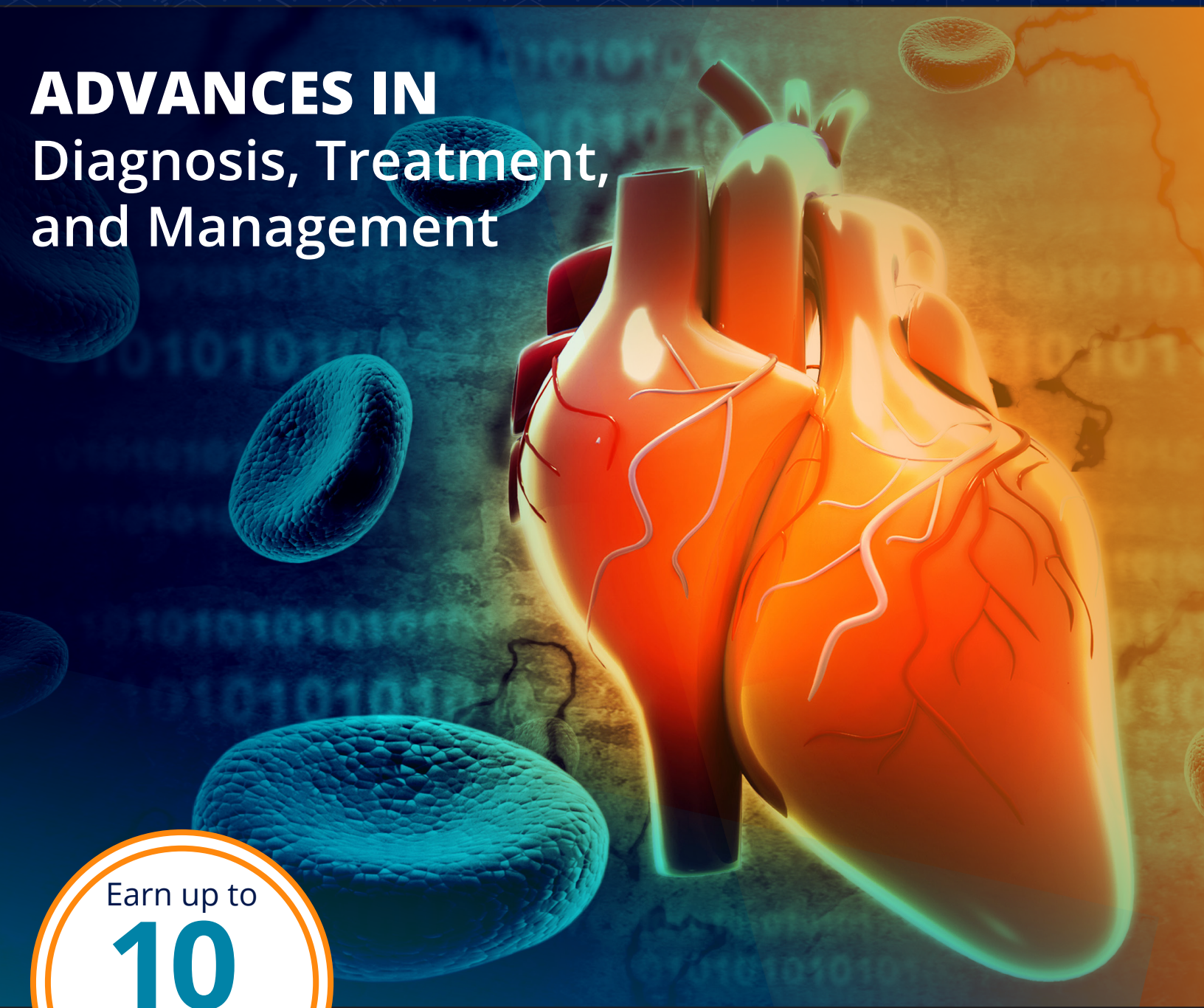
STEMI WATCH 2018



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ACTIVITY OBJECTIVES

After reading *STEMI Watch: Diagnosis and Beyond*, the participant will be able to:

1. Discuss current scientific research and data regarding the diagnosis of ST segment elevation myocardial infarction (STEMI).
2. Discuss the pathogenesis and treatment of STEMI.
3. Cite new information regarding new drugs for STEMI and new uses for traditional drugs.
4. Identify ancillary issues of importance for healthcare providers who treat STEMI patients.
5. Discuss advances in STEMI treatment.

Physicians and nurses participate in this CME/CE activity by reading the articles, using the provided references for further research, and studying the relevant questions at the end of the book. Participants will then be directed to a website, where they will complete an online assessment to show what they've learned. They must score 100 on the assessment in order to complete the activity, but they are allowed to answer the questions multiple times if needed. After they have successfully completed the assessment, they will be directed to an online activity evaluation form. Once that is submitted, they will receive their credit letter.

TARGET AUDIENCE

This activity is intended for cardiologists, coronary care nurses and staff, emergency medicine physicians and nurses, emergency medical service providers, ECG staff, percutaneous coronary intervention center staff, and freestanding emergency department staff.

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TABLE OF CONTENTS

STEMI WATCH 2018

Module 1: Advanced Cardiac Life Support

Advanced Cardiac Life Support Updates

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Introduction

Heart disease is the leading cause of death worldwide. Death can be reduced when practitioners provide evidence-based care, especially during acute resuscitations. The American Heart Association (AHA) Advanced Cardiac Life Support (ACLS) guidelines are recognized as the authoritative reference for acute cardiopulmonary resuscitation. Emergency medicine providers should be thoroughly familiar with the AHA ACLS guidelines.

ACLS guidelines were established first in 1974 and have been periodically updated since.[1] In 2015, the AHA introduced a web-based format for the latest guideline update, which allows them to be updated continuously.[2] This article will discuss the newest AHA evidence-based guidelines and recommendations. The major updates include an emphasis on the quality of cardiopulmonary resuscitation (CPR), a change in layperson CPR, changes in cardiac arrest medication, and renewed emphasis on post-cardiac arrest management.

Quality of Cardiopulmonary Resuscitation

Chest Compressions

Uninterrupted chest compressions and defibrillation remain the most important components for resuscitation of cardiac arrest. For that reason, the AHA has reorganized the importance of sequential tasks for cardiac arrest from "ABC" to "CAB." Coronary perfusion pressure (CPP) is dependent on continuous chest compressions at an adequate rate and depth. CPP is one of the most important factors to achieving a return of spontaneous circulation (ROSC). Chest compressions are the first priority for both adult and pediatric patients, and interruptions in compressions must be minimized before and after defibrillation, or if halted to perform other interventions.

Inadequate compression rate and depth are the most common opportunities for improvement in resuscitation.[2] The 2010 guidelines stated that a rate of > 100 was appropriate. However, the 2015 guidelines have instituted an upper limit of 120 compressions per minute, making the new guideline compression rate 100 to 120 compressions per minute.[3] An upper limit was instituted after a large study demonstrated that compression rate above 120/minute was associated with an increased likelihood of inadequate compression depth, thus decreasing coronary blood flow and cardiac output.[4] The minimum adult chest compression depth is 2 inches (5 cm), established by the 2010 guidelines. The 2015 guidelines, similarly, have added a maximum depth of 2.4 inches (6 cm) because data suggested potential for significant injury from excessive chest compression depth.[3,5]

Compression Devices

Given the increased popularity of automatic compression devices, the 2015 ACLS guidelines also address their use. Automatic compression devices have been shown to have no greater success rate than manual CPR; but conversely, there are also no worsened outcomes when compression devices are used by trained personnel. In some cases, using these devices can increase safety and allow providers to focus on other elements of resuscitation, but manual chest compressions remain the preferred delivery method. Some newer studies are showing that both rate and compression depth are improved during manual chest compressions with feedback devices, and this may be the best option for CPR.[6,7,8]

Airway/Breathing

Chest compressions should not be interrupted during the early phase of resuscitation to place an advanced airway. Placement of a definitive airway is a frequent cause of paused compressions, with data showing that 25% of all interruptions in chest compressions were due to placement of an advanced airway.[9] The 2015 guidelines state that it is reasonable to delay positive-pressure ventilation if compressions are being performed and passive oxygenation is applied. AHA guidelines from 2010 recommend supraglottic airway devices as an appropriate alternative to endotracheal intubation during the initial phase of resuscitation, and this recommendation remains in the most recent update.[10]

If an advanced airway is indicated (patient is difficult to bag mask ventilate or more than three rounds of CPR have been completed with passive oxygenation), cricoid pressure

Perimortem cesarean delivery should be performed by an appropriately trained provider within four minutes of the onset of cardiac arrest if ROSC is not achieved.[38]

Cardiac Arrest Associated With Pulmonary Embolism

In patients with a confirmed PE leading to cardiac arrest, thrombolysis, surgical embolectomy, and mechanical embolectomy are reasonable emergency treatment options (class IIa recommendation). There are not enough data to recommend one method over the other. In patients with suspected pulmonary embolism, thrombolysis may be considered (class IIb recommendation). Surgical embolectomy and mechanical embolectomy have insufficient evidence in cases of suspected PE.[38]

Cardiac or Respiratory Arrest Due to Opioid Overdose

It is recommended to give intranasal (IN), intramuscular (IM), or IV naloxone to cardiac arrest patients suspected of having an opioid overdose. EMS providers should not delay transport of the patient while waiting for a response to naloxone (class I recommendation). Any patient who receives naloxone should be transported to a facility with advanced healthcare services (class I recommendation).[38]

Cardiac Arrest Due to Local Anesthetic Poisoning

A systematic review of human case reports showed that the majority of patients in cardiac arrest due to local anesthetic toxicity who received IV lipid emulsion had significant improvement.[44] This was specifically true for bupivacaine toxicity where intravenous lipid emulsion increased the rate of ROSC, increased BP during hypotension, resolved dysrhythmias, and improved mental status. The most common dose consisted of 20% emulsion of long-chain triglycerides given in an initial bolus of 1.5 mL/kg lean body mass over one minute, followed by an infusion of 0.25 mL/kg per minute for 30-60 minutes. Because of such findings, the AHA has made giving IV lipid emulsion in patients with local anesthetic systemic toxicity a class IIb recommendation.[38,45]

Cardiac Arrest During Percutaneous Coronary Intervention

The 2015 AHA guidelines for cardiac arrest during PCI state that ECPR may be a viable option in patients suffering a cardiac arrest during PCI because the arrest was due to a potentially reversible cause (class IIb recommendation). The AHA recommends implementation and adherence to institutional guidelines for the appropriate selection of patients for mechanical support devices to ensure that these devices are used as a bridge to recovery, surgery or transplant, or other treatment (class I recommendation).[38]

Table 2. The Hs and Ts: Common Causes of Cardiac Arrest

6 Hs	6 Ts
• Hyper-/Hypokalemia • Hypoxia • Hypovolemia • Hypothermia • Hydrogen ion (acidosis) • Hypoglycemia	• Toxins • Tamponade (cardiac) • Thrombosis (coronary) • Thromboemoblism • Tension pneumothorax • Trauma

Possible Future Updates

Pulseless Electrical Activity

For years, the AHA guidelines have emphasized, and continue to emphasize, the "Hs and Ts" when it comes to PEA management (*see Table 2*); however, recent studies are showing improved outcomes from a more simplified approach.[46,47,48,49] In these studies, other possible algorithms for wide vs. narrow QRS PEA are evaluated. (*See Figure 1.*)

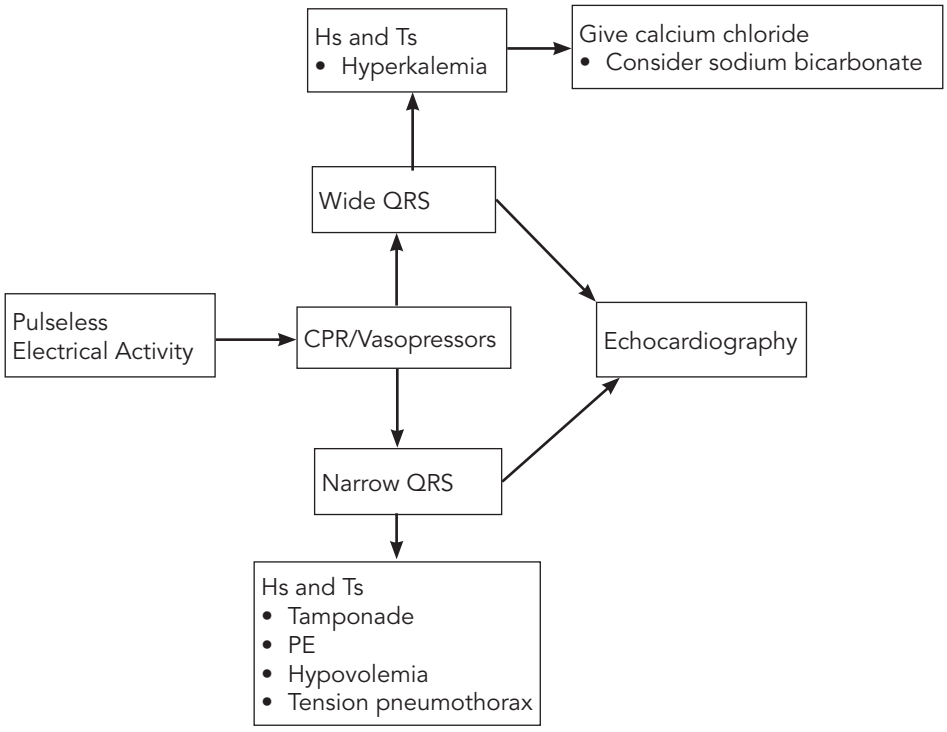
Initially, management is the same: CPR and vasopressors, specifically epinephrine. However, depending on the width of the QRS complex, management changes. For wide complex PEA, these studies suggest giving calcium chloride initially, and consider sodium bicarbonate. This will help if PEA is due to hyperkalemia or sodium channel blockers. For narrow complex PEA, initial action is fluid resuscitation and then consider PE, hypovolemia, tamponade, or tension pneumothorax. Interventions for both wide and narrow complex PEA should be followed by bedside echocardiography.

If there is no cardiac activity after these initial steps, these protocols recommend terminating CPR. If a large right ventricle and a small left ventricle are found, consider TPA for PE, needle decompression for pneumothorax, or disconnect the ventilator for possible hyperinflation. A small right ventricle with a small left ventricle could indicate hypovolemia or distributive shock; fluids, transfusions (if bleeding), and vasopressors should be initiated. With a pericardial effusion, tamponade physiology should be considered, and pericardiocentesis should be performed. Finally, if a hypocontractile left ventricle is seen on echocardiogram, vasopressors and inotropic agents should be continued with consideration for mechanical support.[46,47,48,49]

Double Sequential Defibrillation

Double sequential defibrillation (DSD) has been studied since 1986. The first study was conducted on dogs and showed promising outcomes.[50] In this study, the authors showed that providing two defibrillations delivered one after the other using different pad placements to generate nonidentical current pathways through the heart was able

Figure 1. Pulseless Electrical Activity Management



to terminate experimental ventricular fibrillation in these animals with both reduced peak voltage and total energy. See Figure 2 for suggested double sequential defibrillation pad placement.

The first human study was conducted in 1994. In this study, five patients with refractory ventricular fibrillation received seven to 20 shocks at 200-360 joules before receiving DSD. In each of these refractory cases, the patient was defibrillated on the first attempt of DSD. The investigators used the anterior-posterior and apex-sternum orientations to deliver the shocks.[51] Recognizing the limitation of this observational study, it showed that DSD does have potential in patients with refractory ventricular fibrillation.

In 2015, an EMS service published a study with 10 patients with refractory ventricular fibrillation after at least five failed attempts at single defibrillation with epinephrine and an antiarrhythmic medication having been delivered. DSD was successful in defibrillating seven patients, and three of those obtained ROSC; however, none of those patients survived.[52]

Although these studies have been limited, they do suggest the ability to terminate refractory ventricular fibrillation using DSD, which may be included in future recommendations as further studies are conducted and evidence is strengthened.

Post-Cardiac Arrest Care

Cardiac arrest management does not end with ROSC. Just as important for patient outcome is the implementation of post-cardiac arrest care to address both the cause of the arrest and the resulting physiologic abnormalities. Post-cardiac arrest syndrome (PCAS) consists of an ischemic/reperfusion response with brain injury and myocardial dysfunction.[53] The majority of post-arrest patients are comatose and unable to provide a history. Good use of available resources, including EMS, family members, and the medical chart, is important. Pertinent factors to document are any known prodromal symptoms, CPR details (witnessed vs. unwitnessed arrest, bystander CPR, length of CPR, and initial rhythm), and any medications administered.[54] Post-arrest management will be discussed by system below. See Table 3 for a systems-based management checklist.

Cardiovascular Care

Coronary Angiography. As soon as possible after ROSC is obtained, a 12-lead electrocardiogram (ECG) should be performed to look for any evidence of ST-segment elevation. This will help determine whether coronary angiography should be performed immediately.[55] (*See Figure 3.*)

Unchanged from previous recommendations, patients with ST-elevation myocardial infarction (STEMI) should undergo emergent coronary angiography and PCI.[55] Thus far, only observational studies have been published concerning post-resuscitation PCI, but these studies show improved survival to hospital discharge and improved neurological outcomes associated with emergent coronary angiography in post-arrest STEMI patients. These interventions are recommended regardless of the patient's mental status or cooling status.

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ill patients is highly controversial, although studies have shown moderate control is superior to tight control.[67] Early hyperglycemia after cardiac arrest has been associated with unfavorable neurologic outcomes in several studies.[53] There is no evidence that a specific blood glucose range has improved outcomes in the post-arrest population, but avoiding extremes on either side is reasonable.[53,55]

Renal Care

Resuscitated patients typically develop severe metabolic and electrolyte abnormalities. This is related to both the period of resuscitation and the ischemia-reperfusion injury in the post-arrest period.[68] All serum electrolytes should be monitored closely, but it seems that potassium and magnesium have the biggest potential for impact.[54] More studies are needed to determine whether attempting to correct pH with bicarbonate or ventilator management is helpful. Secondary renal injuries also are common. These typically are transient, but care must be taken to avoid nephrotoxic drugs if acute kidney injury is present.[54,68]

Infectious Disease Care

Infection and sepsis may play a role in both pre- and post-arrest periods. Studies have found that pneumonia is present in close to half of OHCA patients undergoing TTM.[69,70] In addition, bacteremia was seen in 38% of patients presenting with OHCA in one study.[71] Although the AHA has not made any recommendations on the use of empiric broad-spectrum antibiotics in the post-arrest period, it may be an area of interest in the future. Consideration should be given to obtaining blood cultures on post-arrest patients. Aggressive respiratory care to prevent pneumonia also should be initiated in the ED and continued in the critical care setting, including elevation of the head of the bed and standard pulmonary toilet.[72]

Hematologic Care

Blood counts and coagulation studies should be checked in all post-arrest patients. Anemia may have contributed to arrest and should be corrected with blood products as needed, along with attempts to determine the source of blood loss.[54] Patients with active bleeding that is non-compressible in nature should not undergo therapeutic hypothermia, as hypothermia causes coagulopathy. This is most prevalent at temperatures below 35° C, when clotting and platelet function are impaired.[73] If the patient is already cooled, it may be appropriate to raise the temperature to 36° C, while maintaining a TTM protocol to prevent hyperthermia.[54,55]

Conclusion

Resuscitation during a cardiac arrest and post-resuscitation management are complex processes. The recommendations and algorithms developed by the AHA are meant to serve as a guide to ensure the best possible patient outcomes based on the most up-to-date evidence. Although there are opportunities for further research, evidence-based management of the cardiac arrest patient is most likely to improve patient survival and neurologically favorable outcomes.

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Module 2: Bradycardia

Evaluation of Bradycardia in the Emergency Department

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Dr. Murano and Dr. Stark report no financial relationships with companies related to the field of study covered by this CME activity.

My last shift was hectic, as usual. Urgent care sent over a patient for a minor complaint because his pulse was 40 beats per minute. He was otherwise healthy, and although not at first glance an "athlete," he was fit and worked a physically demanding job. An ECG confirmed sinus bradycardia. Several different staff — triage, techs, and nurses — repeatedly asked me to treat his asymptomatic benign bradycardia.

But bradycardia is not always benign. In the Northeast, I saw several patients in whom "asymptomatic" bradycardia was the key to Lyme disease. In others, bradycardia is a marker of very high risk among patients with acute myocardial infarction.

This paper is a great review of some basics in physiology and treatment. It will be a refresher on the identification of serious arrhythmias. There are also important reminders — digoxin, calcium channel blocker or beta-blocker overdoses, and Lyme disease. It reminds us that vital signs are indeed vital, at least until we determine they can be ignored.

Introduction

Bradycardia is defined as a heart rate less than 60 beats per minute. From the healthy athlete to the most ill patient, bradycardia is a sign commonly seen in the emergency department. Bradycardia may represent a normal or incidental finding or it may be the result of a conduction abnormality that is detected during the evaluation of a symptomatic patient. The wide variety of etiologies responsible for bradycardia makes it, on its own, a typically nonspecific sign. Both intrinsic and extrinsic factors can be responsible for this often asymptomatic finding. It is crucial for the emergency physician to be able to evaluate efficiently and treat its more malignant expressions. In this article, the normal anatomy and pathophysiology of bradydysrhythmias in adults will be reviewed, followed by the latest recommendations in evaluation and management.

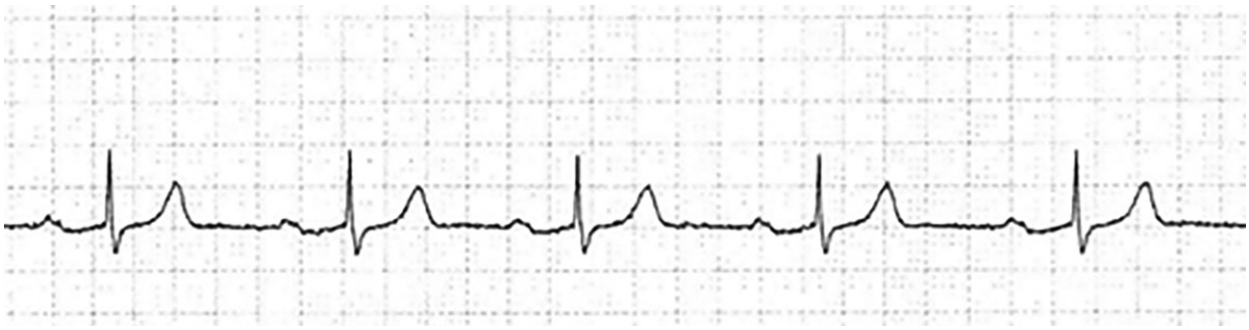
Physiology

Heart rates of less than 60 beats per minute do not necessarily mean that there is an underlying pathologic state. In fact, there are several instances in which bradycardia is a normal physiologic finding. For example, there is variation in the range of resting heart rates in otherwise healthy individuals. One study demonstrated heart rates as low as 46 beats per minute in men and 51 beats per minute in women in healthy individuals without cardiovascular disease.[1] It also established that heart rates decrease during sleep. Resting bradycardia in trained athletes is considered a normal finding. Initially thought to be due to a decrease in intrinsic heart rate or alteration of the autonomic balance, bradycardia in athletes now is believed to be a multifactorial mechanism. Remodeling of the sinoatrial (SA) node, genetic factors, cardiac hypertrophy, and baroreflex alteration all have been hypothesized to play a role in modifying an athlete's resting heart rate.[2]

The anatomy and circulation to the conducting system is important in the understanding of the pathophysiology behind bradydysrhythmias. In the normal conduction of the heart, impulses spontaneously arise from the SA node and travel through the right atrium to the atrioventricular (AV) node and then to the bundle of His in the ventricular septum. The bundle of His bifurcates into two groups of Purkinje fibers, known as the left bundle and the right bundle, which rapidly conduct impulses that initiate ventricular contraction. The heart has a complex network of sympathetic and parasympathetic innervation that affects the automaticity of the SA and AV nodes. Parasympathetic tone decreases SA node automaticity, slows AV node conduction, and slows the heart rate; sympathetic tone has the opposite effect.

The coronary arteries supply blood to the conduction system, and occlusions may lead to conduction disturbances in those anatomic areas supplied by the affected vessel. The blood supply to the SA node is provided either via the sinus-node artery, which is a branch of the right coronary artery (RCA) in approximately 65% of the population or a branch of the circumflex coronary artery in approximately 35% of the population.[3,4] The blood supply to the AV node is via the AV nodal artery — a branch of the proximal posterior descending artery, which arises from the RCA in approximately 90% of the population and from the circumflex coronary artery in 10%.[3,4] The RCA also supplies blood

Figure 2. First-Degree AV Block



There is 1:1 AV conduction, and the PR interval is constant and > 0.2 seconds.

accompanying QRS complex. Although the PR intervals are constant, they may be normal or prolonged. Since Mobitz type II blocks most commonly are due to pathology below the AV node, the QRS complex typically is wide. However, the QRS complex may be narrow if the pathology is located in the AV node. There may be more than one blocked atrial impulse before conduction occurs. The term "high-grade" AV block refers to Mobitz type II blocks that have more than one atrial impulse that is not conducted, leading to more than one consecutive P wave before a QRS complex. When there are two consecutive P waves before a QRS complex appears, it is described as a 2:1 block; three consecutive impulses before a QRS complex is described as a 3:1 block, and so forth.

Third-degree AV block also is termed complete heart block because AV conduction is absent and atrial and ventricular activity are independent of one another. The P-P intervals remain constant, as do the R-R intervals, but there is no relationship between the two. (*See Figure 3.*) In this circumstance, a junctional escape pacemaker takes over and leads to ventricular contractions and, thus, determines the ventricular rate. The escape pacemaker is at a rate slower than that of the atrial pace. When the third-degree block is at the AV node, the QRS complex typically is narrow and the ventricular escape rhythm typically is between 40 to 60 beats per minute. Third-degree AV blocks that are at the infranodal level have ventricular escape rhythms slower than 40 beats per minute. When the third-degree AV block is at the bundle of His, the QRS complexes can be either narrow or wide. However, third-degree AV blocks below the bifurcation of the bundle of His produce QRS complexes that are wide.

In the setting of acute myocardial infarction (AMI), AV nodal block has significant prognostic and therapeutic significance. In AMI, first-degree AV block may occur in up to 13% of patients.[12] First-degree AV block may progress to a high-degree AV block (second- or third-degree AV block) in AMI patients. The mortality rate is two to three times greater in patients who have a high-degree AV block in the AMI setting. AV nodal block in the setting of AMI frequently will present within the first 24 hours of the ischemic event and is transient the majority of the time. Third-degree AV block may occur in up to 19% of AMI patients.[12] Approximately 8% of patients with inferior wall MI have third-degree AV block at the AV node.[13]

Table 3. Summary of Atrioventricular Node Blocks

Block Type	Description
First degree	• PR interval is > 200 msec, constant 1:1 AV conduction
Second degree	
Mobitz type I (Wenckebach)	• Progressive increase in PR interval until conduction failure results in P wave without a corresponding QRS complex
Mobitz type II	• PR intervals are constant • PR intervals may be normal or prolonged • Abrupt conduction failure results in a P wave • without a corresponding QRS complex • May have > 1 atrial impulse not conducted, • resulting in multiple P waves before QRS • complex appears
Third degree	• AV conduction is absent • Atrial and ventricular activity are independent • of one another • P-P intervals are constant • R-R intervals are constant

Junctional and Idioventricular Escape Rhythms

There are auxiliary pacemakers located in the atria, AV junction, and the ventricles. When there is a failure of the normal impulse formation or conduction, these pacemakers

CME/CE QUESTIONS

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CME/CE Questions

1. Which of the following rhythms is characterized by a progressively lengthening PR interval until there is a dropped QRS complex?
   a. Sinus bradycardia
   b. First-degree AV block
   c. Second-degree AV block Mobitz type I*
   d. Second-degree AV block Mobitz type II
   e. Third-degree AV block

2. Which of the following rhythms is characterized by a transient failure of an impulse to be conducted to the atrial myocardium, resulting in a pause between the P waves, with no P wave or QRS complex, that equals the length of two or more P-P intervals?
   a. Sinus arrest
   b. Sinus block*
   c. Sinus bradycardia
   d. Second-degree AV block Mobitz type I
   e. Second-degree AV block Mobitz type II

3. What is the first-line treatment for patients with bradycardia who show signs of hypoperfusion?
   a. Atropine*
   b. Epinephrine
   c. Dopamine
   d. Transcutaneous pacemaker
   e. Transvenous pacemaker

4. What are the two preferred central venous access locations for transvenous pacemaker placement?
   a. Left internal jugular and left subclavian veins
   b. Left internal jugular and right subclavian veins
   c. Right internal jugular and right femoral veins
   d. Right internal jugular and right subclavian veins
   e. Right internal jugular and left subclavian veins*

5. Which of the following medications is appropriate for patients with unstable bradycardia who do not respond to atropine?
   a. Dopamine or epinephrine*
   b. Calcium
   c. Digoxin immune fragment antigen-binding
   d. Glucagon
   e. Insulin

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Module 3: STEMI Treatment

ABSTRACT & COMMENTARY

Complete vs. Infarct-related, Artery-only Revascularization in STEMI

By Jeffrey Zimmet, MD, PhD, Associate Professor of Medicine, University of California, San Francisco; Director, Cardiac Catheterization Laboratory, San Francisco VA Medical Center

Dr. Zimmet reports no financial relationships relevant to this field of study.

SYNOPSIS: This trial randomized patients with ST elevation myocardial infarction and at least one non-infarct artery with angiographically significant stenosis to either fractional flow-reserve-guided complete revascularization by percutaneous coronary intervention or to no revascularization of noninfarct arteries. The primary composite endpoint was significantly lower in the complete revascularization group, driven by a reduction in later revascularization.

SOURCE: Smits PC, Abdel-Wahab M, Neumann FJ, et al. Fractional flow reserve-guided multivessel angioplasty in myocardial infarction. *N Engl J Med* 2017;376:1234-1244.

Up to half of patients presenting with ST elevation myocardial infarction (STEMI) have angiographically significant stenoses in other vessels at the time of intervention. Guidelines from the American College of Cardiology, the American Heart Association, and the European Society of Cardiology recommend treatment of the infarct-related artery only. Several clinical trials over the past several years have challenged this concept. The PRAMI trial from 2013 randomized 465 patients with STEMI to infarct artery-only percutaneous coronary intervention (PCI) vs. "preventive" PCI of other angiographically significant lesions, and reported a significant benefit in terms of recurrent angina, nonfatal myocardial infarction (MI), and cardiac death. The DANAMI 3 trial, published in 2015, randomized 627 STEMI patients to either no further invasive treatment (beyond PCI of the infarct artery) or to complete fractional flow-reserve (FFR)-guided revascularization prior to discharge. This trial demonstrated a benefit in terms of major adverse cardiac events, but this was driven primarily by a reduction in the need for subsequent ischemia-driven revascularization. The CvLPRIT trial, published in 2015, enrolled 295 patients and showed a benefit in terms of the composite endpoint, but was too small to show a significant decrease in any individual components of this endpoint.

Smits et al conducted the largest trial to date looking at this question. Between mid-2011 and late 2015, 885 patients presenting with STEMI and multivessel disease were enrolled at 24 centers in Europe and Asia. Patients were randomized 1:2 to either FFR-guided complete revascularization or to treatment of the infarct-related artery only. In contrast to earlier studies, all angiographic stenoses of 50% or more were interrogated by FFR in all patients in both groups. However, intervention was performed only in the complete revascularization group, and the patient and outpatient providers were kept blinded to the FFR results (but not to the angiography results). The primary endpoint was a 12-month composite of all-cause death, any revascularization, and cerebrovascular events. Elective, clinically indicated PCI procedures performed within 45 days of the STEMI presentation were excluded (primarily lesions believed clearly to be angiographically severe at the time of the index procedure).

FFR was successfully performed in all but 18 of 885 patients. Among the complete revascularization patients, 158 of 292 (54.1%) exhibited at least one vessel with FFR < 0.80 and underwent PCI. More than 80% of these PCIs were performed during the index procedure, while the remainder were completed within three days. Among the infarct artery-only patients, 275 of 575 (47.8%) exhibited FFR-positive lesions, and 59 patients underwent staged elective revascularization within 45 days based on clinical and angiographic data; only 44 of these had positive FFRs.

At one year, 23 patients in the complete revascularization group (7.8%) and 121 patients in the infarct artery-only group (20.5%) experienced major adverse cardiac events (MACE, hazard ratio [HR], 0.35; $P < 0.001$). This difference was driven almost entirely by a higher incidence of revascularization in the infarct-only group. Mortality was not significantly different between groups. Although there was a trend toward a reduction in MI in the complete revascularization group, this did not meet statistical significance (HR, 0.50; 95% confidence interval, 0.22-1.13; $P = 0.10$). Among subsequent revascularization procedures, approximately one-third were for unstable angina.

The authors concluded that for patients presenting with STEMI and multi-vessel disease, FFR-guided complete revascularization in the acute setting resulted in a decrease in MACE, driven primarily by a reduction in subsequent revascularization procedures.

STEMI WATCH 2018

Module 4: ECG Reviews

How Would You 'Date' This Infarct?

By Ken Grauer, MD, Professor Emeritus in Family Medicine, College of Medicine, University of Florida

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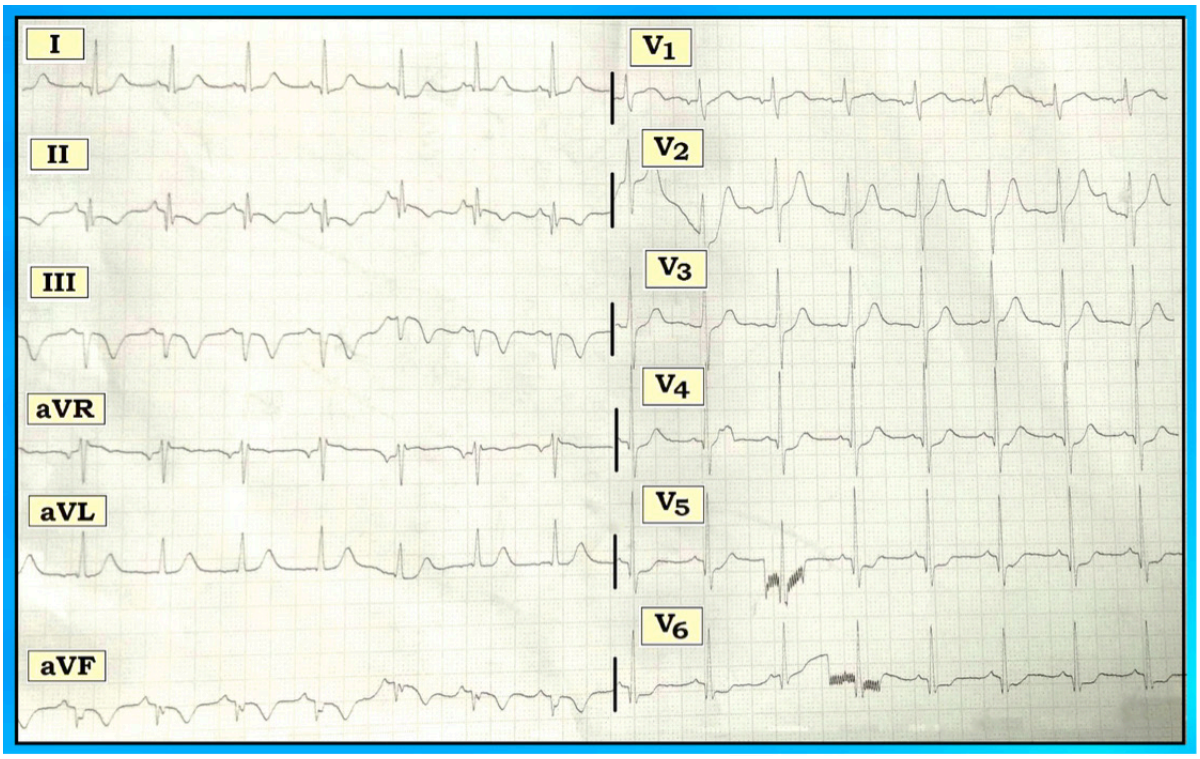

The ECG in the figure above was obtained from a 48-year-old man who presented to the ED with a three-day history of chest discomfort. It clearly shows evidence of infarction. How would you "date" the infarct?

The rhythm is fairly regular at 85-90 beats/minute. Upright sinus P waves are seen in lead II. The PR, QRS, and QT intervals are normal. The axis is leftward, but not by enough to qualify as left anterior hemiblock (i.e., the net QRS deflection in lead II is not predominantly negative). There is no chamber enlargement.

Q waves are present in multiple leads. Small, narrow (probably septal) q waves are seen in leads I and aVL. The Q wave in lead II is narrow but deep. Although there may be a tiny initial positive deflection (r wave) in at least some of the beats in lead III, the QRS complex is notched (fragmented), and clearly all negative in lead aVF. This defines the QRS in lead aVF as a QS complex. Thus, the overall appearance of the QRS in the inferior leads strongly suggests there has been inferior infarction. Finally, there are Q waves in leads V4, V5, and V6. Although narrow, the Q waves in leads V5 and V6 are somewhat deeper than expected to be simple "septal" q waves.

R wave progression is not normal in the chest leads. Normally, there should be a predominant negative deflection (S wave) in lead V1, with the area of "transition" (where R wave amplitude supersedes S wave depth) not occurring until after V2 or V3. Instead, R wave amplitude in lead V1 already equals S wave depth in this lead, with prominent R waves seen by lead V2. Although a number of entities may produce a disproportionately tall R wave in lead V1, recent posterior infarction should be at the top of this list. ST-T waves show numerous abnormalities. Although ST segments are not elevated, there is ST segment coving with fairly deep T wave inversion in each of the inferior leads. Additionally, there is 1-2 mm of ST depression in leads V3-V6, with more subtle ST-T wave abnormalities in leads I and aVL. Finally, the T wave in lead V2 looks prominent, and appears to be disproportionately tall. ST-T wave changes of "reperfusion" typically manifest the appearance seen here; that is, with no more than minimal residual ST segment elevation, and with