattern). Chaotic rhythms are usually due to atrial fibrillation, irrespective of the appearance of the baseline or QRS duration. Other less common causes include multifocal atrial tachycardia and frequent extrasystoles (i.e. atrial, ventricular, or junctional).

To simplify the process of measuring duration and assessing regularity, EMS personnel should run an ECG strip. From this, they or the medical oversight physician can measure in “small boxes” how wide the QRS duration is and look for irregularity. Each small box represents 0.04 seconds at normal paper speed. Having providers seek out “How many small boxes wide is the QRS complex?” will limit mathematic or conversion errors. Similarly, evaluating printed strips also helps detect irregularity, which may be difficult to appreciate on a monitor screen if the ventricular rate is greater than 150/minute. In these cases, close tracking on a 6-second ECG strip may help detect chaos and identify atrial fibrillation.

Those rhythms with a QRS duration of less than three small boxes (0.12 seconds) are narrow-complex dysrhythmias. Conversely, any rhythm with a QRS duration of greater than three small boxes is a wide-complex dysrhythmia. Nearly all narrow complex rhythms originate from atrial or nodal (i.e. supraventricular) sources. Wide complex rhythms can originate from a ventricular or a supraventricular source. In the latter situation, some abnormality in ventricular conduction is responsible for the prolonged QRS duration. In the field, attempts to separate the myriad causes of wide-complex tachydysrhythmias (WCTs) rarely alter therapy and are unnecessary. Treatment should be based on the clinical stability of the patient, basic history, and the simple ECG characteristics previously defined.

***Unstable tachydysrhythmias***

Aside from sinus tachycardia, all unstable patients with a WCT or a narrow-complex tachydysrhythmia (NCT) deserve countershock(s), irrespective of the exact source, ventricular or supraventricular. The QRS duration will help dictate care after countershock, but does not fundamentally drive the initial care in unstable patients with a tachydysrhythmia.

The initial energy level used to treat tachycardias is based on the QRS pattern. If the QRS pattern is regular or nearly regular in any unstable patient with a tachydysrhythmia and a palpable pulse, synchronized cardioversion with 100 J should be used, followed by step-wise energy increases to 200 J with a biphasic device or 360 J with a monophasic device, if necessary. Some rhythms may require less energy, but attempts to carefully titrate this life-saving therapy in unstable patients is of little practical benefit. Synchronized countershock is recommended to avoid post-countershock ventricular fibrillation (VF). However, sensing problems often make reliable identification of the QRS complex needed for synchronization impossible. We recommend an unsynchronized shock promptly if any sensing problem occurs. Any patient without pulses and/or an irregular tachydysrhythmia should be immediately given a high-energy unsynchronized countershock.

Patients with internal pacemakers or automatic implantable cardioverter defibrillators (AICDs) are still at risk of cardiac dysrhythmias. Although meant to cardiovert dysrhythmias, AICDs do not always convert these rhythms, and sometimes these devices deliver shocks inappropriately. If a patient has an unstable tachydysrhythmia and the AICD is not firing or is ineffective, externally cardiovert as previously recommended, with pads in the anterior-posterior configuration and 10 cm away from the internal device pouch. Postconversion care with medical therapy will be unaffected.

If an AICD is repeatedly firing absent a ventricular dysrhythmia (wide complexes), a magnet over the device may inactivate the shock mechanism, simplifying patient care and improving patient comfort (see Volume 1, [Chapter 15](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c15.xhtml)). However, given the rarity of this event, the EMS director should weigh deployment of magnets and training for all providers versus likely benefits. Prompt transfer to the ED is wise in many settings.

If countershock fails in an unstable patient with a WCT, give either amiodarone (5 mg/kg) or lidocaine (1–2 mg/kg) as a bolus and repeat the countershock. The ALIVE trial [8] and recent AHA guidelines [1] recommend amiodarone as the first-line agent in unstable and especially pulseless WCT. Lidocaine is still the easiest to deliver quickly, but is considered a second-line agent due to variable success in terminating ventricular tachycardia (VT) [1].

If the QRS complexes are chaotic, the most common diagnosis is atrial fibrillation. When chaos and a QRS duration of more than three small boxes appear together, atrial fibrillation with altered conduction is the diagnosis. All unstable fast chaotic rhythms should be cardioverted with 50–100 J unsynchronized initially, and titrated up as needed. No post-countershock medications are needed.

One practical point: if regularity versus irregularity cannot be established during assessment of a patient with an unstable WCT or NCT, 100 J is an appropriate starting energy level for countershock. Similarly, if simplicity of treatment protocols is sought, 100 J is reasonable for all unstable non-sinus tachycardias, because the extra energy delivered to the rapid atrial fibrillation patient is unlikely to cause harm or worsen discomfort compared to 50 J.

**Step 4: focus actions to evaluate stable but symptomatic and borderline patients**

Up to this point, little specific history and only a few basic physical examination and ECG reading skills have been required. This is intentional, so as not to “clutter” the field evaluation of those who need it the most (i.e. the unstable patient) or do not need it at all (i.e. the asymptomatic patient). The remaining patients are those with symptoms, albeit none clearly identifying instability. Here, a few questions and actions can help to deliver the appropriate prehospital care.

**History**

The field teams should focus on cardiac-related previous problems in stable patients. For example, a patient who presents with new-onset WCT with a history of previous myocardial infarction is much more likely to have ventricular tachycardia than a supraventricular rhythm with abnormal conduction. Similarly, one with a history of a previous dysrhythmia who presents with similar symptoms again is likely to have recurrence rather than a new dysrhythmia. Neither of these clinical rules is infallible, but this information can help guide therapy. Other points are also helpful. For instance, a patient with a history of poorly controlled hypertension presenting with a lowered but “normal” blood pressure suggests a dramatic change, prompting more intensive treatment.

History can influence the dosing of field agents. Subjects with liver or heart failure, and those aged 65 years and older, should receive lower lidocaine infusions or follow-up boluses. Those patients with renal failure are at risk for hyperkalemia and rhythm changes. The current medications can provide a clue to any previous conditions or guide field drug therapy. A patient treated with digoxin or a beta-blocker plus warfarin for palpitations may have atrial fibrillation. Finally, although rare, a brief search for drug allergies or intolerances (“Has any heart drug been bad for you?”) may help avoid a complication. The key is to take a focused history, looking for information regarding heart disease and other specific conditions.

**Physical examination**

In addition to a search for signs of instability, some manipulations can help when assessing and managing tachycardias. Specifically, actions that alter atrioventricular node conduction (“vagal maneuvers”) can help terminate or uncover a specific dysrhythmia [2,9]. In a patient less than 50 years old, carotid body massage can be attempted. This procedure is often restricted or prohibited in the field because of poorly documented concerns about embolization. The Valsalva action can be used with massage in young patients or as the sole maneuver in those over 50 years old. Other maneuvers, including ocular and rectal massage, ice packs or cold-water dunking, and rapid inflation of pneumatic antishock garments, are not recommended.

**Stable narrow-complex tachydysrhythmias**

In patients who are symptomatic but stable or who have one borderline symptom of instability (e.g. dizzy or anxious with a low blood pressure), certain actions are indicated. Patients with a regular NCT between 120 and 140 per minute are likely to have a sinus tachycardia and require no antidysrhythmic treatment. Stable patients with a regular NCT at a rate of 140 per minute or greater should have vagal stimulating maneuvers performed to terminate the rhythm. Sometimes, this maneuver uncovers sinus P-waves, clarifying the sinus or atrial etiology. When P-waves are seen, treatment is directed at the cause, not the rhythm.

Those with minor symptoms (e.g. isolated subjective dizziness or palpitations) do not require field treatment beyond vagal maneuvers. For those with more prominent symptoms during a regular NCT at 140 per minute or greater, give adenosine (6–12 mg as a rapid IV bolus followed with a flush) [1–3,8]. The smaller initial dose (6 mg) is effective about 60% of the time, and it should be repeated within 2 minutes at the higher dose if no effect is seen. If adenosine causes slowing followed by a return to tachycardia, repeat or larger doses will not help. The cause is a non-reentrant source, often an atrial rhythm, possibly atrial tachycardia, fibrillation, or flutter.

Adenosine is effective in 85–90% of patients with regular NCT. The drug has a duration of effect of 20 seconds or less, and recurrence of an NCT may occur in 10–58% of cases. It is common for patients to complain of transient chest pain, flushing, or dyspnea during adenosine treatment. Some patients may experience bradycardia or asystole after adenosine. Usually, this lasts only seconds, but it may require temporary external pacing if prolonged. Contrary to popular belief, adenosine can occasionally terminate VT, although the majority of such patients are unaffected [10].

Verapamil (2.5–5 mg IV initially followed by 5–10 mg in 15 minutes if unsuccessful) and diltiazem (0.15 mg/kg initially, followed by 0.20–0.25 mg/kg in 15 minutes if unsuccessful) will terminate 85–90% of regular NCT [11,12]. However, both can cause hypotension and congestive heart failure, though diltiazem is alleged to have slightly lower rates of this in equipotent doses. Because of these disadvantages, many prefer to use adenosine in the field. Whenever giving adenosine, verapamil, or diltiazem in the field, it must be absolutely clear that the QRS duration is less than three small boxes (0.12 seconds). This will help avoid the hemodynamic collapse that can occur with these drugs in VT or atrial fibrillation with an accessory pathway. Most patients tolerate the transient effects of adenosine, often “fooling” providers into thinking no harm is possible if given in error. The potential harm is real, albeit much less frequent than with calcium channel blockers. If hypotension occurs after IV verapamil or diltiazem in the absence of bradycardia, treatment with saline infusions, IV calcium salts (5–10 mL of a 10% calcium chloride solution) or catecholamines (i.e. dopamine or epinephrine) should be given.

Many WCTs are erroneously classified in the field as narrow (up to 20% of cases). Therefore, many medical oversight physicians prefer adenosine to treat all regular and symptomatic NCT, avoiding the risks associated with giving a calcium channel blocker to a patient with WCT. For those patients with chaotic NCT, atrial fibrillation is the likely rhythm. If mildly symptomatic and stable, no field treatment is required. An example is an elderly patient with an irregular NCT at a rate of 130/minute complaining of weakness. Although rapid atrial fibrillation may contribute to the symptoms, no field treatment is needed in the absence of other clear signs or symptoms of decompensation. Those with instability deserve immediate countershock with 50–100 J. If transport is prolonged and the patient has either borderline symptoms or a rate of 140–180/minute, metoprolol (5–10 mg intravenously) or diltiazem (0.15–0.25 mg/kg intravenously) will control the ventricular rate in 85–90% cases of rapid atrial fibrillation [10,11].

One pitfall in the treatment of stable NCT must be highlighted. When the ventricular rate is greater than 220/minute, the risk of decompensation rises and the ability to detect irregularity is limited [2]. Therefore, all adults with a very fast regular NCT (heart rate >220/minute) should be either cardioverted with 100 J or treated with adenosine and prepared for cardioversion. If the rate rises to greater than 250/minute, cardioversion is the best choice given the risk of deterioration. Irregular NCT greater than 220/minute deserves countershock promptly as previously noted (50–100 J).

**Stable wide-complex tachydysrhythmias**

Wide-complex tachydysrhythmias can be due to VT or a SVT with abnormal conduction. Until proven otherwise, field providers should assume any new WCT is due to VT. Hospital data suggest that about two-thirds of patients with new WCT have VT. With a history of previous myocardial infarction, the frequency of VT increases to 90%. Although it is possible to assemble evidence to detect supraventricular rhythms from a detailed examination and 12-lead ECG, these data are not easily obtainable in the field. Thus, actions in managing WCT should either treat or cause no harm in VT.

All unstable patients with WCT should be cardioverted with 100 J, with escalating energy doses if needed. When stable or borderline, a few simple measures can help stratify patients. It is always an option to observe this group, intervening only if conditions worsen.

If P-waves precede each QRS complex during a stable WCT with a rate of 140/minute or less, a supraventricular source is likely, especially sinus or atrial tachycardia, although VT is a remote possibility. Treatment focuses on correcting any potential causes (e.g. pain, hypovolemia, or hypoxemia) and observation. Irregular QRS complexes suggest atrial fibrillation or multifocal atrial tachycardia. Neither requires field rhythm-directed therapy in stable patients, although other actions (e.g. oxygen, bronchodilators) may be needed.

When no clear P-QRS relationship exists, differentiating between SVT and VT is difficult during a WCT. The following key features help decide a clinical course of action.

* A patient with new-onset WCT and a history of previous myocardial infarction or VT very likely will have VT.
* VT will often not slow during vagal maneuvers. Therefore, slowing of a WCT during these efforts suggests SVT. However, the absence of change does not diagnose VT.
* Most VT does not respond to adenosine, whereas SVT usually slows or terminates. Conversely, lidocaine has little effect on most SVT and will terminate 75–85% of VT.
* VT is usually regular and rarely seen at a rate of greater than 220/minute. Any chaotic WCT should be considered atrial fibrillation with abnormal conduction. When a chaotic WCT at a rate of greater than 220/minute occurs, atrial fibrillation from Wolff–Parkinson–White syndrome is present. This rhythm is prone to deterioration.

From these clinical observations, the following scheme can be used in approaching the stable or borderline (one minor sign or symptom of instability alone) patient with a WCT.

* All stable patients with regular WCT at a rate of 120–220/minute should receive vagal maneuvers. Those who slow but then elevate again should receive adenosine (6–12 mg IV). If no slowing with vagal maneuvers occurs, one of three paths should be taken.
	+ Young (age <50 years) previously healthy patients with stable (or borderline) regular WCT that slows with vagal maneuvers should receive adenosine. If this fails or there is no response to vagal maneuvers, or if the patient has had prior VT or prior MI, assume VT and give amiodarone (5 mg/kg IV over 5 minutes) or possibly lidocaine (1.0–1.5 mg/kg IV up to 3 mg/kg). The AHA has emphasized the role of amiodarone over lidocaine despite limited direct comparisons. If lidocaine converts the rhythm, repeat boluses at 5–10 minutes of 0.5 mg/kg should be given during transport to prevent recurrence. Continuous infusions after lidocaine loading are generally impractical in the field unless prolonged transport times are likely and infusion pumps are available.
	+ Because of the risk of deterioration, any patient with WCT at a rate of greater than 220/minute deserves countershock with 100 J, irrespective of symptoms.
	+ Patients with a chaotic WCT usually have atrial fibrillation with altered conduction. If stable with a heart rate of less than 200/minute, they deserve close observation and expeditious transport. If the rate elevates to 220/minute or higher, immediate countershock with 100 J is indicated.

Other agents are available but have a limited role in the field. Procainamide (50–100 mg IV every 1–2 minutes up to a maximum of 15–18 mg/kg or until side-effects occur) treats both VT and SVT but is difficult to give in the field.

**Controversies**

**Rhythm strip versus monitor interpretation**

Besides clearly abnormal rhythms (e.g. obvious VT or severe bradycardia), ECG interpretation should be taken from a tracing and not from the monitor screen. It is tempting to avoid printing strips, but misclassifications may result from a “screen look.” Strips are valuable in the ED evaluation, documenting conditions before and after field treatment, which helps unravel the causes in certain dysrhythmias. At least two leads should be sampled.

**Synchronization and sedation during countershock**

When possible, delivering a countershock synchronized with the intrinsic QRS complexes is preferred. Synchronization helps avoid depolarization during the vulnerable phases of repolarization, theoretically decreasing the risk of post-countershock VF. During most dysrhythmias, the defibrillator unit senses the underlying QRS pattern and delivers the shock at the appropriate time. When the rhythm is extremely fast or irregular or the QRS complexes are markedly abnormal (i.e. very wide or small), sensing is difficult. In these cases, an unsynchronized countershock is appropriate. Electrophysiological data do not support the notion that this will increase the likelihood of VF. If post-countershock VF occurs, repeat countershock is usually successful in restoring an organized rhythm.

The usual controversy surrounding field countershock is the awake unstable patient. Medical oversight must clearly communicate the need for this unpleasant but life-saving intervention for appropriate patients. Sedation with a benzodiazepine before countershock may improve patient comfort. However, countershock should not be delayed in unstable patients while awaiting clinical sedation.

**Prophylactic lidocaine for premature ventricular contractions**

In the past, lidocaine was given for all patients with suspected acute coronary ischemia and any evidence of ventricular ectopy. Research clearly details that most patients do not benefit from this medication and some may be harmed [12–14]. If the premature ventricular contractions (PVCs) are asymptomatic or trivial, there is no proven benefit from treatment. PVCs associated with more pronounced symptoms should receive an antidysrhythmic, usually lidocaine. Although oft-cited lists of ominous ECG “warning” signs exist (e.g. multiform, >6/minute, couplets, R-on-T, or runs of PVCs), treatment of these and other asymptomatic PVCs does not confer any benefit. Do not use prophylactic lidocaine for all patients with chest pain. Lidocaine may reduce the risk of VF but will increase the risk of asystole.

**Pediatric dysrhythmias**

When evaluating pediatric tachycardias, a crucial difference compared with adults must be stressed. Children under the age of 5 years can sustain a sinus tachycardia at much higher rates (up to 225/minute) in response to physiological stresses. Therefore, a search for hypovolemia, hypercarbia, and hypoxemia is mandatory in stable children with NCT before drug therapy is used. A volume challenge with 10–20 mL/kg of saline IV is useful before other therapies. Although some guidelines make a distinction between energy levels when performing synchronized versus unsynchronized countershock, the use of this distinction is dubious. To keep treatments simple but effective, unstable children deserve countershock with 2 J/kg. Antidysrhythmic principles are otherwise similar to those outlined previously, with agents given in the appropriate weight-based doses. Pediatric non-cardiac arrest bradycardias are also usually secondary to another cause, often respiratory distress or hypoxia. When symptomatic, these rhythms are treated primarily with epinephrine and airway maneuvers and rarely need transcutaneous pacing or atropine (0.02 mg/kg/dose).

**Torsades de pointes**

This rare dysrhythmia classically presents with paroxysms of syncope and polymorphic “twisting” of the QRS complexes ([Figure 14.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c14.xhtml?favre=brett#c14-fig-0001)). Torsades de pointes (TdP) in adults is usually “pause dependent,” flourishing when intrinsic heart rate drops below 80–100/minute. A variety of antidysrhythmics (essentially all aside from lidocaine and calcium channel or beta-adrenergic blocking agents), antihistamines, antimicrobials, and psychoactive drugs, along with metabolic disorders, can precipitate TdP. Field treatment consists of countershock when unstable and transcutaneous pacing or isoproterenol (titrated to a heart rate >120/minute). Magnesium sulfate, 2 g as a rapid IV bolus, is also suggested for those who fail countershock.



[**Figure 14.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c14.xhtml?favre=brett#R_c14-fig-0001) The classic one-lead ECG appearance (lead II here) of torsades de pointes. Note the shifting of the QRS complex axis and appearance.

A more practical problem is mistaking VT or VF for TdP. VT or VF often display some changes in QRS complex appearance. Field providers may mistake these variations for the classic, but rare, QRS twisting. If recurrent polymorphic VT occurs in a patient with one or more of the aforementioned risks, treatment should be started. Otherwise, orders and protocols should focus on the treatment of common VT.

**Rhythm disturbances in renal failure patients**

This group often falls prey to metabolic derangements that alter rhythms, in addition to having high rates of underlying heart disease. Hyperkalemia is a common complication of renal failure that can cause a bradycardia or a wide complex rhythm, although the latter is usually not above a rate of 100–120/minute and often much slower. Treatment should include IV calcium (10 mL of 10% of CaCl2), sodium bicarbonate (1–2 ampules intravenously), nebulized albuterol, and insulin plus glucose. The last three interventions rapidly (but temporarily) shift potassium into the cells and should be part of protocols for any renal failure patient with new-onset symptomatic bradycardia or a wide-complex rhythm. Because of the risk of hypoglycemia, insulin and glucose infusions in the field are best done under medical oversight supervision rather than by protocol.

Lidocaine can cause asystole in the presence of hyperkalemia. The role of other agents, including amiodarone, is unknown in the rare event of hyperkalemia and new-onset WCT [15]. If a rhythm-specific intervention is needed in unstable patients with suspected hyperkalemia, electricity (pacing for slow, countershock for fast rates) is a safe choice.

**Protocols**

When developing protocols, focus on the simple data and steps. For example, both the bradycardia and tachycardia protocols should start with a division between “stable/no symptoms” and “symptomatic and unstable or borderline.” Those in the “stable/no symptoms” category should be observed, expeditiously transported, and monitored, with precautionary IV insertion and oxygen. As a corollary, unstable patients with bradycardia or tachycardia should receive prompt electrical therapy (pacing or countershock), airway support, monitoring, and IV insertion occurring either in tandem with or after electrical therapy. Remind the providers to save rhythm strips and to give sedation if possible, but not to withhold life-saving treatment trying to “get a good strip” or titrating sedation. Unless the signs of instability are subtle, medical oversight contact should follow the initial treatment of unstable patients.

For patients who are symptomatic without signs of instability, EMS personnel should assess a rhythm strip first. In the tachycardia protocols, three questions should be asked: rate, QRS duration in small boxes, and regularity. Narrow-complex tachycardias that are regular deserve either vagal maneuvers (carotid massage and/or Valsalva) or adenosine. Those patients with irregular narrow-complex rhythms deserve calcium channel blocker therapy if symptomatic but stable. Patients with wide-complex regular rhythms who are stable or borderline should receive lidocaine or amiodarone, and countershock if these medications fail or deterioration occurs. Finally, those with irregular WCTs should be transported without therapy unless unstable, in which case, they should be treated with countershock.

**Conclusion**

Prehospital dysrhythmia evaluation must be tailored to the time restraints, physical limitations, and outcome needs that are specific to the field setting. Decision trees should be simple and effective, focusing on treating patients, and not rhythms *per se*. Protocols must identify and treat all unstable patients. Those without symptoms or with trivial symptoms do not require rhythm-directed therapies. For symptomatic but stable patients, a few key steps should be taken to help manage each case.

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