Arrhythmias in the Office

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The incidence of patients who present to the office with arrhythmia and hemodynamic instability is unknown. Emergency medical systems data, based on ambulance runs, are available only for patients who have had a cardiac arrest. When faced with an unstable or potentially unstable patient, however, we must be prepared to act quickly, safely, and accurately. This following text addresses the general approach to such a patient; provides necessary information on office emergency preparation, including training, rapid response team protocol, and the use of automated external defibrillators; and addresses the identification and initial office management of the various rhythms that are capable of threatening a patient’s life.

Preparing for office emergencies

Being prepared to deal with an emergency in the office setting is not an easy task. It requires protocols, equipment, and training, all of which take considerable effort and time. The current recommended level of preparedness is not clear from the literature. Brief reports in the literature demonstrate the variability in equipment and training [1,2]. In 1984, a survey of office physicians reported that 11% had adequate equipment to manage common office emergencies. Seventy-nine percent were basic cardiac life support (BCLS)-certified, 35% were advanced cardiac life support (ACLS)-certified, 19% had defibrillators, 35% had intravenous (IV) catheters, and 40% had laryngoscopes. Only 42% of the participants responded to the questionnaire [1].

Training may be the most important effort that leads to office preparedness. Investigators from Department of Pediatrics at Duke University...
School of Medicine conducted a prospective, randomized, controlled trial of primary care practices (pediatric, family practice, and health departments) that evaluated the effectiveness of an office-based educational program [2]. This program was designed to improve the preparation of primary care practices for pediatric emergencies. Practices that agreed to participate were assigned randomly to the intervention or the control group. Unannounced mock codes were conducted in the intervention practices. Practices were expected to respond to the mock code using their own staff, equipment, and local emergency medical system. After the exercise, there was a structured debriefing session. The primary outcome measures were obtained by survey 3 to 6 months after intervention and included (1) purchase of new pediatric emergency equipment and medications, (2) receipt or updating of basic life support/pediatric advanced life support/advanced life support training by staff members, and (3) development of written emergency pediatric protocols. The control practices received no interventions during the trial and completed a similar outcome survey. Thirty-nine practices (20 intervention, 19 control) completed the trial. Intervention practices were more likely to develop written office protocols (60% versus 21%) and receive additional basic and advanced life support training 3 to 6 months after the intervention (118 versus 54). There were no significant differences in the purchase of new equipment or medications.

Literature that is specific to arrhythmias in the office setting is scarce. Ultimately, an arrhythmia that presents with concerning complaints (Box 1)

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<th>Box 1. Symptomatic arrhythmias: warning symptoms of hemodynamic compromise</th>
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<tr>
<td><strong>Symptoms</strong></td>
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<td>• Lightheaded</td>
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<td>• Chest pressure/pain</td>
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<td><strong>Signs</strong></td>
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<td>• Hypotension</td>
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<td>• Heart rate &gt;130 beats/min (bpm) or &lt;50 bpm</td>
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<td>• Temperature &lt;35°C</td>
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<td>• Cyanosis</td>
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<tr>
<td>• Cool extremities</td>
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<tr>
<td>• Altered mental status</td>
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<td>• Tachypnea</td>
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needs to be managed in an emergency department (ED) with ACLS measures. Response times of ambulances vary, but they can be up to 25 minutes [3–6]. Response times from an internally affiliated emergency team might be faster. Hence, the focus of this discussion is based on providing the tools to assess and manage a patient for the first 30 minutes in the office setting.

The most important step for all patients who present with an arrhythmia or with any life-threatening presentation is being prepared for a cardiopulmonary arrest. Preparedness requires basic cardiac life support training, a simple office emergency protocol, some basic equipment, and rapid access to an emergency response team.

Education

The author recommends training all office personnel in basic life support and the use of an automated external defibrillator (AED). This training is easily obtainable from the American Heart Association (available at http://www.americanheart.org. The basic life support health care provider course teaches cardiopulmonary resuscitation (CPR) skills for helping victims of all ages, and the use of an AED. It is intended for health care providers, such as physicians, nurses, respiratory therapists, physical and occupational therapists, physician’s assistants, aides, medical or nursing assistants, and other allied health personnel. The course length is 6 to 8 hours. Education is the first step in setting up your office protocol for emergencies.

Equipment

Many commercially prepared first aid kits are available for the office setting. They range from first aid kits (eg, Rescue One First AID) to ACLS rescue kits similar to code carts (eg, Banyan Stat Kit). Each office practice should make its own assessment of needs. Prices range from $130 to $995. In addition to the kit, a separate key component is an AED or conventional defibrillator for practices that manage adults who are at risk. Retrospective chart review data demonstrate that dialysis centers have the highest relative incidence of cardiac arrest (0.746 per practice annually), followed closely by cardiology, internal medicine, family medicine, and urgent care centers (0.01 per practice annually). All other medical and dental practices have a low incidence (≤0.002 per practice annually) [7,8].

Basic office emergency protocol

Form a rapid response team

It is vital to have an identified team within your office staff that responds to internal emergencies. A “code” is a rapid response signal that brings help to the patient, irrespective of the severity of the patient’s status. It is used commonly in in-hospital settings and ranges from help for a hypoglycemic
event, syncope, or allergic reaction to a cardiopulmonary arrest. A good rule is to “overtriage” and call a code before making a full assessment. This approach maintains the code team skills, brings immediate help to the patient, and most importantly, prevents further deterioration of the patient’s status.

Establish response team responsibilities

Responsibilities of the response team include:

- Calling for help (911 or the emergency response team for your institution). Have a code team member identified who will have this responsibility.
- Relocating the patient, if possible. Ideally a two- or three-member team allows for a rapid transfer of the patient to an area within the office that has a first aid kit, an oxygen tank, and an AED. If this is impossible because of logistics, have a small cart that can be brought to the patient.
- Incorporating a family member to the team. If the patient’s ability to provide a history is impaired, family members can provide valuable information; often do not get in the way of the team; and most importantly, are intimately aware of the status of the patient, the efforts that are being made to help, and are not surprised as easily when poor outcomes result [9–11].
- Providing basic life support (BLS), if necessary. A brief summary of BLS and the use of an AED are described later in this article.
- Debriefing. After a rapid response team is activated it is critical to learn from the experience and to prepare for future events as best as possible.

General approach to the patient with a witnessed collapse in the office setting

Check the victim for a response. If there is a response (the victim answers or moves), check the level of alertness, chief complaint, blood pressure, pulse, and oxygenation; if a significant abnormality is present, ask any staff member to call for help. Office practices that are not adjacent to a hospital or without established efficient emergency protocols should call 911 immediately. After the emergency medical systems are activated, move the patient to an examination room, ask for the first aid kit or its equivalent, and address abnormal vital signs systematically before moving on (eg, if hypoxic, administer oxygen supplementation; if hypotensive, keep the patient supine and establish an IV with a 250- to 500-mL fluid bolus in 15 minutes if possible). Blood glucose also should be checked early on in the evaluation of the patient. If hypoglycemia is present, provide glucose. A gel is available in most kits if the patient cannot swallow. If an IV is established, provide 1 ampule of d50 (50% dextrose). If no IV is available, 1 to 2 mg of intramuscular Glucagon can be administered, which increases serum glucose levels reliably. Get a targeted history and physical, if possible, and reassess
the patient frequently until help arrives. Significant changes in the pulse that suggest a potential lethal arrhythmia are discussed in further detail later in this article.

If the victim does not respond, shout for help, send someone for help, or if on your own, consider leaving the victim and going for help, or calling 911 or a code team immediately. It is important to remember a major principle of emergency medicine, never create a second victim: the rescuer. Take precautions for infectious disease—use mouth masks and gloves. Return to the patient and open the airway by tilting the head and lifting the chin (Fig. 1). Keeping the airway open, look, listen, and feel for breathing (more than an occasional gasp). Look for chest movements and listen at the victim’s mouth for breath sounds. Feel for air on your cheek (Fig. 2).

If the patient is breathing (other than an occasional gasp), place him/her in the recovery position (on his/her side). The airway of an unconscious victim who is breathing spontaneously is at risk of obstruction by the tongue and from aspiration of mucus and vomit. Placing the victim on his/her side helps to prevent these problems and allows fluid to drain from the mouth. Check for continued breathing.

If the victim is not breathing, do a head tilt and chin lift. Use barrier device precautions—pocket face masks (preferably with a one-way valve) or a bag valve and an oropharyngeal airway should be immediately available; insert it and begin ventilations. Give two effective rescue breaths, each of which makes the chest rise and fall. If there is difficulty achieving an effective breath, recheck the victim’s mouth and remove any obstruction. Recheck that there is adequate head tilt and chin lift. Make up to five attempts to achieve two effective breaths. Even if unsuccessful, move on to assessment of circulation.

Assess the patient for signs of circulation. This includes looking for any movement, including swallowing or breathing, and checking if the carotid pulse is present. Check the pulse for at least 10 seconds; if there is a pulse, continue rescue breathing, if necessary, until the victim starts to breathe on his/her own. About every minute, recheck for signs of circulation; take no more than 10 seconds each time. If the victim starts to breathe on his/her own but remains unconscious, place the victim in the recovery position. Check the victim’s condition and be ready to turn the victim onto his/her back and restart rescue breathing if breathing stops.

Fig. 1. Rescue breathing head tilt and remove foreign body or dentures if present. (Courtesy of the Mayo Clinic, Rochester, MN; with permission.)
If there are no signs of circulation or if you are at all unsure, start chest compression. After 15 compressions, tilt the head, lift the chin, and give two effective breaths; continue compressions and breaths in a ratio of 15:2 until help arrives (Fig. 3).

The tachycardias

Ventricular fibrillation and ventricular tachycardia

Usually, ventricular tachycardia (VT) and ventricular fibrillation (VF) are caused by acute coronary syndromes that lead to ischemic areas of myocardium. Other nonischemic causes are evolution from stable to unstable VT, and untreated premature ventricular complexes with R-on-T phenomenon. Multiple drugs, or electrolyte or acid–base abnormalities can prolong the relative refractory period and cause VT or VF. Finally, primary or secondary QT prolongation are well-known reasons for the development of these arrhythmias. Initially, the goal is to resuscitate the patient by focusing on the airway, breathing, and circulation and performing rapid defibrillation. Box 2 describes the ECG criteria of VF and pulseless VT. Figs. 4 and 5 provide rhythm strips on both.
Automated external defibrillators

The importance of early defibrillation in the treatment of sudden cardiac death cannot be overstated. American Heart Association guidelines seek to ensure that a defibrillator reaches the victim at the earliest appropriate opportunity; hence, the rationale for phoning first or calling a “code” that brings a defibrillator to the patient’s side. This is to ensure that the health care staff does not become so consumed with providing CPR that it persists far too long before summoning the emergency system. Therefore, the initial call for help in the office after assessing unresponsiveness should result in
someone arriving with a defibrillator—in any of its forms—in-hospital code response team, outpatient clinic on-site emergency code cart, or out-of-hospital emergency medical services (EMS) unit.

An AED is a device that incorporates a rhythm–analysis system and a shock-advisory system for victims of cardiac arrest. The AED advises a shock, and the operator must take the final action to deliver the shock. The International Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [12] conclude that early CPR is the best treatment for cardiac arrest until the arrival of an AED and advanced cardiac life support care. Early CPR can prevent ventricular fibrillation from deteriorating to asystole, may increase the chance of successful defibrillation, and contributes to the preservation of heart and brain function. For victims of sudden cardiac arrest that is due to VF or pulseless VT, the single greatest determinant of survival is the time from collapse to defibrillation. A survival rate as high as 90%—among victims of witnessed VF cardiac arrest—has been reported when defibrillation is achieved within the first minute of collapse. These survival rates for out of hospital cardiac arrest have been reported in cardiac rehabilitation programs that were equipped with defibrillators. Survival rates decline 7% to 10% with every minute that defibrillation is delayed, such that a victim of cardiac arrest without defibrillation beyond 12 minutes has only a 2% to 5% chance of survival [12].

The most important reason to use an AED is its ease, because there is no need to learn what a shockable rhythm looks like, no need to adjust Joules or worry about a monophasic or biphasic mode. Additionally, most AEDs remind you to continue CPR if the rhythm is not shockable.

Operating an automated external defibrillator.

1. Power on.
2. Attach electrode pads.
3. Press “Analyze.”

Do this up to three times if advised. Repeat cycles of three shocks and 1 minute of CPR until “no shock indicated” message is displayed. After “no shock indicated” messages, check pulse. If no pulse, perform CPR for 1 minute and analyze again. Continue same cycle until help arrives.

Fig. 5. Ventricular tachycardia. (Courtesy of the Mayo Clinic, Rochester, MN; with permission.)
Although these steps are followed with all AEDs, there are variations in models that change how the steps are performed. Because of this, it is important to train staff on the specific model that they will be using.

Maintaining an automated external defibrillator. Maintaining AEDs appropriately is vital to ensure a continuous state of readiness. Most malfunctions in AEDs are due to improper maintenance or battery failure. To limit potential problems, manufacturers have developed AEDs that perform automatic self-testing, which saves time, improves testing consistency, and minimizes unnecessary battery expenditure. Battery options now include a rechargeable lead acid battery and a high-capacity, extended shelf-life lithium sulfate battery that needs no recharging and no maintenance. Maintenance checklists provide for a standardized inspection and should be used to ensure that the AEDs are kept in a state of readiness. Inspections and checklists help to identify and prevent deficiencies by providing a uniform way to inspect devices, and by increasing the user’s familiarity with the equipment.

AEDs are part of the basic life support course for health care providers; they allow for nonphysician staff to deliver the most important life saving measure without having to interpret a rhythm. In the author’s opinion, all office-based practices that see adult patients who are at risk for sudden cardiac death should have one readily available.

Conventional/defibrillator/monitor. If an AED is not available and a conventional/defibrillator/monitor is, then:

1. Power on monitor and defibrillator (could require one or two controls).
2. Attach three-lead monitor cable, display rhythm through quick-look sternal-apex paddles.
3. Assess for a shockable rhythm by viewing monitor display (there should be no CPR or other patient manipulation during rhythm assessment).
4. Charge to 200 J, 300 J, or 360 J monophasic or clinically equivalent biphasic for shocks 1, 2, and 3.
5. Shock (“Clear!”) up to three times if a shockable rhythm is present, following the same assess, charge, and shock sequence.

After three shocks or after any non-VF/VT rhythm on monitor, check pulse. If no pulse, perform CPR for 1 minute and analyze again. Continue same cycle until help arrives.

Tachycardias with a pulse

If a patient has a pulse, measure blood pressure and assess the cardiac rhythm and rate. Common tachycardias that are encountered in an office-based setting and are capable of producing hemodynamic instability include supraventricular tachycardia (SVT), atrial fibrillation (AF), and atrial flutter.
Paroxysmal supraventricular tachycardia

In paroxysmal SVT (PSVT) there is a reentry phenomenon where impulses arise and recycle repeatedly in the atrioventricular (AV) node because of areas of unidirectional block in the Purkinje fibers. Frequently, there also is an accessory conduction pathway in most healthy people in whom many factors can provoke the paroxysm (eg, caffeine, hypoxia, cigarettes, stress, anxiety, sleep deprivation, numerous medications). PSVT occurs with an increased frequency in patients who have chronic obstructive pulmonary disease, coronary artery disease, or congestive heart failure. Usually, the clinical manifestations are palpitations that are felt by the patient at the onset. Other symptoms include lightheadedness, anxiety, and an otherwise uncomfortable feeling. They also can experience poor exercise tolerance with high ventricular rates (150–180 bpm). Syncope, altered mental status, and cardiac arrest are uncommon. When a patient loses a pulse and has this organized rhythm by definition, it is labeled a pulseless electrical activity, which is a rare event. Box 3 describes the ECG criteria, and Fig. 6 demonstrates a sample rhythm strip.

When a patient presents to the office with these symptoms and PSVT is recognized, an attempt at a therapeutic/diagnostic maneuver with vagal stimulation is reasonable. Some patients will have experience with specific maneuvers that have been useful for them and some will have tried it before seeking care. Therefore, it is important to review briefly the past medical history, medications, and experience with vagal maneuvers. If this has not been performed and the patient is stable, it is reasonable to attempt such an intervention. Waxman and colleagues [13] showed that carotid massage and the Valsalva maneuver (VM) are the most powerful physical maneuver for termination of PSVT, and it has a significant vagal effect on AV node conduction. In their study, maneuvers that reflexly increase vagal tone were used to terminate the tachycardia in 68 consecutive patients who had PSVT. Fifty-seven episodes were terminated with carotid sinus massage (CSM), 5 were terminated with VMs, and 6 were terminated pharmacologically with phenylephrine. Luber and colleagues [14], in a retrospective chart

*Box 3. Supraventricular tachycardia: defining ECG features*

- **Rate**: exceeds upper limit of sinus tachycardia (>120 bpm); seldom <150 bpm; up to 250 bpm
- **Rhythm**: regular
- **PR**: seldom seen because rapid rate causes P wave loss in preceding T waves or because the origin is low in the atrium
- **QRS complex**: normal, narrow (usually 0.10 s)

Key: regular, narrow-complex tachycardia without P waves.
review of 111 patients who presented to the ED with PSVT, reported that 26 patients (23%) spontaneously converted to having a normal sinus rhythm (NSR) without therapy while waiting. In 44 patients who received vagal maneuvers, 22 had successful conversion. The rest had pharmacologic conversion (41 of 48 patients with adenosine). Overall, 71% of patients were discharged from the ED. Only 4% had a recurrence of their SVT after discharge.

There is debate on what maneuver should be attempted first. Lim and colleagues [15] conducted a prospective, randomized study of CSM versus VM for PSVT in the ED in 148 patients. The VM converted PSVT to NSR in 19% of the cases, whereas CSM converted PSVT to NSR in 10% of cases. A combination of both had an overall success rate of 27.7%. CSM is performed gently, yet firmly, for 2 minutes to abort the episode. In patients with a history of carotid artery disease or who are at risk for a stroke, a VM or other maneuvers are preferable. If these maneuvers are unsuccessful, administration of adenosine, 6 mg IV for adults, is a reasonable option, although this therapy is contraindicated in patients who have asthma or reactive airway disease and in patients with heart rates above 200 bpm, because of the possibility of degeneration into a ventricular tachycardia (when the blocking capabilities of the AV node are inhibited). Management of these patients is done best in an emergency setting. Occasionally, patients who have hemodynamic instability from PSVT require direct current (DC) cardioversion, which can be performed by paramedics at arrival or by trained physicians.

Atrial fibrillation and atrial flutter with rapid ventricular response

The hallmark of these arrhythmias is atrial impulses that run faster than the sinus node impulses. In AF, impulses take multiple, chaotic, random pathways through the atria. Atrial flutter is characterized by impulses that take a circular course around the atria and set up the flutter waves. The mechanism of impulse formation is a reentry pathway that leads to tachycardia.

Patients who present with new-onset AF need an evaluation for the underlying cause as well as management of the arrhythmia. Etiologies of AF include conditions that distend or irritate the musculature of the atria,
such as cardiac ischemia, congestive heart failure, valvular heart disease, cardiomyopathies, and recent cardiac surgery. Pulmonary etiologies, such as chronic obstructive pulmonary disease, pulmonary embolism, and pulmonary hypertension, also can cause AF. Systemic etiologies, including hyperthyroidism and medication use, should be considered. There is a subset of patients in whom the work-up for an underlying cause is unrevealing, and lone AF is diagnosed [16]. Table 1 describes the ECG criteria for AF and atrial flutter. Figs. 7 and 8 are examples of these rhythms.

Signs and symptoms are functions of the rate of ventricular response to atrial fibrillatory waves. AF with rapid ventricular response (RVR) symptoms are mostly due to a loss of “atrial kick” that may lead to a decrease in cardiac output and decreased coronary perfusion; patients often complain of dyspnea at rest or on exertion. Other patients perceive the irregular rhythm as “palpitations” and even can be asymptomatic, especially at rest. Other presenting symptoms include chest pain or discomfort, fatigue, or lightheadedness.

There are four essential question to ask when faced with a patient who has a fibrillation/flutter and an RVR:

1. Is the patient clinically unstable? (Does the patient have ischemic chest pain? Is the patient hypotensive?)
2. Is the cardiac function impaired? (Does the patient have a known depressed left ventricular function?)
3. Is Wolff-Parkinson-White syndrome (WPW) present? (Is there a history of WPW, and are earlier ECGs available for comparison?)
4. Have the symptoms been present for less than 48 hours or longer than 48 hours?

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Atrial fibrillation and flutter</th>
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<td><strong>Fibrillation</strong></td>
<td><strong>Flutter</strong></td>
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| **Rate:** wide ranging ventricular response to atrial rate of 300–400 bpm | • Atrial rate 220–350 bpm  
• Ventricular response is a function of AV node block or conduction of atrial impulses  
• Ventricular response rarely >150–180 bpm because of AV node conduction limits |
| **Rhythm:** classic “irregularly irregular” | • Regular  
• Set ratio to atrial rhythm (eg, 2:1 or 3:1) |
| **P waves:** chaotic atrial fibrillatory waves | • No true P waves seen  
• Flutter waves in “sawtooth” pattern |
| **PR interval:** cannot be measured | • Cannot be measured |
| **QRS:** remains δ 0.10–0.12 s unless QRS complex distorted by fibrillation/flutter waves or by conduction defects through ventricles |
Is the patient unstable?

One must treat unstable patients urgently; call 911, administer oxygen, place the patient in the supine position, and obtain a blood pressure. If hypotension is present, an IV should be placed. In addition, have someone obtain the office AED or a conventional monitor/defibrillator. While resuscitative efforts get underway, attempt to get a targeted history. If an AED is available, power on, analyze, and if advised to shock, clear everyone and do so. AF or flutter are not perceived by the AED as a shockable rhythm; furthermore, the AED does not measure or interpret other physiologic parameters. In this scenario, one can wait for EMS to arrive or if AF or flutter with RVR is suspected, switch to a conventional defibrillator/monitor. After an IV is established and an RVR is confirmed (usually >150 bpm), power on the defibrillator, set on synchronize, place the energy at 100 J, clear all staff, deliver a shock, and verify response. If no response, increase subsequently to 150 J, 200 J, 300 J, and 360 J. Usually, after the first or second attempt the patient converts to a sinus tachycardia or NSR. In the infrequent event that the patient loses a pulse, start CPR and provide support as described in the pulseless rhythm section of this article.

Is cardiac function impaired?

If a patient does not have hypotension but is experiencing significant chest pain/pressure or dyspnea, provide 325 mg aspirin, establish IV access, provide a 250-mL bolus, and simultaneously review the patient’s medications. If help has not arrived, control the heart rate. This can be achieved effectively by administering metoprolol; a reasonable starting dose is 5 mg IV. Diltiazem is an excellent alternative and is one of the most frequently used in the ED, mostly because of the lack of frequent contraindications.
that are associated with β-blockers and the high rate of success with a first
dose. A ventricular rate of less than 130 bpm is ideal; however, this should
not be the target in the office setting because hypotension after drug admin-
istration is not uncommon. Obviously, this scenario is handled best in the
ED. Finally, if a patient develops severe chest pain or has significant ST seg-
ment abnormalities, it is not unreasonable to perform synchronized cardio-
version while awaiting transport, especially if the patient has had symptoms
for less than 48 hours. If the patient exhibits signs of congestive heart failure
or has known poor left ventricular function, synchronized cardioversion or
amiodarone is the best therapeutic intervention.

Is Wolff-Parkinson-White syndrome present?
AF or flutter in combination with WPW (Fig. 9) deserves special mention
because of the potential for development of an unstable tachycardia if
treated inappropriately. Patients who have AF and WPW should not receive
conventional rate control with β-blockers, calcium channel blockers, digi-
oxin, or adenosine because of the potential inhibition of the AV node
that might be inhibiting chaotic atrial rates. If this AV node is inhibited,
the potential for a reentry of an atrial impulse through an accessory path-
way increases dramatically, and VT or VF might result.

Have symptoms been present for less that 48 hours?
In the stable patient who has minimal complaints and an RVR (usually
<130 bpm), administration of a β-blocker or calcium channel blocker is
preferable if available. There is no urgency to intervene in this case; patients
can be referred by ambulance to the ED where further management will be
performed.

Young patients who have normal or near normal hearts and a history of
recurrent paroxysmal AF (PAF) have been treated in Italy with a “pill in the
pocket approach” [17]. This approach is reserved for patients who have con-
verted in the hospital or the ED with a single dose of oral propafenone or
flecainide with success and without side effects (78% of all patients who
had PAF). This out-of-hospital self-administration was done after the onset

Fig. 9. Wolff-Parkinson-White syndrome: normal sinus rhythm with delta wave (arrow) notching of positive upstroke of QRS complex. (Courtesy of the Mayo Clinic, Rochester, MN; with permission.)
of heart palpitations in 210 patients (mean age ± SD, 59 ± 11 years). During a mean follow-up of 15 ± 5 months, 165 patients (79%) had a total of 618 episodes of arrhythmia; of those episodes, 569 (92%) were treated 36 ± 93 minutes after the onset of symptoms. Treatment was successful in 534 episodes (94%); the time to resolution of symptoms was 113 ± 84 minutes. Among the 165 patients who had recurrences, the drug was effective during all of the arrhythmic episodes in 139 patients (84%). Adverse effects were reported during one or more arrhythmic episodes by 12 patients (7%), including atrial flutter at a rapid ventricular rate in 1 patient and “noncardiac” side effects in 11 patients. The numbers of monthly visits to the ED and hospitalizations were significantly lower during follow-up than during the year before the target episode (P < .001 for both comparisons). The investigators concluded that in a selected, risk-stratified population of patients who had recurrent AF, pill-in-the-pocket treatment is feasible and safe, with a high rate of compliance by patients, a low rate of adverse events, and a marked reduction in ED visits and hospital admissions. No study in the United States has evaluated such an approach.

Other infrequent tachycardias, such as multifocal atrial tachycardia and junctional tachycardia, are the result of emphysema, cardiomyopathy, asthma, or pulmonary hypertension. Usually, they are managed by controlling the underlying exacerbation of the primary disease and not the actual arrhythmia (eg, management with β-agonists of asthma or emphysema and not the arrhythmia) [12].

The bradycardias

Bradycardia is a common finding during the clinical evaluation of healthy and ill patients; its exact incidence is unknown. It is found commonly in two settings. Often, bradycardia is detected incidentally in the asymptomatic patient during a routine history and physical examination that is performed during an office visit. A rhythm strip or ECG that is obtained for other purposes may bring the bradycardia to the physician’s attention. Additionally, bradycardia may be detected in the symptomatic patient. Often, symptoms and signs are nonspecific and include dizziness, fatigue, weakness, or shortness of breath; however, symptoms can present dramatically with syncope, hypotension, or a depressed level of consciousness. Establishing a correlation between signs and symptoms and simultaneous rhythm abnormalities or a change in rhythm is key to diagnosis [18]. The following discussion highlights ways to identify specific types of bradycardias, including sinus bradycardia, AV block, junctional rhythm (Fig. 12), and idioventricular rhythm (Fig. 13). Even bradycardias that typically portend a benign prognosis can produce profound signs and symptoms that should be managed more aggressively [19].

There are many potential etiologies to consider in the bradycardic patient, both intrinsic and extrinsic. Acutely, it important to consider lifethreatening and potentially treatable causes, before proceeding with a
Box 4. Causes of bradycardia

*Life-threatening*
- Myocardial ischemia
- Electrolyte imbalance
  - Hyperkalemia
  - Hypokalemia
- Medication toxicity
  - β-Blocker
  - Calcium channel blocker
  - Digoxin
- Sick sinus syndrome

*Other\(^a\)*

**Intrinsic**
- Idiopathic degeneration (aging)
- Infiltrative diseases
  - Amyloidosis
  - Sarcoidosis
  - Hemochromatosis
- Collagen vascular diseases
  - Systemic lupus erythematosus
  - Scleroderma
  - Rheumatoid arthritis
- Myotonic muscular dystrophy
- Surgical trauma
  - Valve replacement
  - Correction of congenital heart disease
  - Heart transplantation
- Infectious diseases
  - Lyme disease
  - Chagas disease
  - Endocarditis

**Extrinsic**
- Autonomically-mediated syndromes
  - Neurocardiogenic syncope
  - Carotid-sinus hypersensitivity
  - Situational disturbances
    - Coughing
    - Micturition
    - Defecation
    - Vomiting
• Drugs
  o Antiarrhythmic agents
  o Clonidine
• Hypothyroidism
• Hypothermia
• Neurologic disorders

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more comprehensive evaluation (Box 4). Causes to consider immediately include myocardial ischemia or infarction, electrolyte imbalance, medication toxicity, or sick sinus syndrome. If any of these etiologies is suspected, transport to the nearest ED should be considered strongly. A crucial electrolyte imbalance to consider is hyperkalemia, especially in a patient who has known renal failure [20]. If hyperkalemia is a suspected cause in a patient who has profound bradycardia, obtaining confirmation with ECG, administering albuterol by metered dose inhaler or nebulizer, establishing IV access, and treating the patient with calcium chloride and sodium bicarbonate before transport to the nearest ED can be life saving. Additional medication effects to consider include β-blocker, calcium channel blocker, or digoxin toxicity. Box 4 also highlights other causes of bradycardia to consider after the emergency has been handled and the patient is in the hospital or returns to the office for follow up.

As outlined in the ACLS algorithm for bradycardia, management depends on the severity of the patient’s symptoms and the nature of the arrhythmia. For patients who have significant signs and symptoms that are due to the bradycardia, such as depressed consciousness or hypotension, immediate treatment is indicated. Atropine is the recommended initial pharmacologic intervention before transport to the nearest ED. If the patient fails to respond to atropine, transcutaneous pacing is appropriate. In the patient who does not have significant signs and symptoms and in whom the bradycardia was discovered incidentally, management depends on the propensity of the bradycardia to degenerate to complete heart block. In Mobitz type II second-degree AV block (Fig. 10) and third-degree heart block (Fig. 11), the bradycardia is likely to progress; permanent transvenous pacing may be indicated, even in the asymptomatic patient. Placement of pacing electrodes in preparation for transcutaneous pacing before transport to the ED is appropriate [12]. In patients who have more benign bradycardias without significant signs and symptoms, continued elective evaluation in the office setting is appropriate. This can include sinus bradycardia and first-degree AV block. ECG criteria for common bradycardias that are encountered in the outpatient setting are described in Box 5.
Transfer considerations

Before consideration of transfer, initial efforts should focus on stabilizing the patient as described above. It should be recognized early in the process that the unstable patient is best cared for in a hospital-based setting that is equipped to handle the full extent of a life-threatening emergency. After it becomes clear that a patient may have a life-threatening condition (including most sustained arrhythmias), arrangements should be made to transfer. Two important principles in the transfer of patients include stabilizing the patient’s condition before transfer and arranging for an appropriate mode of transport.

Stabilizing the patient’s condition before transfer

All available resources should be used to initiate resuscitation of a patient. This can include the use of an AED, placement of a peripheral line, and administration of oxygen (in some settings ventilating with a bag-valve mask or even endotracheal intubation may be needed). The extent of the resuscitation depends on the available equipment and personnel. It is important for the office practice to be aware of the ACLS algorithms and be prepared to initiate steps should a patient present in an unstable fashion.

Appropriate mode of transport

When a patient is transported to another facility, it is a time of potential vulnerability. Therefore, the provider must arrange for a level of transport that is equipped to care for the patient’s presenting complaints and any likely complications or deteriorations [21]. Usual options to the office practice include
private automobile with the patient driving, private automobile with a family member or companion driving, basic life support (capable of CPR), and advanced life support (capable of providing ACLS level of care). It may be appropriate to consider helicopter transport when longer transports with a high level of care are necessary. The mode of transport often is looked at simply as a mechanism to transport the patient to a hospital. Sometimes overlooked is the ability of a skilled paramedic to provide life-saving care. Because of this a patient who has unstable vital signs, including hypotension (systolic blood pressure less than 90 mm Hg), tachy- or bradyarrhythmias, or ongoing chest pain, generally should be transferred by ACLS-capable providers. These include emergency medical team (EMT) paramedics and flight nurses. Patients who have the above conditions are not ideal candidates for BLS or private automobile transports because—even if they are feeling well—they may deteriorate in route. Patients who may be able to be transported by private automobile (although they should not drive) include the healthy patient in whom a nonlife-threatening arrhythmia has resolved. Whenever there is doubt, it is in the patient’s interest to err on the side of caution and to provide the highest level of transport that is available.

Much concern has focused on Emergency Medical Treatment and Active Labor Act (EMTALA) regulations surrounding transfers. The EMTALA was enacted in 1986 as part of the consolidated Omnibus Reconciliation Act of 1985, in response to concerns that some EDs were refusing to treat uninsured patients or were transferring them inappropriately to other facilities [22]. Since its inception, the law has expanded in scope and has had varying interpretations [23]. Although the legislation offers some helpful tips about the transfer of patients, it does not apply to patients who are cared for in the office/outpatient setting. A potential exception to that may be a clinic practice that is on hospital grounds and meets the definition

Fig. 13. Idioventricular rhythm. (Courtesy of the Mayo Clinic, Rochester, MN; with permission.)

Fig. 12. Junctional rhythm. (Courtesy of the Mayo Clinic, Rochester, MN; with permission.)
of a “dedicated ED.” This is a facility that is licensed by the state as an ED and is held out to the public as a place that provides care for emergency medical conditions, and cares for patients on an urgent basis without a previously scheduled appointment. In this setting, the key elements of transferring are described in the EMTALA (Box 6). Compliance with these basic

Box 6. Emergency Medical Treatment and Active Labor Act transfer requirements

- Transferring facility provides medical treatment within its capacity to minimize risk of transfer to the patient.
- An accepting physician has been contacted at the receiving hospital and has agreed to treat the patient.
- The receiving hospital has space and qualified personnel available to treat the individual.
- Copies of medical records related to the reason for transfer are sent with the patient.
- Transfer is effected through qualified personnel and equipment as required, including appropriate life support measures.

Box 5. Bradycardia ECG criteria

Non–life-threatening
- **Sinus bradycardia:** sinus rhythm with a heart rate ≤60 bpm
- **First-degree AV block:** prolonged PR interval (>200 ms)
- **Second-degree AV block:** Mobitz type I (progressively lengthening PR interval until the P wave is not followed by a QRS complex)

Life-threatening
- **Mobitz type II:** nonconducted P wave with a constant PR interval before and after the nonconducted beats (see Fig. 10)
- **Third-degree AV block:** complete failure of AV nodal conduction with P waves completely dissociated from the QRS complexes (see Fig. 11)
- **Junctional rhythm:** slow regular rhythm with QRS complexes <120 ms. Rate is usually 40–60 bpm. Retrograde P waves may or may not be present. Typically is an escape mechanism during high-grade AV block (see Fig. 12).
- **Idioventricular rhythm:** slow regular rhythm with QRS complexes >120 ms. Rate is usually 30–40 bpm. P waves, if present, bear no relation to QRS complexes. Also an escape mechanism during high-grade AV block (see Fig. 13).
tenants represents good patient care and professionalism. It is particularly important to contact the receiving facility to be sure that there is an accepting physician, and to send the patient with all appropriate documentation of testing and care that has been rendered.

References