Dysrhythmia Assessment and Management Scenarios

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An Important Word About the Therapies Discussed Here

The authors have done everything possible to ensure that the recommendations are up to date and in accord with generally accepted standards. However, as new scientific information becomes available through basic and clinical research, recommended treatments and drug therapies undergo constant change. The American Heart Association’s Advanced Cardiac Life Support (ACLS) treatment guidelines—the basis for most of the recommendations contained here—undergo revisions every few years. The most current ACLS treatment algorithms should be consulted. Recommended treatments herein are based on the 2000 guidelines for emergency cardiac care. Therapies described should be applied by the reader in accordance with professional standards of care used in regard to the unique circumstances in each situation.

The reader’s scope of practice should conform to national, state, and regional treatment guidelines along with local practice standards. The reader is advised to always check product inserts and consult with the Physicians’ Desk Reference (PDR) and the American Hospital Formulary Service books for changes and new information regarding dose and contraindications before administering any drug. Caution is especially urged when using new or infrequently ordered drugs.
Introduction

The purpose of this material is to illustrate how the skills of dysrhythmia interpretation relate to clinical assessment to affect the selection of appropriate dysrhythmia treatment algorithms. The goal is not to stress a specific therapy, but to demonstrate how ECG interpretation, patient assessment findings, and treatment options are evaluated and selected. In other words, the question “How is a wide-complex tachycardia treated?” cannot be answered without considering several related factors, such as how the patient is tolerating the dysrhythmia and how urgently therapy is needed.

Although dysrhythmia recognition is taught as one distinct phase of cardiac care, clinical dysrhythmia interpretation must be done in conjunction with patient assessment. Only by considering the effect that a particular dysrhythmia is having upon a patient’s well-being can the significance of the abnormal rhythm disturbance be determined and a decision made as to which therapy, if any, is indicated. For instance, synchronized electrical cardioversion is indicated to treat a rapid supraventricular rhythm that is associated with shock, but an electric shock is often not needed if the patient is tolerating the fast dysrhythmia.

Clinical Scenarios

Case #1: “The case of the weak and dizzy elderly woman.”

Case Presentation: An Emergency Medical Services ALS ambulance receives a call for a “sick, elderly female.” Upon arrival, they are greeted by a man who
called 9-1-1 because his 84-year-old wife has been complaining of dizziness and generalized weakness for the last two hours. The patient, awake and appearing weak, describes briefly “passing out” while getting up from the couch to go to the bathroom. She was feeling fine until about two hours ago, when she suddenly became dizzy and had to be helped to lie down.

The patient denies chest pain, shortness of breath, diarrhea, palpitations, nausea or vomiting, blood in her bowel movements, abnormally dark stools, or prior episodes similar to today’s events. Her past medical history is significant for hypertension for which she is taking a diuretic and an ACE-inhibitor. Her medication has not been changed recently, and she takes her medication as directed.

**Physical Examination:** Her mental status is alert and oriented; blood pressure: 84/60 mmHg; pulse 36/min.; and respirations: 26/min. Pulse oximetry shows 98% saturation. Her skin is pale and sweaty, and her neck veins are not distended. Her breath sounds are clear and the heart sounds are regular without a murmur. Her abdominal and neurologic examinations are normal. Oxygen is administered and cardiac monitoring is started along with insertion of an intravenous intermittent infusion device. The ECG monitor recorded the tracing shown in Figure 1.

[INSERT FIGURE 1 HERE]
**Interpretation:** Third-degree A-V heart block with a bradycardic ventricular escape rhythm at a rate of 25/minute

**Reasoning:** This is complete A-V heart block because the tracing shows the typical findings of independent atrial and ventricular activity along with a very slow regular ventricular rhythm (25/minute). The QRS complexes are wide and distorted, indicating the presence of a low ventricular escape pacemaker. Atrial activity consists of P waves at a rate of 60/minute. When the P-R intervals are measured, a constant value cannot be found because the distance between the P waves and the QRS complexes is constantly changing. The P waves and QRS complexes are unrelated to each other since the upper and lower heart chambers are being paced by different pacemakers.

**Treatment Considerations:** The primary survey reveals that the patient is alert, and her airway and breathing are adequate. The patient’s circulation is inadequate because of the slow heart rate, but she is not in need of cardiac compressions. High-concentration oxygen via a non-rebreather mask or a nasal cannula should be administered. The “D” of the ABCDs pertains to disability (neurologic assessment) and differential diagnosis involving a consideration of what is causing the patient’s condition. Her neurologic examination is normal.

An intravenous lifeline is established should emergent medication become necessary before arriving at the emergency department. Continuous cardiac monitoring is being done. The patient’s symptoms appear to be directly related to
her slow heart rate. The patient is hypotensive and showing signs of adrenergic
discharge: pallor and diaphoresis. Although she seems to be compensating
reasonably well for the abrupt slowing of her heart rate, a blood pressure of 80
mmHg is very low for a patient who is normally hypertensive. She is dizzy and
initially lost consciousness. It is clear that this patient’s symptoms are related to
the bradycardic rate and that the slow rate needs to be treated. What treatment
is needed at this point?

Although third-degree heart block in elderly patients is usually due to gradual
fibrosis of the A-V conduction system, the patient needs to be assessed and
treated expectantly as if she were experiencing an acute coronary event. MIs
occurring in the elderly often present atypically; a significant percentage of such
patients do not experience the traditional complaint of chest pain. In such
atypical cases, elderly MI patients describe only a vague abdominal or chest
discomfort, nausea, pronounced sweating, sudden weakness, or fatigue.

**Treatment plan:** The Emergency Cardiac Care Treatment Algorithm for Adult
Bradycardia is found in Figure 2. If the patient’s condition is critical or unstable,
several of these measures may need to be implemented in rapid succession.
For instance, artificial pacing and atropine administration may be done
simultaneously for an unstable patient. For a patient who is relatively stable, as
this patient is, treatment may start with either atropine or artificial pacing.
Indicated therapeutic options include:
• Atropine (0.5 to 1 mg) should be given in repeated doses every three to five minutes up to a total of 0.03 to 0.04 mg/kg (approximately 3 mg for a 70 kg patient).

• External (transcutaneous) artificial pacing for severely symptomatic patients is the treatment of choice and should not be delayed while establishing intravenous access for atropine administration.

• Dopamine infusion at 5 to 20 mcg/kg/minute can be added and the dose increased quickly if hypotension accompanies the slow rate.

• Epinephrine infusion at 2 to 10 mcg/minute can be started before dopamine if the patient shows severe symptoms.

Figure 2

Emergency Cardiac Care Treatment Algorithm for Adult Bradycardia*

*Absolute bradycardia is a rate below 60/min while relative bradycardia is a rate less than expected given the underlying condition.

Initial Assessment

• ABCD: Airway, breathing, circulation, differential diagnosis

• Is there a need for basic life support?

• What is causing the patient’s problem?

• Is there a need for advanced life support?

• Is there a need for respiratory or circulatory adjuncts to secure the airway, breathing, or circulation?

• Endotracheal intubation, IV access, ECG monitor, vectored history, and physical examination

Are there severe symptoms that are related to bradycardia?
Is an advanced type of heart block present (second-degree, Mobitz type II, or third-degree)?

No

- Anticipate ECG rhythm to deteriorate; monitor patient and prepare for pacing if condition worsens
- Observe for changes in condition

Yes

- **Treatment**
  - Atropine 0.5 to 1 mg boluses
  - Transcutaneous pacing
  - Dopamine infusion at 5-20 mcg/kg/minute
  - Epinephrine infusion at 2 to 10 mcg/minute

**Treatment Option Discussion**

Once the decision is made to accelerate the patient’s heart rate, the next major question is “How fast should the rate be?” Attaining a specific heart range goal with atropine is difficult. Atropine decreases vagal (parasympathetic) tone, thereby allowing sympathetic nervous tone to predominate. In the case of a slow ventricular escape that develops due to impulse blockage in the bundle branches, atropine is often ineffective due to the lack of parasympathetic fibers. Even if atropine were effective, the increased rate can be faster than desired, possibly leading to worsening ischemia and possible extension of an infarction.

A reasonable goal is to increase the rate enough to raise the patient’s blood pressure to the point of ensuring adequate coronary artery perfusion, but not so
fast that it would worsen cardiac ischemia. For this reason, many clinicians favor
external pacing instead of atropine because the desired heart rate can be
accomplished gradually and precisely. Pacing can be increased in a stepwise
fashion beginning just a few beats above the native rate until the systolic blood
pressure is about 100 mmHg.

Pacing should be started immediately if atropine fails to increase the rate or if the
patient is significantly hypotensive and symptomatic. Analgesia and sedation are
usually necessary to facilitate patient comfort and acceptance of external pacing.
The procedure should be explained to the patient prior to starting pacing or the
minor chest discomfort will cause additional anxiety. Verify adequate cardiac
capture by confirming that central pulses are palpated with each paced complex.

- **Clinical Note:** Always consider that hypotension associated with
  bradycardia may be due to other conditions such as hypovolemia or
cardiac dysfunction. A slow heart rate may be due to the patient’s
medications, which may include beta-adrenergic blocking agents or
calcium channel antagonists.

Another choice to accelerate the heart rate is a catecholamine infusion, using
either epinephrine or dopamine. The drawback, especially in the prehospital
environment, is the need to mix the medication and set up the IV equipment,
which is why this is rarely done. A catecholamine infusion stimulates both alpha-
and beta-adrenergic receptors, enhancing both blood pressure and heart rate.
An epinephrine infusion is particularly helpful in treating hypotension that is
associated with bradycardia. Again, the concern about increased myocardial oxygen demand exists, but there is greater rate control than with atropine bolus injections.

- **Clinical Note:** An interesting but rare cause of a third-degree atrioventricular heart block in young adults is advanced Lyme disease. Lyme disease can be associated with conduction defects due to inflammation of the conduction pathway.

- **Clinical Note:** If the patient is in critical condition and in danger of developing imminent cardiac arrest, multiple interventions are started simultaneously, such as endotracheal intubation, administration of atropine, and epinephrine infusion. Likewise, atropine would be given at shorter dosing intervals, every three minutes.

**Case #2: “The case of the hospitalized patient recovering from an MI.”**

**Case Presentation:** The patient, a 54-year-old man, was recovering in the intensive care unit from an uncomplicated inferior wall MI that he experienced 24 hours before. Fortunately, the thrombolytic therapy opened the occluded coronary artery and his chest pain, along with the acute ECG changes, subsided. The nurse went to his room in response to a dysrhythmia alarm and found the patient unresponsive, propped up in bed, not breathing, and without a pulse. The nurse summoned help, lowered the head of the bed, and observed the ECG rhythm shown in Figure 3.
**Interpretation:** Ventricular fibrillation

**Reasoning:** The rhythm consists of a rapid, disorganized ventricular tachydysrhythmia. Distinct QRS complexes are absent. Instead, the ECG baseline zigzags across the tracing. Discrete P waves are also not visible, so an atrio-ventricular relationship cannot be determined. The patient’s clinical appearance confirms the interpretation.

**Initial Treatment Considerations:** Resuscitative measures must be instituted at once in order to convert this life-threatening ECG dysrhythmia to a nonlethal rhythm. Aside from summoning help and confirming the cardiac arrest, ventricular fibrillation must be countershocked as rapidly as possible. Since a defibrillator is present in each ICU room, three immediate defibrillation shocks were performed in quick succession using increasing energy levels of 200 joules, 200 to 300 joules, and 360 joules. The Emergency Cardiac Care Treatment Algorithm for Ventricular Fibrillation/ Pulseless Ventricular Tachycardia is found in Figure 4. The defibrillation attempts were not successful, so CPR was started and endotracheal intubation was done. The patient already has IV access. Additional treatment includes:

- A 1-mg bolus dose of epinephrine or a 40-unit IV bolus of vasopressin is given once, followed by a saline flush. The dose of epinephrine should be repeated at 3- to 5-minute intervals for the duration of the arrest while vasopressin is only given once. Vasopressin has been
added to the protocol based on the evidence that the drug is a natural antidiuretic hormone that has potent vasoconstrictor properties, which raises coronary artery perfusion. Since vasopressin has a half-life of 10 to 20 minutes, it requires only a single dose compared with epinephrine. After 10 to 20 minutes, epinephrine should be tried if vasopressin is unsuccessful.

- Thirty to sixty seconds of CPR follows all medication administration in order to circulate the medication, and defibrillation is performed again at 360 J.

**Figure 4**

**Emergency Cardiac Care Treatment Algorithm for Ventricular Fibrillation/ Pulseless Ventricular Tachycardia**

**Initial Assessment/Basic CPR Measures**

- Check for responsiveness
- Open the airway
- Ventilate nonbreathing patients
- Begin chest compression
- Defibrillate (200 J, then 200-300 J, and then 360 J)

Refractory VF/pulseless VT is assumed for rest of protocol.

**Advanced ALS Measures**

- Airway: endotracheal intubation or laryngeal mask airway
- Ventilate after confirming position of endotracheal tube
- Start IV, ECG monitor to determine rhythm
- Drugs and differential diagnosis
Initial Medication

- Epinephrine 1 mg IV push every 3-5 minutes or
- Vasopressin 40-unit single IV bolus

Defibrillate at 360 J after 30-60 seconds.

Further Medication Considerations

Anti-arrhythmics
- Amiodarone
- Lidocaine
- Magnesium
- Procainamide

Buffering Agent
- Sodium Bicarbonate

Subsequent Antidysrhythmia Treatment Considerations: What therapy should be considered if these initial steps are not effective? Shock-refractory VF/pulseless VT has a low conversion rate. In line with recent cardiac arrest research, antidysrhythmic medication, adrenergic agents, and buffering medication have been shifted to a secondary role in resuscitation. Multiple antidysrhythmic medication use has a pro-arrhythmic effect. Therefore, the treatment of VF and pulseless VT refractory to shocks should focus on assessing the cause of the arrest and correcting treatable conditions as shown in Figure 5.

Figure 5

Reversible Causes for Ventricular Fibrillation and Pulseless Ventricular Tachycardia
Despite the aggressive treatment strategies used over the last 25 years, credible evidence does not exist to show that buffers and adrenergic and antidysrhythmic agents are effective in successfully treating shock-resistant ventricular fibrillation. The best hope for resuscitation remains early detection and early defibrillation. In one double-blind study, amiodarone was shown to be more effective than lidocaine in converting prehospital VF but did not lead to improved hospital discharge—the overall goal of all interventions. However, for persistent or recurrent ventricular fibrillation or pulseless ventricular tachycardia, consider the following therapies:

- Amiodarone in a 300-mg rapid intravenous bolus. If successful conversion results but VF/pulseless VT recurs, repeat amiodarone at the initial dose. Amiodarone has characteristics of all four major.
classes of anti-arrhythmic drugs: it generates anti-sympathetic action, blocks sodium and potassium channels, and lengthens the action potential.

- Lidocaine (1 mg/kg) intravenously. When the initial dose is unsuccessful, it is repeated 3 to 5 minutes later to a maximum total dose of 3mg/kg. A single dose of 1.5 mg/kg is an alternative. Despite lidocaine being the traditional drug used for VF/pulseless VT, no study has shown that it is an effective drug in humans for shock-resistant cases. For that reason, lidocaine is now recommended after amiodarone until further evidence is available.

- Magnesium sulfate in a 1- to 2-g dose for polymorphic forms of VT or in known hypomagnesemic states.

- Procainamide intravenously at 30 mg/min up to 17 mg/kg (average of 1200 mg per 70-kg patient) can be given for refractory cases, but it is not recommended because it is often not practical to administer during cardiac arrest due to the preparation and administration time.

- Sodium bicarbonate intravenously at 1 mEq/kg for cases of severe acidosis or hyperkalemia. This is also useful for tricyclic antidepressant or aspirin overdoses.

**Case discussion:** The “D” in the ABCDs of the initial assessment stands for *differential diagnosis*, or exploring likely causes for the arrest. The rescuers need to determine if there are treatable causes, such hypovolemia, hypoxia, acidosis,
electrolyte disorders such as hypo- or hyperkalemia or hypocalcemia, or toxins from drug overdose (tricyclic toxicity). If these disorders are not recognized, standard ACLS treatment for ventricular fibrillation/ventricular tachycardia probably will not be effective.

Case #3: “The case of the man who ‘passed out’ during his dialysis session.”

Case presentation: A 70-year-old male with a history of hypertension, end-stage renal disease, and insulin-dependent diabetes mellitus arrives in the emergency department by BLS ambulance after experiencing a brief syncopal episode during his dialysis treatment thirty minutes before arrival. The EMTs report that the dialysis center staff found the patient diaphoretic and hypotensive and administered oxygen and 500 cc of normal saline. This occurred just as he was finishing his dialysis session. His bedside glucometer determination was 140 mg/dl. Vital signs in the ED include a blood pressure of 130/60 mmHg and a regular pulse of 150/min. The patient is alert, afebrile, and his respirations are 26/min. His lung sounds are clear and there is no pedal edema. The ECG tracing is shown in Figure 6. The dialysis center sent a baseline ECG taken one week ago that shows a normal sinus rhythm. He denies chest pain or shortness of breath but is complaining of palpitations, nausea, and generalized weakness.

[INSERT FIGURE 6 HERE]

Interpretation: Atrial flutter with a 2:1 A-V conduction and a rapid ventricular rate at 150/minute
**Reasoning:** The rhythm is grossly regular with a rapid ventricular rate, and the ventricular complexes are narrow. P waves are not visible; they have been replaced by a regular fluctuation of the baseline consisting of flutter waves, which are best seen in leads $V_1$ and $V_2$. Slight baseline artifact is observed in leads II and aVL. The ST segments are difficult to evaluate due to the flutter waves. The A-V relationship consists of 2:1 conduction, meaning that every other flutter wave is not conducted. The atrial rate is 300/minute, and 2:1 conduction prevents the ventricles from being overstimulated. However, the ventricular rate of 150/minute is very fast for a patient with limited cardiac reserve.

**Treatment Considerations:** Because this patient has multiple medical problems, it is easy to forget to follow a systematic dysrhythmia approach. However, a standard approach helps to ensure that all of the ECG aspects are covered. A dialysis patient who is also diabetic is “medically fragile” due to his relative immunocompromised status. Almost any symptom could indicate a serious underlying condition. Syncope, diaphoresis, nausea, and hypotension in an elderly diabetic patient strongly suggest that an acute coronary event or a pulmonary embolism be considered, in addition to evaluating the new tachydysrhythmia. It is not uncommon for the description of chest pain in diabetic patients to differ from the classical description and location. Atypical case features should not deter the practitioner, especially when treating elderly and diabetic patients, from considering acute coronary syndrome as the cause of a sudden change in the patient’s condition.
**Initial Assessment:** The initial therapy for all patients involves attention to the "ABCDs": a check of responsiveness, airway, breathing, and pulse, all of which are normal in this case. Pulse oximetry, continuous cardiac monitoring, and serial automatic blood pressure assessments are begun soon after the ABCDs are examined. The standard approach for "ruling out" an MI is prudent, meaning that the patient is treated as if he were experiencing an MI even if the condition turns out to be another problem. Following this principle, supplemental oxygen administration, intravenous access, 12-lead ECG determination, and serum myocardial release marker testing are all performed.

The general approach to atrial fibrillation/flutter with rapid ventricular rate is based on the clinical evaluation. Unstable patients are treated emergently using electrical therapy to convert the rhythm. For stable patients, the plan is to control the rate, convert the rhythm, and, if not convertible, add anticoagulation and maintain rate control.

**Dysrhythmia Treatment:** The new ACLS guidelines differ from previous approaches by emphasizing specific rhythm interpretation and recognizing those tachycardic patients with severely compromised cardiac output who exhibit signs of heart failure or who have a previously determined ejection fraction less than 40% (Figure 7). This approach has replaced the single protocol used in prior ACLS guidelines with three algorithms and a table. However, it is believed that
this new approach will better target specific tachydysrhythmia treatment. This scenario will focus on the approach to new-onset atrial flutter. The approach divides patients with atrial flutter into two major groups: unstable versus stable.

In hemodynamically unstable patients, the rapid ventricular rate in a new-onset atrial flutter is the major reason for decreased cardiac output. Two other mechanisms can also lower the blood pressure: a) the rapid ventricular rate impairs ventricular filling by decreasing diastolic filling time; or b) rapid rates increase myocardial oxygen demand and cause ischemia in patients with diminished cardiac reserves. The immediate goal of emergency cardiac care is to slow the ventricular response with medication, thereby improving myocardial function. Hemodynamically unstable cases should be converted immediately using synchronized electrical cardioversion in the following energy levels: 100 J, 200 J, 300 J, and 360 J.

In hemodynamically stable patients, conversion of atrial flutter to normal sinus function, using medication or electrical countershock, can be done after the patient’s ventricular heart rate is stabilized and, if needed, anticoagulated to prevent embolic complications since the noncontracting atria are prone to forming atrial thrombi. Patients with stable hemodynamics and congestive heart failure should receive medication that controls the rate without further depressing contractility, such as digoxin, diltiazem, or amiodarone. To avoid pro-arrhythmia effects, only one of these drugs should be used.
Tachycardia: rate >100/minute but symptoms are unusual until rate is over 150/minute

**Initial Assessment**

- Basic airway, breathing, and circulation measures, along with differential diagnosis
- Advanced airway and breathing techniques, along with vectored history and physical examination

**Stable versus Unstable Condition**

- Unstable patients are treated with electricity: immediate cardioversion.
- Stable patients are evaluated to determine if the rhythm is a wide or narrow complex:
  - Wide-complex tachycardias are usually ventricular tachycardia (see VT or wide-complex tachycardia of uncertain type algorithm).
  - Narrow-QRS-complex tachycardias are supraventricular tachycardias (PSVT) or atrial flutter/fibrillation (see narrow-complex treatment or atrial fibrillation/flutter table in Figure 8).

**Clinical Note:** Patients with atrial flutter/fibrillation who also have Wolff-Parkinson-White syndrome (pre-excitation due to an accessory pathway) receive special consideration. While this is a rare situation, the traditional drugs used to treat atrial flutter or fibrillation can dramatically accelerate the ventricular rate instead of slowing the heart rate.
The rate-control and rhythm-conversion tables for atrial flutter/fibrillation are found in Figures 8 and 9. Four general tachydysrhythmia categories are used to direct therapy: atrial fibrillation/flutter, narrow QRS-complex tachycardias, stable monomorphic or polymorphic tachycardia, and wide QRS-complex tachycardias of unknown type. In this case study, the dysrhythmia is atrial flutter and the patient is hypotensive and tachycardic.

Patients with atrial flutter who are stable generally have a ventricular rate less than 130 per minute and seldom require immediate electrical cardioversion. Drug therapy with one of the following agents is the best option. Calcium channel blockers or beta-blocking agents are most commonly used.

- Intravenous diltiazem (Cardizem®) administration is usually effective in slowing the ventricular response within ten minutes in atrial fibrillation. The average adult diltiazem dose of 20 mg is given intravenously over three to five minutes. Diltiazem may cause an initial blood pressure decrease, but this is a small effect and subsides as myocardial function improves. If the rate decrease is not adequate, additional diltiazem can be given (average dose: 25 mg over three minutes). A diltiazem intravenous infusion can be used to continue the rate reduction.
- Verapamil (Isoptin®, Calan®) is a calcium channel blocker that is effective in slowing the ventricular response. The intravenous
dose is 2.5 to 5 mg over two minutes and can be repeated in 15- to 30-minute intervals up to a total of 20 mg. Verapamil is associated with more hypotension than diltiazem.

- Beta-blocking agents, including IV and oral metoprolol, atenolol, and propranolol, can be used to slow ventricular response in patients who do not have A-V heart block or bronchospastic lung disease.
  - Metoprolol (Lopressor®): 5 mg intravenously every five minutes up to 15 mg or until an acceptable ventricular heart rate results. An oral dose may follow the IV dose at 50 mg twice a day for the first 24 hours and increased to 100 mg twice a day thereafter.
  - Atenolol (Tenormin®): 5 mg by slow intravenous administration. Ten minutes later a second 5-mg dose can be given. An oral dose of 50 mg may be given twice a day.
  - Propranolol (Inderal®) at a total dose of 0.1mg/kg divided into three doses by slow intravenous administration at two- to three-minute intervals. The injection should not exceed 1 mg/min.

- **Clinical Note:** Using a beta-adrenergic blocking agent in conjunction with a calcium channel blocker, such as diltiazem, should be avoided
as the combined effect commonly causes a significant fall in blood pressure and has caused hypotension and cardiovascular collapse. If rapid rate control is required, digoxin as the sole agent is not useful because it requires several hours to be effective. Beta-adrenergic blockers and calcium channel blockers have a faster result. In cases of borderline vital signs, choosing medications commonly delays advancing to electrical cardioversion. Therefore, avoid prolonged attempts at medication control of new-onset atrial fibrillation if the patient is unstable.

Other drugs have more specific indications in stable atrial fibrillation/flutter. Digoxin can be used, although it has the slowest action and is less potent than calcium channel blockers or beta-adrenergic blockers. The dosing of digoxin is 10-15 mcg/kg lean body weight intravenously over three to five minutes. Digoxin takes several hours to become effective. One benefit of digoxin is that it does not decrease contractility. Digoxin may be useful in cases of congestive heart failure associated with AF or atrial flutter.

Procainamide (Pronestyl®) is effective in controlling atrial as well as ventricular tachydysrhythmias, and it is especially useful when a wide-complex rhythm of uncertain origin--ventricular tachycardia or supraventricular tachycardia with aberration--is present. Drawbacks that limit wider use of procainamide are the loading regimen of twenty to thirty minutes and the common side effect of
hypotension. The dose is 20 mg/min by IV infusion until there is desired ventricular slowing, hypotension develops, the QRS-complex width widens by more than 50%, or a total of 17 mg/kg is given. It should be reserved for patients with adequate blood pressure.

Amiodarone (Cordone®) is useful for rate control but may lead to conversion to NSR. Therefore, it should be used when the dysrhythmia has been present for less than 48 hours or after adequate anticoagulation has been accomplished.

In stable patients with new-onset atrial fibrillation/flutter of less than 48 hours duration, medication can be used to convert the dysrhythmia to sinus rhythm. This is not done during the prehospital phase of emergency care, nor is it usually done in the ED. It is commonly reserved for when the patient is in the intensive care unit. One of the following medications can be used:

- Amiodarone has been previously described.
- Flecainide is an antidysrhythmic drug that is not approved for use in the United States in IV form. It is given in an oral dose of 2 mg/kg.
- Propafenone has nonselective beta-adrenergic blocking properties. It slows conduction and depresses contractility. The IV dose is 2 mg/kg and must be infused slowly at 10 mg/min.
- Procainamide has been previously described.
- Ibutilide is available only in IV form. It is a sodium channel blocker with conduction-slowing action. Ibutilide carries an increased risk of
ventricular pro-arrhythmia including *torsades de pointes*. The dose is based on weight; a reference text should be consulted.

**Clinical note:** Adenosine is not indicated for treatment of atrial fibrillation/flutter due to its extremely short half-life. A single dose may be appropriate for diagnostic purposes in stable patients when a narrow complex tachycardia at 150/minute is present and there is possible atrial flutter with 2:1 A-V conduction. Adenosine can unmask hidden flutter waves and confirm the interpretation.
Figure 8

Rate Control in Atrial Flutter/Fibrillation in Stable Patients

Rate control is the immediate concern; rhythm conversion is secondary.

Stable patients without Wolff-Parkinson-White syndrome or congestive heart failure:

- Calcium channel blocker or
- Beta-adrenergic blocker or
- Digitalis (least potent and slowest onset)

Patients with congestive heart failure but without Wolff-Parkinson-White syndrome:

- Digoxin or
- Diltiazem or
- Amiodarone (can be used in conjunction with digitalis if needed; conversion to NSR may occur so usually used with AF/flutter of less than 48 hrs or if adequate anticoagulation attained)

[END FIGURE 8]
Figure 9
Rhythm Conversion of Atrial Flutter or Fibrillation

The treatment is based on the duration of dysrhythmia. Dysrhythmia of less than 48 hours can be converted without anticoagulation. For dysrhythmia longer than 48 hours, there is a high risk of embolic complication.

Atrial flutter or fibrillation less than 48 hours* without CHF and without W-P-W syndrome

- Medication (use one drug**):
  - Amiodarone or
  - Ibutilide or
  - Flecanide or
  - Propafenone or
  - Procainamide
- Synchronized electrical cardioversion

Atrial flutter or fibrillation less than 48 hours* with CHF and without W-P-W syndrome

- Synchronized electrical cardioversion or
- Amiodarone

* not on therapeutic Coumadin® level

** Two drugs increase the chance of pro-arythmia effect.

[END FIGURE 9]
Case #4: “The case of the woman with the fast, wide rhythm.”

Case Presentation: A 65-year-old female who had a “heart attack” three months ago called 9-1-1 because of palpitations and lightheadedness that began 30 minutes ago. She denies chest pain but feels short of breath and nauseated. The patient’s medication includes a lipid-lowering drug, daily aspirin, beta-adrenergic blocker, and ACE-inhibitor. Her vital signs include blood pressure of 122/84 mmHg, regular pulse of 200/minute, and respirations of 24/minute. The ECG rhythm shows a wide-complex tachycardia (Figure 10). Her skin is dry, pale, and cool. Her neck veins are not distended. Her breath sounds have rales one-third of the way up from her lung bases bilaterally. Her heart sounds are rapid and regular without murmurs. Her extremities have slight (1+) pedal edema.

[INSERT FIGURE 10 HERE]

Interpretation: Ventricular tachycardia at 200/minute

Reasoning: The rapid ventricular rhythm is regular, the QRS shape is distorted, and the QRS duration is greater than 0.14 second. Regularly occurring P waves are not visible, so an A-V relationship cannot be determined. This wide-complex tachycardia occurring in a patient with a past MI makes the likelihood of a malignant (ventricular) dysrhythmia all but certain.

- Clinical note: An unlikely cause of such a wide-complex tachycardia is a supraventricular dysrhythmia with aberrant conduction. In
emergency cardiac care, wide-complex tachycardias in symptomatic patients are best assumed to be VT unless the patient is known to have had prior episodes of SVT with aberrant conduction. This is especially true in the prehospital field where the ability to further identify the wide-complex tachycardia is limited and time pressures are great. Therefore, avoid administering adenosine to such patients as precipitous deterioration may result and will involve the delay of appropriate treatment.

**Treatment considerations:** Assessment of this patient’s “ABCDs” along with vital sign determination and a brief physical exam are good starting points for patient evaluation. The patient’s airway is patent. She is alert and has stable vital signs. The patient is receiving oxygen via nasal cannula and has a functioning intermittent infusion device should IV medication be needed. Next, attention is directed to determining if the patient requires emergency cardiac care medication at the scene or en route to the hospital. If the patient does require therapy, a choice must be made between medication or electricity.

**Antidysrhythmia therapy:** The 2000 American Heart Association guidelines call for immediate electrical cardioversion for unstable patients who are experiencing serious signs or symptoms with unstable tachycardias of any type, including VT. In the hypothetical case presented, the elderly patient is tolerating the dysrhythmia reasonably well but is showing signs of congestive heart failure. Her
dysrhythmia does not require an immediate electric shock (although this is an option). However, the presence of rales and dizziness requires urgent therapy. If medication is not effective or if the patient’s condition worsens, DC cardioversion can be performed.

Stable monomorphicon VT pertains to patients with monomorphicon VT (having a constant appearance, unlike the changing pattern, or torsades de pointes, found in polymorphicon VT), who are not experiencing symptoms or showing signs of shock or inadequate tissue perfusion. Figure 11 illustrates the treatment approach to cases of stable monomorphicon VT.

Figure 11

Treatment of Stable Monomorphicon Ventricular Tachycardia

For Normal Cardiac Function (no CHF signs)

Preferred Drugs*

• Procainamide or
• Sotolol

Acceptable Second-Line Drugs

• Amiodarone or
• Lidocaine

* Procainamide, sotalol, amiodarone are preferable to lidocaine.
Congestive Heart Failure (decreased cardiac ejection fraction)

- Amiodarone
- Lidocaine; then
- Synchronized cardioversion at 100 J, 200 J, 300 J, and 360 J

If the ECG is not clearly VT or SVT with aberrant conduction, the emergency cardiac care treatment algorithm for adult patients with wide-complex tachycardia of unknown type who are in stable condition but showing signs of low ejection fraction (heart failure) is listed in Figure 12.

Figure 12

Wide-Complex Tachydysrhythmias: Stable VT

Shape and Rhythm: Monomorphic VT

Impaired Cardiac Functioning; Signs of CHF:
Consider electrical cardioversion if patient has pronounced signs
Medication:
- Amiodarone or
- Lidocaine; then
Electrical cardioversion

Normal Cardiac Functioning; No CHF
First-Line Medication:
- Procainamide or
Stable, wide-QRS complex tachydysrhythmias are divided into three general types:

- Known SVT rhythms that are complicated by aberration or bundle branch block
- Traditional (monomorphic) VT or polymorphic VT, also known as *torsades de pointe*. Treatment is based on whether the baseline QT interval is normal or prolonged. Prolonged QT-related VT is much more likely to be polymorphic VT.
- Wide-complex tachycardia of unknown type, in which the specific dysrhythmia type is uncertain. In unknown-type wide-complex tachydysrhythmias, which are very common, treatment depends on whether there are signs of heart failure present. CHF is treated with electrical cardioversion or amiodarone. If CHF is absent, electrical cardioversion, amiodarone, or procainamide can be used.

Because the QRS complex shapes are identical and do not wax and wane as polymorphic VT does, this case presented in this scenario is likely to be
monomorphic VT. It is important to limit the number of antidysrhythmic drugs given as they exert a strong pro-arrhythmic effect.

One option is intravenous amiodarone. This drug’s actions include alpha- and beta-adrenergic blocking, as well as blocking of calcium, sodium, and potassium channels. One of its major effects is to lengthen the cardiac action potential, thereby prolonging the refractory period. It is effective for hemodynamic stable VT. In patients with impaired heart function, it has a lower incidence of pro-arrhythmic effect. IV amiodarone is given as a 150-mg infusion over 10 minutes with careful attention to the development of hypotension and bradycardia, the two most common side effects. Amiodarone can be followed by a 1-mg/min infusion for 6 hours, decreasing to 0.5 mg/min thereafter. If recurrent episodes of VT occur, additional 150-mg infusions over 10 minutes can be done up to a total daily dose of 2 g.

DC cardioversion is another option, using escalating energy levels of 100 J, 200 J, 300 J, and 360 J. The procedure should be explained to the patient, and an awake patient—as described in this scenario—should be pretreated with a benzodiazepine and/or a narcotic to lessen the discomfort of the electric shock.

- **Clinical Note.** All antidysrhythmic drugs have the potential to actually cause dysrhythmias, an occurrence known as pro-arrhythmic effect. This can be avoided by limiting the number of antidysrhythmic drugs
used. Should one drug be ineffective for VT, use of electrical cardioversion is an option.

Prevention of dysrhythmia recurrence after control of the acute dysrhythmia is important. Serial ECGs, cardiac enzymes and other heart damage markers, supplemental oxygen supply, cardiac monitoring, and pulse oximetry are indicated. Serum electrolyte analysis should be monitored. Infusing an antidysrhythmic agent after restoration of sinus rhythm is controversial. However, starting an infusion of the drug that was effective in converting the rhythm, whether it is lidocaine or amiodarone, seems acceptable.

- **Clinical Note:** During treatment of a life-threatening dysrhythmia such as ventricular tachycardia or fibrillation, it is easy to focus solely on therapy for the dysrhythmia and get distracted from searching for underlying precipitating causes. Treatable causes such as ischemia, congestive heart failure, or electrolyte disorders (hypomagnesemia and hypokalemia are especially common in hospitalized patients) may be correctable and will prevent the reemergence of breakthrough dysrhythmias. Electrical instability does not occur in isolation, so do not neglect to attack the underlying cause(s) of the malignant dysrhythmia.