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Optimizing Survival Outcomes For Adult Patients With Nontraumatic Cardiac Arrest

Abstract

Patient survival after cardiac arrest can be improved significantly with prompt and effective resuscitative care. This systematic review analyzes the basic life support factors that improve survival outcome, including chest compression technique and rapid defibrillation of shockable rhythms. For patients who are successfully resuscitated, comprehensive postresuscitation care is essential. Targeted temperature management is recommended for all patients who remain comatose, in addition to careful monitoring of oxygenation, hemodynamics, and cardiac rhythm. Management of cardiac arrest in circumstances such as pregnancy, pulmonary embolism, opioid overdose and other toxicologic causes, hypothermia, and coronary ischemia are also reviewed.

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CME Objectives

Upon completion of this article, you should be able to:

1. Describe the elements of high-quality basic life support.
2. Discuss the evidentiary basis and current guidelines for advanced life support interventions.
3. Describe essential considerations in postresuscitation care following restoration of spontaneous circulation.
4. List modifications to standard resuscitation protocols that may be considered in special resuscitation situations.

Prior to beginning this activity, see "Physician CME Information" on the back page.

Case Presentations

Shortly after starting your night shift, you get STAT paged to bed 34. You rush to the room and find the nurses giving chest compressions. You wrack your brain to remember sign-out – wasn't this the man who came in with chest pain, no ECG changes, but a concerning history? He was stable, just waiting on an inpatient bed! Not anymore, you sigh. You hold compressions to take a look at the monitor, and see V-fib. "Charge to 200 joules," you say. "Clear!" calls the tech. The patient jumps with the force of the electricity. The nurse resumes compressions, but a few moments later, the patient moans, moving his hand to his chest. As you hold on compressions and continue to stabilize the patient, you wonder if he would be a good candidate to go to the cath lab . . .

Shortly afterward, you get a call from paramedics about a young man who collapsed suddenly while dancing at a nightclub. "He's in full cardiac arrest," they say, and you rush back to the critical care bay. You arrive in the resuscitation bay just as EMS arrives. "It's V-fib," the paramedic tells you. "He got 1 shock on the way over, and we're due for a rhythm check now. EMS had them doing CPR at the scene, and they were doing a pretty good job. It's only been about 10 minutes since the 911 call– the club is right around the corner from here." You pause CPR and glance at the monitor. It's still V-fib, so you deliver a shock at 200 joules and one of the nurses takes over CPR. "Great compressions," you tell him, and you mean it - just the right depth and rate, and no pauses for nonsense. "I've got access, Doc," says the technician. "Perfect," you respond, "let's give a milligram of epi." You sound like you're in control, but your mind is racing Why V-fib in a seemingly healthy young man?

Introduction

Cardiac arrest refers to the abrupt cessation of effective mechanical function of the heart. It may be caused by a variety of cardiac and noncardiac diseases.[1] There are more than half a million adult cardiac arrests in the United States each year, with approximately 325,000 outside the hospital and 200,000 in the hospital. There is a slight male predominance, with 57% of cases occurring in men.[2] Cardiac arrest occurs in all age groups, though incidence increases with age.[2]

Survival to hospital discharge occurs in approximately 10% of out-of-hospital arrest cases overall, though survival rates are more than 30% for bystander-witnessed cases.[3] In-hospital arrest survival is approximately 20%,[4,5] though this estimate varies substantially by hospital[6] and time of arrest.[7] The presenting cardiac rhythm is an important predictor of outcome in cardiac arrest. The first documented rhythm is ventricular fibrillation (VF) in 17% of cases, ventricular tachycardia (VT) in 7%, pulseless electrical activity (PEA) in 37%, and asystole in 39%.[8] Survival to hospital discharge is better overall for shockable rhythms, with more than 33% of patients surviving VF/VT arrests compared to approximately 10% for PEA and asystole.[8,9]

Critical Appraisal Of The Literature

The body of literature on cardiac arrest is vast, with more than 40,000 articles in PubMed. Fortunately, the International Liaison Committee on Resuscitation (ILCOR) regularly reviews the literature and synthesizes it into practice guidelines for its member organizations, including the American Heart Association (AHA). The AHA publishes guideline updates every 5 years, and these are the "gold standard" in cardiac arrest science. The guidelines summarize clinical resuscitation protocols and their evidentiary basis, as well as noting areas where scientific evidence is lacking. The 2015 updates, published on October 15, 2015 in the journal *Circulation*, served as the starting point for this review, with additional targeted literature searches performed to address specific clinical questions.

The 2015 AHA guidelines emphasize the importance of immediate, continuous, high-quality cardiopulmonary resuscitation (CPR) and early defibrillation, while recognizing the relative paucity of evidence supporting more "advanced" interventions. Key points from the 2015 guidelines include the following:[10]

- Prehospital care
  - Expansion of recommendations for emergency medical services (EMS) dispatchers to instruct bystanders in compression-only CPR for all cases of suspected cardiac arrest
- Basic life support (BLS)
  - Continuation of emphasis on all aspects of CPR quality, especially rate and depth of chest compressions, as these are the most common errors made by CPR providers
  - Addition of an upper limit for chest compression rate (100-120/min)
  - Addition of an upper limit for chest compression depth (2-2.4 in)
  - Emphasis on decreasing preshock pauses in chest compressions
  - Addition of a specific goal for chest compression fraction (> 60% of total resuscitation time)
- Advanced/hospital-based life support
  - Continued recommendation that healthcare workers provide both compressions and breaths for all arrest victims, regardless of etiology
  - Addition of specific respiratory rate after intubation (1 breath/6 sec)
  - Recommendation to maximize inspired oxygen during resuscitation
  - Recognition of the potential utility of ultra-

sound, with the caveat that it must not interfere with CPR or defibrillation
    - Removal of vasopressin from arrest algorithms
    - Addition of rhythm-specific guidelines for timing of epinephrine
        - Shockable rhythm: after defibrillation, as this is the first priority
        - Nonshockable rhythm: as soon as possible
- Postarrest care
    - Recommendation to consider percutaneous coronary interventions (PCI) in all successfully resuscitated arrest patients with suspected coronary syndromes
    - Recommendation for targeted temperature management at 32°C-36°C for at least 24 hours postarrest
    - Recommendation to avoid prognostication of neurologic outcome until 72 hours after return of spontaneous circulation

Etiology And Pathophysiology

Both inside and outside the hospital, sudden cardiac arrest in adults is attributable to primary cardiac disease in approximately 60% to 80% of cases.[11,12] Among cardiac causes of arrest, acute coronary syndromes (ACS) are the most common, accounting for 35% to 40% of cases.[11,13] In cardiac arrest due to coronary syndromes, VF or VT is most often the initial rhythm,[2] caused by the direct effects of myocardial ischemia on electrical conduction. Less commonly, a massive myocardial infarction may cause severe contractile dysfunction leading to insufficient cardiac output to generate a pulse, despite preserved electrical conduction. Under these circumstances, a PEA arrest ensues. In addition to ACS, other cardiac diseases can also lead to VF/VT arrest. Patients with valvular lesions may develop ischemia leading to a rhythm disturbance. Patients with cardiomyopathies and electrical conduction abnormalities are at increased risk for developing potentially lethal ventricular dysrhythmias.

Among noncardiac causes of arrest, hypoxia is the most common, accounting for approximately 20% of cases.[11,13] Other frequent causes include hypovolemia, pulmonary embolism, pericardial tamponade, and sepsis.[11] PEA is the rhythm most often associated with arrest due to noncardiac causes, and has been linked to a wide range of systemic derangements.[2]

Asystole is the end-stage rhythm for all forms of cardiac arrest, regardless of etiology. Every rhythm will eventually deteriorate to asystole if resuscitation is unsuccessful.

Differential Diagnosis

As a diagnosis, cardiac arrest does not have a wide differential diagnosis, as it should be clear when a patient is in arrest: they are unresponsive, with apnea and no palpable carotid pulse. However, carotid pulse assessment is not always accurate,[14-17] and agonal gasps may be confused with normal respiration,[18] potentially clouding the diagnostic picture. AHA guidance has focused on minimizing "type II" error; ie, failing to diagnose arrest when it is actually present. It is critical to avoid this type of error in cardiac arrest, as arrest patients have virtually no chance of survival without prompt resuscitative care, meaning that false-negative diagnoses are universally fatal. For this reason, cardiac arrest has become a diagnosis of assumption: one should assume it to be present in appropriate patients until proven otherwise. To this end, pulse checks were completely removed from lay rescuer protocols in 2000, and have been de-emphasized for health professionals as well.[19] Similarly, EMS dispatchers have been trained to encourage bystander CPR for all cases of suspected cardiac arrest rather than confirming arrest before beginning resuscitation.[20]

Some experts have questioned this approach, expressing concern about the dangers of unnecessary CPR. While it is true that CPR is associated with high rates of skeletal chest wall injuries, these injuries rarely lead to internal organ damage, and are not life-threatening.[21] Furthermore, many experts believe that patients who receive needless CPR are likely to revive in response to this stimulus, enabling rescuers to stop before inflicting injury.[19] While CPR is not entirely benign, it is clear that the consequences of withholding needed CPR are far more dire than those of performing it unnecessarily, and in the face of clinical uncertainty, it is safest to assume cardiac arrest is present.

Once arrest has been diagnosed, knowledge of the underlying cause (which can have a broader differential diagnosis) may be helpful for optimizing treatment. Arrest etiology is strongly correlated with cardiac rhythm. VF/VT arrests are usually attributable to primary cardiac etiologies, most commonly coronary syndromes. PEA has a much broader differential diagnosis, often referred to as the "Hs and the Ts."[22] **(See Table 1.)**

Formerly known as *electromechanical dissociation*, PEA was once thought to be the result of uncoupling of electrical conduction from mechanical contraction in the heart. However, studies demonstrating

Table 1. The "Hs And Ts" In Pulseless Electrical Activity

Hs	Ts
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade, pericardial
Hydrogen ion (acidosis)	Toxins
Hyper/hypokalemia	Thrombosis, coronary
Hypothermia	Thrombosis, pulmonary

preservation of myocardial contraction during PEA challenged this belief, suggesting that, in many cases, PEA is indistinguishable from severe cardiogenic shock.[23,24] In some causes of PEA, such as hypovolemia, tamponade, and tension pneumothorax, myocardial contraction may not be impaired at all. In these cases, cardiac output is decreased because of either inadequate circulating volume or obstruction of cardiac filling. In other cases, myocardial contraction may be reduced or entirely absent. Regardless of the cause of PEA, successful resuscitation depends on effective treatment of the underlying cause, in addition to standard resuscitative measures.

Prehospital Care

Two-thirds of cardiac arrests occur outside of the hospital, and effective prehospital care is crucial to the survival of arrest patients. The difference in survival rates between witnessed and unwitnessed out-of-hospital arrests underscores the pivotal role that prehospital care plays in determining arrest outcome. BLS is the cornerstone of out-of-hospital cardiac arrest care, and it includes rapid recognition of arrest, activation of EMS, performance of CPR, and defibrillation when indicated for shockable rhythms.[25] These interventions are prioritized because of their critical and time-sensitive nature; CPR provides perfusion to vital organs pending more definitive intervention, and defibrillation restores normal electrical conduction in cases of VF/VT. Recognition of the lifesaving potential of BLS has led to widespread training of laypersons in CPR and increasing availability of automated external defibrillators (AEDs) in public places.

In a registry of out-of-hospital cardiac arrests, only 33% of patients received bystander CPR, and only 4% received bystander defibrillation before EMS arrival. Arrests that were witnessed and/or occurred in public places were more commonly treated with bystander CPR. There were significant racial disparities in bystander CPR performance, with 40% of whites receiving bystander CPR compared to 33% to 34% of blacks and Hispanics. Unsurprisingly, the subgroup with the highest survival rate (30%) consisted of patients with witnessed arrests, bystander CPR, and shockable rhythms.[12]

Current guidelines emphasize the importance of ensuring that EMS dispatchers recognize all possible cardiac arrest cases, and provide bystander instruction in compression-only CPR. Bystanders commonly misreport agonal gasps in arrest patients as signs of life, thereby impeding the initiation of CPR instruction.[18] Rather than asking whether breathing is present in an unconscious patient, EMS dispatchers are now instructed to ask whether breathing is "normal," as unconscious patients with abnormal breathing are likely to be in cardiac arrest.[25] This protocol ensures that dispatchers identify the vast majority of cardiac arrest cases and provide appropriate bystander CPR instruction.[26] Dispatcher instruction increases rates of bystander CPR performance, thereby increasing likelihood of survival.[27]

While any CPR is better than no CPR, the quality of CPR is a critical determinant of survival outcome in cardiac arrest. Though many health professionals think of CPR as a simple skill, inadequate CPR performance is extremely common. It is essential that EMS personnel provide high-quality CPR for all arrest patients for the full duration of prehospital care. There are 5 core elements of CPR quality identified by the AHA:[28]

1. Minimize interruptions in chest compressions
2. Maintain appropriate compression rate
3. Ensure adequate compression depth
4. Allow full chest wall recoil between compressions
5. Avoid excessive ventilation

Emergency Department Evaluation

For all cardiac arrest patients, the first priority is initiation or continuation of BLS, including high-quality CPR and defibrillation, if indicated. Gathering clinical information is a secondary consideration. A single team member should be designated to obtain history so that the rest of the team can focus on resuscitation. For out-of-hospital cardiac arrests, history will usually be obtained from EMS, though family may sometimes be available. Essential history for all arrests includes:

- Time of arrest (or time of EMS call if arrest time is unknown)
- Whether arrest was witnessed
- Whether bystander CPR was performed
- First cardiac rhythm
- Treatments prior to arrival
- Availability of airway and/or vascular access

While additional history is always beneficial, it is particularly important in cases of PEA arrest. As discussed earlier, PEA is associated with a wide variety of systemic and noncardiac etiologies, in contrast to VF/VT, which is typically caused by cardiac events. The broader differential diagnosis that must be considered in PEA necessitates a thorough history for elucidating the cause and directing treatment. Given the emergent nature of cardiac arrest, it may be unrealistic to collect this information immediately, so the input of EMS, family, friends, and bystanders should be sought as soon as feasible. Important additional history includes:

- Events immediately prior to the arrest
- Recent illnesses or symptoms
- Pertinent past medical and surgical history
- Daily medications, and whether overdose or medication error are suspected

- Allergies to medications or severe environmental allergies
- Tobacco, alcohol, and/or illicit drug use

In cardiac arrest, the only essential physical examination maneuvers are pulse check and observation for signs of life, which occur during regularly scheduled pauses in CPR for cardiac rhythm assessment. In the absence of a pulse and/or signs of life, cardiac arrest may be assumed to be present, and resuscitation should proceed accordingly. That said, some physical examination findings may help elucidate the underlying cause of cardiac arrest, and may help direct treatment, particularly in PEA. **(See Table 2.)**

Diagnostic Studies

Cardiac arrest is a clinical diagnosis. No diagnostic tests are indicated for confirmation, and treatment should be initiated based on clinical assessment. The only laboratory study that should be routinely considered is a fingerstick blood glucose test to determine whether hypoglycemia or hyperglycemia may be implicated in the etiology of arrest. While hypokalemia or hyperkalemia may be implicated in cardiac arrest, this should be suspected based on clinical history and electrocardiogram (ECG) morphology and treated empirically, rather than relying on laboratory confirmation. Additional laboratory and imaging studies should be deferred until spontaneous circulation has been restored.

Table 2. Physical Examination Findings And Possibly Related Causes Of Arrest

Physical Examination Finding	Possibly Related Cause of Arrest
Unilaterally absent breath sounds	Tension pneumothorax
Neck vein distension	Pericardial tamponade, tension pneumothorax
Mottled, cold extremities	Hypoperfusion states (ie, shock of any etiology)
Dry mucous membranes, skin tenting	Hypovolemia
Evidence of trauma or gastrointestinal bleeding	Hemorrhage
Unilateral leg swelling	Pulmonary embolism
Signs of recent surgery	Pulmonary embolism, hemorrhage
Gravid uterus	Pulmonary embolism, hypoxia related to seizure/eclampsia
Needle track marks, pill bottles/paraphernalia	Toxic ingestion/overdose
Arteriovenous fistula/graft, dialysis catheter	Hyperkalemia, acidosis

Ultrasound has become a popular modality of assessment in cardiac arrest, mostly for diagnosing potential causes of arrest and assessing myocardial contractility. A small trial recently demonstrated that ultrasound could successfully identify reversible causes of arrest, but survival outcomes were the same regardless of the use of ultrasound.[29] Other studies have shown that ultrasound is particularly useful in PEA for diagnosing treatable conditions such as hypovolemia and pericardial tamponade, but only case reports suggest that this information changes outcome.[30,31] There is evidence that ventricular standstill on ultrasound is a useful prognostic indicator, as it is strongly associated with failure of resuscitation and may be helpful in making decisions regarding termination of care.[32,33] The 2015 AHA guidelines state that ultrasound may be used as an adjunct to standard assessment methods, but they explicitly stipulated that it may not be permitted to interfere with CPR, defibrillation, or other essential interventions.[22] We recommend using diagnostic ultrasound in the assessment of cardiac arrest patients, but limiting it to 10-second intervals during rhythm checks so as not to interfere with CPR. Chest compressions offer unequivocally proven survival benefit, while diagnostic ultrasound, though potentially useful in some cases, does not.

Treatment

Basic Life Support

High-quality BLS is the cornerstone of resuscitation both inside and outside of the hospital. For out-of-hospital arrests, the emergency department (ED) care team should ensure seamless continuation of prehospital resuscitation, minimizing interruptions in CPR and ensuring appropriate timing of rhythm checks and defibrillation. For in-hospital arrests, the team should promptly initiate resuscitation. In all cases, care should be taken to optimize CPR quality, as this is a critical determinant of survival. CPR quality metrics include the following goals and guidelines:

Chest Compression Rate Goal: 100-120 compressions/min

Inappropriate compression rate is among the most common resuscitation errors.[10] The 2010 AHA guidelines required compression rates of > 100/min, but new data suggest that excessively fast rates are just as detrimental to survival as slow rates.[34,35] The 2015 guidelines have, therefore, added an upper limit on compression rate, and providers should now deliver 100 to 120 compressions/min. From a physiologic standpoint, this makes sense, as fast rates decrease filling time, thereby reducing cardiac output. Furthermore, there is evidence that compression depth suffers at rates > 120 compressions/min,[35] thereby

reducing stroke volume. Rates of 100-120 compressions/min maximize cardiac output, ultimately improving likelihood of survival.

Chest Compression Depth Goal: 2 to 2.4 inches

Incorrect compression depth is another very common resuscitation error.[10] The 2010 AHA guidelines required a compression depth of > 2 inches, but the current 2015 guidelines have also added an upper limit of 2.4 inches.[22] There is clear evidence that compression depths > 2 inches produce better survival outcomes than shallower ones.[36,37] In a study of more than 9000 patients, optimal survival was observed with compression depths of 1.6 to 2.2 inches, with no benefit or detriment for greater depths.[37] However, another study demonstrated that injuries are more likely to occur with compression depths > 2.4 inches.[38] While the injuries observed were not life-threatening, the lack of clear benefit to deep compressions combined with the possibility of harm led to the establishment of an upper boundary on depth. In actual practice, excessively shallow compressions are far more common than excessively deep ones,[37] so ensuring adequate depth is still the primary consideration when monitoring CPR quality.

Chest Compression Fraction Goal: > 60% Of Resuscitation Time

CPR continuity is commonly measured as the chest compression fraction (CCF), which refers to the percentage of total arrest time during which compressions are ongoing. There is considerable evidence that CCF correlates with clinical outcome in cardiac arrest patients. One study of patients with shockable rhythms observed the highest rates of survival to hospital discharge among patients with CCF of 60% to 80%,[39] while another study of patients with nonshockable rhythms demonstrated a linear increase in return of spontaneous circulation as CCF increased.[40] Studies specifically examining perishock pauses in CPR have demonstrated an association between shorter pause lengths and improved survival outcome.[41,42] A few studies have shown conflicting results,[43,44] though the preponderance of the evidence suggests that CPR continuity is an important contributor to survival. For this reason, the 2015 AHA guidelines encourage minimizing interruptions in CPR, particularly in the perishock period, and striving for a CCF of at least 60% for all resuscitations.[25]

Allow Full Chest Wall Recoil Between Compressions

There are no human studies on chest wall recoil in cardiac arrest patients, but it makes physiologic sense to avoid leaning on the chest, in order to optimize cardiac filling between compressions, and animal studies show that leaning decreases coronary perfusion pressures. It is therefore recommended to allow full recoil following each compression.[25]

Avoid Excessive Ventilation

There are no human studies regarding the impact of hyperventilation on survival, but there is evidence that this is a common error in resuscitation.[28] Animal literature suggests that positive pressure ventilation may raise intrathoracic pressure, decrease coronary perfusion pressure, impair cardiac output, and negatively impact survival.[45] In light of the adverse physiologic effects of positive pressure ventilation, current guidelines advise limiting respirations to 1 breath every 6 seconds in intubated patients, or 2 breaths following each cycle of 30 compressions in nonintubated patients.[25] Rescuers should only insufflate enough volume to produce minimal chest rise.[28]

Defibrillation

In addition to quality CPR, it is essential that rescuers rapidly defibrillate all patients with shockable rhythms. Delays in defibrillation are associated with lower rates of survival from cardiac arrest.[46] For this reason, the 2015 AHA guidelines recommend that defibrillation be performed as quickly as possible following the identification of a shockable rhythm.[25] The dose-response curve for defibrillation is not known and more data are needed to determine the best means of current delivery to the heart in cardiac arrest. In the absence of this information, current recommendations are to administer the first defibrillation at 200 J if a biphasic defibrillator is used, and to consider this dose or higher (up to the device's maximum energy) for all subsequent shocks. If a monophasic defibrillator is used, the dose of all shocks in cardiac arrest is 360 J.

There has been speculation that a period of CPR prior to defibrillation might improve the metabolic status of the heart, making successful defibrillation more likely. Several high-quality studies have shown no benefit to this approach in terms of survival,[47-49] meaning that there is no reason to delay defibrillation in order to perform CPR beforehand. When a defibrillator is not immediately available, CPR should be initiated pending defibrillation.

In order to ensure rapid defibrillation, it is essential that rescuers quickly identify shockable rhythms. For safety reasons, BLS-level rescuers are required to use automated defibrillators for rhythm identification. Higher-level rescuers are permitted to defibrillate in manual mode, which requires the rescuer to visually identify the rhythm and decide whether to deliver a shock. Operation of the defibrillator in manual mode has been shown to decrease the duration of CPR interruptions,[50,51] and, in one study, was associated with increased return of spontaneous circulation.[51] For this reason, it is recommended that health professionals master rhythm interpretation and manual defibrillator operation in order to maximize CCF and potentially improve clinical outcome.

Shockable rhythms are:

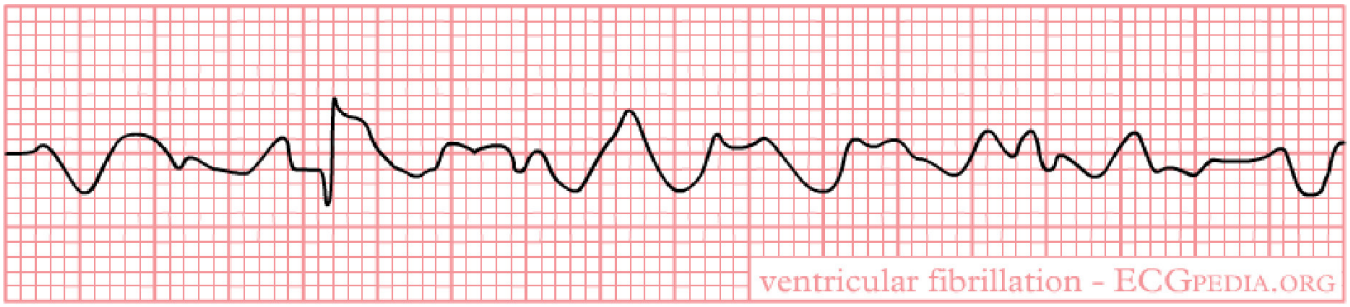
- Ventricular fibrillation **(See Figure 1)**
  - Randomly fluctuating electrical activity
  - No organized QRS complexes
  - Always pulseless
- Ventricular tachycardia **(See Figure 2)**
  - Rate > 150 beats/min and regular
  - Wide, bizarre QRS complexes
  - May be pulseless, unstable, or stable

Airway Management And Ventilation

The 2010 AHA guidelines stipulated that compressions should be initiated before ventilations, in recognition of the primary importance of restoring circulation in adult arrest patients.[52] From a physiologic standpoint, patients with nonasphyxial causes of arrest may be assumed to have normal arterial oxygen concentrations, providing a reservoir that temporarily permits continued oxygen delivery despite absence of ongoing respiration. Restoration of circulation will therefore enable continued tissue-level oxygenation until this reservoir is depleted. After this occurs, respiratory support is necessary to reoxygenate the blood and permit ongoing oxygen delivery. For patients with asphyxial causes of arrest, it may be assumed that oxygen stores have already been depleted, necessitating immediate respiratory support in order to provide tissue-level oxygenation.

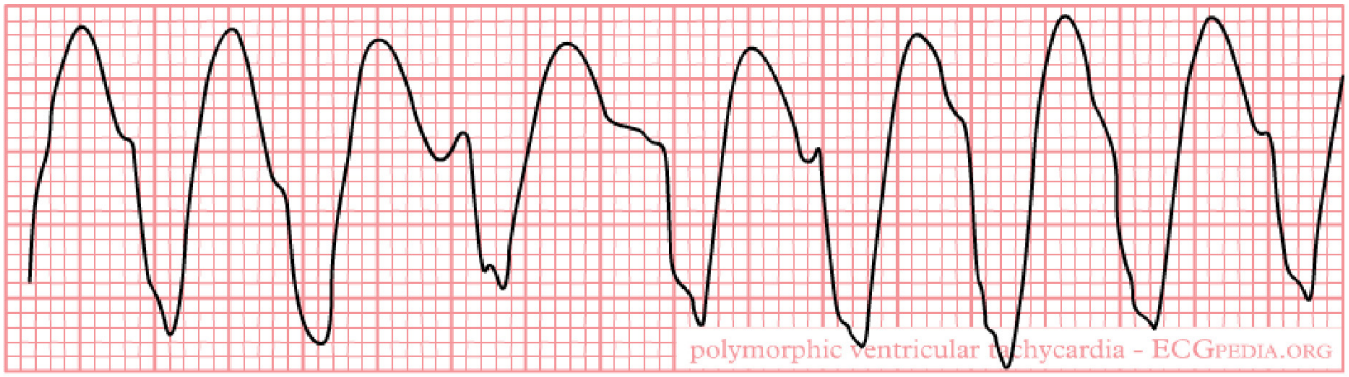
Consequently, the precise point in cardiac arrest resuscitation at which respiratory support becomes necessary will vary according to the patient's prearrest physiologic state and the cause of cardiac arrest. Even for patients with nonasphyxial arrest, the optimal timing for initiation of respiratory support has not been established. For these reasons, healthcare professionals are expected to provide both chest compressions and ventilations for all patients in cardiac arrest. While compression-only CPR has become the standard for layperson bystanders, healthcare professionals should consider the likely cause of arrest when deciding when and how to initiate respiratory support. While a brief period of compression-only CPR may be appropriate for a patient who collapses suddenly from a presumed cardiac event, this approach is not appropriate for a victim of drowning or choking, in whom effective ventilation is of the utmost importance. In the out-of-hospital setting prior to EMS arrival, mouth-to-barrier or mouth-to-mouth ventilation may be required in situations where respiratory support is necessary.

Figure 1. Electrocardiogram Of Ventricular Fibrillation



Source: en.ECGPedia.org. Available at: http://en.ecgpedia.org/index.php?title=File:Rhythm_ventricular_fibrillation.png. Used with permission.

Figure 2. Electrocardiogram Of Ventricular Tachycardia



Source: en.ECGPedia.org. Available at: http://en.ecgpedia.org/index.php?title=File:Vtach.png. Used with permission.

In the ED, ventilation is an integral part of resuscitation, and may be accomplished via bag-valve mask, supraglottic airway, or endotracheal intubation. The 2015 guidelines leave the choice of how to perform ventilation to the discretion of the practitioner, as there is no evidence that airway management approach influences patient outcome.[22] If noninvasive ventilation is used, then compressions and breaths must be coordinated using a 30:2 ratio. If an advanced airway is placed, this is unnecessary, and breaths should be delivered every 6 seconds.[22] It should be emphasized that advanced airway placement requires considerable skill and carries a significant risk of interrupting BLS. It should be attempted only if it can be accomplished with minimal impact on chest compressions and defibrillation.

If intubation is performed, it is essential to confirm and monitor placement of the endotracheal tube. Misplacement and dislodgement of endotracheal tubes are common during CPR, and esophageal intubation can be a fatal error. The 2015 AHA guidelines recommend use of continuous waveform capnography for confirmation and monitoring of airway placement.[22] This approach is highly specific for confirmation of endotracheal tube placement, though sensitivity is poor in cardiac arrest, as false-negative results are quite common due to cessation of normal cellular carbon dioxide production.[53,54] For this reason, noncapnographic methods of confirmation may be required in some cases.

Vascular Access

The 2015 AHA guidelines did not address specific vascular access techniques, though they did emphasize that vascular access is of secondary importance compared to BLS interventions, and should not be permitted to impede CPR or defibrillation.[22] The 2010 guidelines also did not advocate for a single optimal approach to vascular access, though they discussed the relative merits of peripheral, intraosseous, and central access. Intraosseous access was recommended as a reasonable option in cases where peripheral access was not readily available. Central access (subclavian or internal jugular) was noted to offer pharmacodynamic benefits, but carries substantial risk of interrupting CPR.[55] There is no known relationship between choice of vascular access approach and patient outcome, and there is far more evidence supporting the survival benefits of CPR than of parenteral drugs. It therefore seems appropriate to select an access strategy that is simple and minimally disruptive of BLS interventions, such as peripheral line followed by intraosseous needle if unsuccessful. There is evidence that tibial intraosseous placement yields higher success rates and faster access times compared to humeral intraosseous or peripheral line placement.[56,57]

Pharmacologic Interventions

Overall, there is little evidence that any pharmacologic intervention substantially impacts long-term survival outcome in cardiac arrest. For this reason, the AHA has placed relatively less emphasis on drugs compared to CPR and defibrillation in recent iterations of the guidelines. A large prehospital study found no difference in survival among patients treated with only BLS interventions (ie, chest compressions, noninvasive ventilation, and defibrillation) compared to those treated with "advanced" interventions, including vascular access and parenteral drugs.[58] That said, drugs are still included in advanced life-support algorithms, and the evidentiary basis for their use merits consideration. These medications may augment basic measures in some cases, and certain niche applications can be considered.

Vasopressors

Vasopressor agents are used in cardiac arrest to increase peripheral vascular resistance, thereby optimizing perfusion of central organs, including the heart and brain. Epinephrine is the most commonly used vasopressor in cardiac arrest. The benefits of epinephrine stem from its alpha-adrenergic (vasoconstrictor) effects, while its beta-adrenergic effects are potentially deleterious, as they increase myocardial work and may be arrhythmogenic. It is currently unknown whether epinephrine is harmful, beneficial, or neutral in the management of cardiac arrest.

A recent placebo-controlled randomized trial demonstrated beneficial short-term effects of epinephrine, with substantially more patients achieving return of spontaneous circulation in the epinephrine group compared to the control group.[59] No long-term survival benefits were observed in this study, though it was stopped early due to ethical concerns, and was therefore insufficiently powered to detect this finding. The early termination of this trial is vexing, as it was the first randomized trial comparing epinephrine to placebo, and had the potential to add considerably to the understanding of the optimal role for this drug in cardiac arrest. There is some evidence that high-dose epinephrine improves short-term outcome compared to standard-dose epinephrine,[60] but no evidence that it improves long-term or neurologic outcome.[61,62] The 2015 AHA guidelines recommend continued use of standard-dose epinephrine (1 mg every 3-5 min) in cardiac arrest, but high-dose regimens are not recommended.[22]

While there is nothing new in the guidelines about epinephrine indications or dosing, there are updated recommendations regarding the timing of epinephrine administration. Two recent studies of patients with nonshockable rhythms demonstrated time-dependent benefits of epinephrine administration, with better survival and neurologic

outcome among patients receiving epinephrine early in resuscitation compared to those receiving it late.[63,64] These data have prompted a recommendation that patients in nonshockable rhythms receive epinephrine as soon as is feasible.[22] For patients in shockable rhythms, defibrillation remains the first resuscitative priority.

The potential adverse effects of epinephrine on myocardial oxygen consumption and dysrhythmia have prompted interest in alternate vasopressors for use in cardiac arrest. Vasopressin is a powerful vasoconstrictor that does not act on adrenergic receptors, and thus lacks the potentially harmful effects of epinephrine. While there is some evidence for benefit of vasopressin compared to epinephrine in select patients,[65] a large meta-analysis failed to demonstrate any difference in survival outcomes between the drugs.[66] Another study showed no benefit of combination therapy with vasopressin and epinephrine compared to epinephrine alone.[67] Despite the theoretical benefits of vasopressin, there is no evidence that it yields superior outcome to epinephrine, and it was thus removed from the 2015 AHA guidelines.[22]

Antiarrhythmics

Antiarrhythmic drugs work through several physiologic mechanisms, acting on ion channels to reduce automaticity of electrical conduction in the myocardium. Rather than directly converting VF/VT to a perfusing rhythm, the goal of these drugs is to increase the likelihood of successful rhythm conversion after defibrillation. Antiarrhythmics have been included in cardiac arrest algorithms based on their theoretical benefits, despite a paucity of evidence that they actually improve outcome.

Amiodarone is the most commonly used antiarrhythmic drug for cardiac arrest. Two randomized trials have established its benefit for return of spontaneous circulation and short-term survival.[68,69] Lidocaine, procainamide, and magnesium have been studied in cardiac arrest as well, and there is no evidence that they offer survival benefit.[22] A recent multicenter randomized trial comparing lidocaine, amiodarone, and placebo did not show any benefit for survival to hospital discharge or favorable neurologic outcome among patients with out-of-hospital shock-refractory VF/VT arrest.[70] The 2015 AHA guidelines recommend consideration of amiodarone for VF/VT that is refractory to CPR, defibrillation, and vasopressor therapy, stressing the limitations of the evidence underlying this recommendation. Lidocaine may be considered as an alternative to amiodarone.[22]

Other Drugs

The 2010 AHA guidelines specifically recommended against administration of several drugs that had been historically used in cardiac arrest. Atropine, while not harmful for treatment of asystole or bradycardic PEA, has not been found to produce clear clinical benefit and is no longer recommended for adult cardiac arrest. Studies of bicarbonate have produced mixed results, with some evidence of clinical harm. While not recommended for routine cardiac arrest, bicarbonate may still be warranted in cases of pre-existing severe metabolic acidosis, hyperkalemia, or tricyclic antidepressant overdose. Calcium has never been shown to be beneficial in cardiac arrest and should not be used routinely, although, like bicarbonate, it may be useful in cases of hyperkalemia.[55]

Immediate Postarrest Care In The Emergency Department

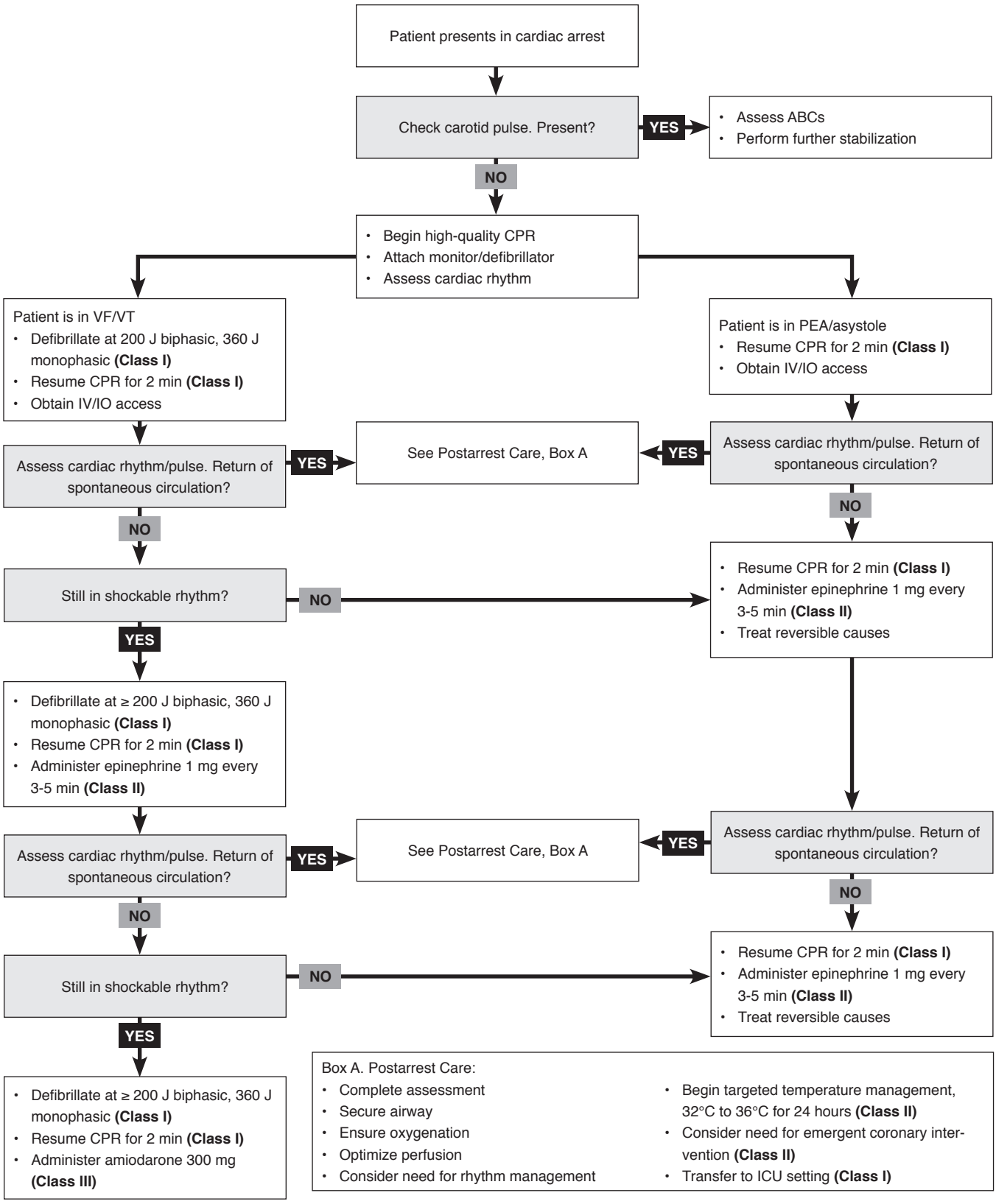
Patients who are successfully resuscitated from cardiac arrest require meticulous monitoring and stabilization to prevent recurrent arrest and to optimize neurologic outcome. Careful attention must be paid to ensuring adequate airway control, oxygenation, and ventilation. Continuous cardiac rhythm monitoring is necessary due to the high incidence of postarrest dysrhythmias. Hemodynamic monitoring is also warranted to ensure adequate systemic, cerebral, and coronary perfusion. In addition to these basic stabilization procedures, there are some specific interventions that merit consideration in the immediate postarrest period.

Pharmacologic Interventions

It is intuitive that antiarrhythmics may be useful in the postarrest period, as their effects on electrical automaticity may prevent recurrent dysrhythmias. Lidocaine has been historically used for this purpose, though there is conflicting evidence regarding its benefits. In 2013, a large observational study found that lidocaine does reduce postarrest dysrhythmias, but had no significant impact on survival.[71] We recommend use of antiarrhythmics in cases of recurrent postarrest ventricular ectopy despite limited evidence of survival benefit, and generally prefer amiodarone for this purpose.

Beta blockers are of interest in postarrest patients, as they decrease catecholamine activity, potentially decreasing myocardial ischemia and arrhythmia risk. In one study, use of postarrest beta blockers was shown to be an independent predictor of survival,[72] and other studies suggest benefit from these drugs during or after cardiac arrest.[73] A small study of VF/VT arrest patients refractory to standard care showed improvement in survival outcome among patients treated with esmolol infusion during resuscitation.[74] Despite this encouraging preliminary data, there is currently inadequate evidence to support the routine use of beta blockers during or after cardiac arrest, and the 2015 AHA guidelines leave this decision to individual providers.[22]

Clinical Pathway For Management Of Cardiac Arrest In The Emergency Department



Abbreviations: ABC, airway, breathing, circulation; CPR, cardiopulmonary resuscitation; ICU, intensive care unit; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

For Class of Evidence definitions, see page 11.

Hemodynamic Management

Myocardial dysfunction and shock are common after cardiac arrest, and vasopressor support is often required to support systemic perfusion.[75] Postarrest hypotension is strongly associated with adverse outcome.[76,77] There are no randomized trials to guide management of hypotension in the postarrest period, and optimal hemodynamic targets remain undefined.[78] A few observational studies have evaluated care bundles that include other postarrest interventions, such as therapeutic hypothermia and PCI, in addition to goal-directed management of hemodynamics. These studies have produced mixed results, and it is impossible to discern the independent effect of hemodynamic management, if any.[79-81] For this reason, the input provided by the 2015 AHA guidelines is quite limited, consisting of a recommendation to promptly treat systolic blood pressures < 90 mm Hg, with the caveat that optimal blood pressure varies between individuals. No guidance is offered regarding how to achieve this goal; however, we recommend norepinephrine infusion for patients with hypotension following cardiac arrest, as it is less arrhythmogenic than alternative vasopressors.[82]

Targeted Temperature Management

There has been immense interest in postarrest therapeutic hypothermia since the first human study of this treatment was published in 2002. This study included 77 patients who remained comatose following out-of-hospital VF/VT arrest, and found better functional outcome among the group treated with hypothermia compared to the normothermic group.[83] Conflicting outcomes have been observed for therapeutic hypothermia among out-of-hospital arrest patients with nonshockable rhythms.[84-86] For inhospital cardiac arrests, a large study found that hypothermia was not beneficial, but the results were confounded by high rates of inadequate temperature control in the treatment group.[87]

Studies of hypothermia have been criticized for inadequately controlling hyperthermia, and for high rates of failure to achieve target temperatures in hypothermia groups.[88] A recent large study addressed this issue by rigorously controlling temperature in 2 groups; one at 33°C and the other at 36°C. This study found no differences in survival or neurologic outcome between the two temperature groups,[89] suggesting that careful temperature control and avoidance of fever may be more important than the precise target temperature selected. For this reason, the guidelines now advocate for targeted temperature management rather than therapeutic hypothermia. Targeted temperature management is recommended for all comatose postarrest patients, with selection and maintenance of a single target temperature between 32°C and 36°C for at least 24 hours.[78]

Selection of the appropriate temperature for targeted temperature management is at the discretion of the clinician, and may take individual patient factors into consideration. Patients with high bleeding risk (eg, those undergoing fibrinolysis) may be maintained at higher temperatures to minimize coagulopathy. Patients with neurologic complications (eg, recurrent seizures) may benefit from more aggressive cooling and could be maintained at lower temperatures. Whatever temperature is selected, it should be carefully monitored and actively maintained for a minimum of 24 hours. The 2015 AHA guidelines offer no guidance regarding how temperature regulation should be achieved, though there is a specific recommendation against chilled fluid boluses in the prehospital setting.[78] We recommend initiation of targeted temperature management in the ED for all postarrest survivors, through careful temperature monitoring. Passive external cooling is typically sufficient to achieve target temperatures, though more aggressive cooling may be warranted for patients with fever.

Prognostic Indicators Of Outcome

Prognostication of outcome is essential in cardiac arrest management, as it guides the decision of whether or not to continue resuscitative efforts. Premature withdrawal of care in a patient with poten-

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

tial for survival is obviously a tragic error. Futile care consumes substantial resources, subjects patients to needless indignity, and may exert adverse psychological effects on loved ones and healthcare workers alike. For these reasons, reliable prognostic indicators are needed to guide termination decisions. Several indicators have been studied, including end-tidal carbon dioxide ($ETCO_2$) and ultrasound.

End-Tidal Carbon Dioxide

CO_2 is a by-product of cellular metabolism and is excreted during exhalation. $ETCO_2$ is most reliably measured using continuous waveform capnography. In cardiac arrest, $ETCO_2$ is a surrogate measure of the cardiac output achieved by chest compressions, as well as the presence of ongoing metabolic activity. Two studies have examined the prognostic value of $ETCO_2$, both finding that values < 10 mm Hg are strongly associated with mortality.[90,91] While these findings are provocative, it should be emphasized that there are numerous sources of error in $ETCO_2$ measurement, including leaks and kinks in tubing, bronchospasm, pulmonary edema, and lung pathology. Additionally, these studies were conducted in intubated patients and cannot be extrapolated to patients who are managed with bag-valve mask ventilation or supraglottic airways. The 2015 AHA guidelines recommend that $ETCO_2$ not be used as the sole prognostic indicator for arrest patients, though it can be considered as part of the global clinical picture in intubated patients.[78]

Ultrasound

A few small studies suggest that ultrasound-diagnosed ventricular standstill may predict mortality in cardiac arrest.[32,33] This method of prognostication merits further study, as it is readily available in most EDs and provides information on physiologic function that may not be otherwise available. The appropriate use of ultrasound in this context was not addressed in the 2015 AHA guidelines.

Termination Of Resuscitation

There are no clear guidelines regarding termination of resuscitation. In the past, studies have shown that longer resuscitations are associated with worse survival outcome.[92,93] However, with high-quality CPR, adequate organ perfusion may be maintained for a considerable period of time, and intact survival has been reported even after prolonged resuscitation. No single parameter can reliably predict survival, and decisions regarding continuation of resuscitation must be individualized, considering a variety of factors.

Special Circumstances

Pregnancy

Advanced pregnancy requires modifications to standard resuscitation techniques. In late pregnancy, the gravid uterus can compress the aorta and inferior vena cava when the patient is supine, impairing venous return to the heart and decreasing cardiac output. Manual left uterine displacement is recommended for all cardiac arrest patients with pregnancy > 20 weeks' gestation in order to alleviate aortocaval compression and improve perfusion with CPR. Resuscitation in the left lateral tilt position is not recommended, as this technique compromises CPR quality.[94] Evacuation of the uterus provides ultimate relief of aortocaval compression, and perimortem cesarean delivery may benefit the circulatory status of the mother regardless of fetal viability. While there are no large studies, a case series showed high rates of successful maternal resuscitation following perimortem cesarean delivery,[95] and this treatment modality should be considered in all pregnant arrest patients > 20 weeks' gestation. The 2015 AHA guidelines stipulate that resources for perimortem cesarean delivery should be mobilized as soon as cardiac arrest is diagnosed in pregnant patients > 20 weeks, and should be performed if standard resuscitation with uterine displacement fails to restore circulation within 4 minutes.[94]

Pulmonary Embolism

Thrombotic diseases such as myocardial infarction or pulmonary embolism are common causes of cardiac arrest.[11] This fact has sparked interest in the role of fibrinolytic agents in cardiac arrest. The 2010 AHA guidelines discouraged the routine use of these agents,[55] primarily due to 2 trials that found no survival benefit for empiric administration of fibrinolytics in arrest patients,[96,97] one of which demonstrated increased risk of intracranial hemorrhage in the treatment group. Both of these studies used fibrinolytics without regard to arrest etiology, and thus may not apply to patients with known or strongly suspected pulmonary embolism. The 2015 AHA guidelines do not offer input regarding patients with suspected pulmonary embolism, but for patients with cardiac arrest due to confirmed pulmonary embolism, they specifically advocate for consideration of clot-directed therapy in the form of fibrinolysis, surgical embolectomy, or catheter embolectomy.[94] The decision of which therapy to pursue is left to the discretion of the clinician, based on available resources and patient factors. It should be noted that outcomes are extremely poor in fulminant pulmonary embolism without clot-directed therapy, and the need for emergent lifesaving treatment may supersede concerns about contraindications to fibrinolysis.[94]

Opioid Overdose

Opioid use disorders are common, and mortality from opioid overdose is increasing.[98] Depending on the quantity ingested and the patient's tolerance, opioids may cause decreased level of consciousness, respiratory depression, apnea, or hypoxic cardiac

arrest. Distinguishing between these manifestations of opioid toxicity may be impossible for first responders, as pulse and respiratory assessment by lay rescuers is known to be unreliable.[14,15] For this reason, treatment of opioid overdose by lay rescuers has, historically, not been recommended. However, the opioid antagonist naloxone does not produce adverse effects in patients without opioid dependence, and the withdrawal it precipitates in opioid-dependent patients is not life-threatening. Naloxone has become readily available to the public following the United States Food and Drug Administration (FDA) approval of naloxone autoinjectors designed for emergent treatment of opioid-induced respiratory arrest.[99] It is therefore reasonable to consider naloxone as an adjunct to standard BLS interventions, as it carries very little risk and immense potential benefit for patients who need it. To this end, the 2015 AHA guidelines support the empiric use of naloxone by first responders as a standard part of BLS management, provided that naloxone administration does not interfere with CPR and defibrillation.[94] Health professionals should continue using naloxone, as usual, for opioid-associated respiratory depression, but no recommendations are offered for its use in cardiac arrest.[94]

Torsades de Pointe

Torsades de pointe is a specific form of polymorphic ventricular tachycardia. **(See Figure 3.)** Unlike monomorphic VT, torsades is commonly caused by noncardiac conditions like electrolyte abnormalities or toxic ingestions. Torsades was not specifically addressed in the 2015 AHA guidelines, but previous iterations recommend treating cardiac arrest associated with this rhythm like any other VF/VT arrest, using defibrillation and CPR as first-line interventions.[55] The important difference in the treatment of torsades is that magnesium sulfate (1-2 grams IV push) may be considered as an antiarrhythmic therapy for torsades related to QT-segment prolongation.[55]

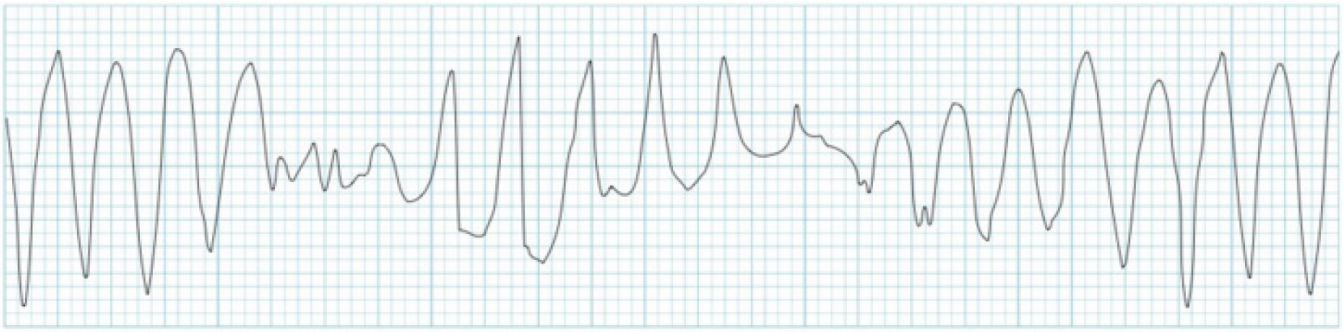
Patients With Other Toxicologic Causes Of Arrest

Toxicologic causes of cardiac arrest may require specific treatments for the underlying physiologic derangement caused by the toxin, in addition to standard cardiac arrest management. For example, alkalinization of patients poisoned with tricyclic antidepressants may be a reasonable adjunct to CPR and vasopressor therapy. The 2015 AHA guidelines only addressed 1 specific toxicologic etiology of arrest: local anesthetic systemic toxicity (LAST). LAST is a rare complication of regional anesthesia that is most often associated with bupivacaine, and can cause cardiac arrest that is refractory to standard interventions. Intravenous lipid emulsions have been recommended in the treatment of LAST. Addition of lipids to the serum can sequester lipophilic medications like local anesthetics, thereby decreasing their physiologic effects. While there are no comparative studies, a systematic review of intravenous lipid emulsions for LAST showed benefit in all published human cases.[100] For this reason, the 2015 AHA guidelines recommend considering intravenous lipid emulsions along with standard resuscitation in patients with LAST. Intravenous lipid emulsions may also be considered in other toxicologic causes of arrest (eg, tricyclic antidepressants, antihypertensives) that are refractory to standard measures.[94]

Hyperkalemia

Hyperkalemia that causes cardiac arrest occurs most commonly in patients with impaired potassium excretion due to renal insufficiency. Medical treatment of hyperkalemia should be initiated in patients with cardiac arrest related to this etiology, along with standard resuscitative measures. The first priority is stabilization of the myocardial membrane, which is accomplished with calcium chloride or calcium gluconate.[55] The next priority is intracellularization of circulating potassium, which is accomplished with sodium bicarbonate, intravenous short-acting insulin

Figure 3. Electrocardiogram Of Torsades de Pointe



Source: www.lifeinthefastlane.com; available at: http://lifeinthefastlane.com/ecg-library/tdp/. Used with permission.

(given with glucose to prevent hypoglycemia), and nebulized albuterol, if feasible, depending on airway management approach.[55] These treatments are temporizing, lowering serum potassium long enough to bridge the patient to a more definitive intervention. Definitive treatments eliminate potassium from the body, and emergent dialysis is the most appropriate option in hyperkalemic cardiac arrest.[55]

Accidental Hypothermia

Accidental hypothermia presents a unique challenge in resuscitation, as patients with low core temperatures may have depressed central nervous system and cardiac activity that is difficult to distinguish from cardiac arrest. Furthermore, when cardiac arrest does actually occur, hypothermia exerts a neuroprotective effect, potentially enabling intact survival even after prolonged periods without perfusion. These observations have led to the adage that "you're not dead until you're warm and dead," meaning that hypothermic patients without signs of life should be transported, resuscitated, and actively warmed regardless of the duration of their downtime. Accidental hypothermia was not addressed in the 2015 AHA guidelines, but the 2010 recommendations on this subject merit review.

There is a common misconception that resuscitative interventions may be dangerous in hypothermic patients due to cardiac irritability. For this reason, rescuers may be reluctant to begin CPR or perform defibrillation, often completing prolonged assessments or attempting rewarming before addressing the problem of cardiac arrest. The guidelines do not recommend modifications to BLS in hypothermia, stipulating that chest compressions, rescue breathing, and defibrillation should be completed as normal, regardless of temperature. Airway management, vascular access procedures, and vasopressor administration should also proceed as for normothermic patients, though the role of antiarrhythmics is less clear.[55]

The major modification to resuscitation in hypothermic patients is the addition of active core rewarming techniques to standard therapies. Cardiopulmonary bypass is the most effective rewarming technique for patients in cardiac arrest. Successful use of bypass has been described in several case reports, and a case series suggests reasonable chances of intact survival for patients treated this way.[101] If bypass is not available, thoracic lavage may also be successfully used, and many case reports describe intact survivors.[102] Less-invasive techniques such as administration of warm intravenous fluids or humidified air do not produce significant heat transfer, and are considered adjuncts to more definitive approaches.[55]

Controversies And Cutting Edge

Percutaneous Coronary Interventions

Coronary ischemia is a common cause of cardiac arrest. Revascularization has become the standard of care for ACS in the nonarrest setting, so it is natural that there would be interest in the role of revascularization following cardiac arrest. The 2010 AHA guidelines recommended obtaining a 12-lead ECG in all successfully resuscitated arrest patients, and performing PCI in those with ST-segment elevation myocardial infarction (STEMI) patterns.[55] However, STEMI patterns may not be present in all arrest patients with ACS. A recent study of coronary angiography in cardiac arrest patients demonstrated that 96% of patients with STEMI had acute coronary occlusions on angiography, but 58% of patients without STEMI also had acute coronary occlusions.[103] These findings suggest that ST elevations alone are insufficient to identify patients who may benefit from PCI following cardiac arrest, and that a more permissive strategy is needed when selecting patients for angiography.

A 2014 study of coronary angiography in non-STEMI arrest patients found that more than a quarter had acute coronary occlusions requiring PCI, and that early PCI was associated with reduced risk of death.[104] A 2011 study focusing specifically on shockable rhythm arrests also demonstrated high rates of coronary occlusion among patients without STEMI, and improved survival among patients who underwent PCI.[105] A 2012 study correlated early coronary angiography with improved survival outcome, again noting that a significant proportion of patients requiring PCI had nondiagnostic ECGs.[106]

Because of the evidence linking early PCI to improved survival outcome, the 2015 AHA guidelines recommend that all patients successfully resuscitated from cardiac arrest of suspected cardiac etiology should be considered for emergent coronary angiography. This recommendation applies regardless of neurologic status or presenting arrest rhythm.[78]

Extracorporeal Cardiopulmonary Resuscitation

Extracorporeal CPR (ECPR) refers to techniques that permit oxygenation and circulation of the blood to occur outside of the body, including extracorporeal membrane oxygenation (ECMO) and cardiopulmonary bypass. These techniques may allow additional time to facilitate treatment for underlying causes of arrest, but they require specialized equipment and expertise that may not be routinely available even in tertiary care centers. There are no randomized trials of ECPR, and observational studies yield mixed results. Some observational studies show promising associations between ECPR and improved survival and/or neurologic outcome,[107-109] while others do

Risk Management Pitfalls For Cardiac Arrest

1. **"I followed the guidelines – how was I supposed to know why he was in PEA?"**
   PEA is often caused by noncardiac conditions, and will resolve only when these underlying conditions are treated. Standard resuscitation interventions do not resolve physiologic derangements causing PEA, and should be viewed as a bridge to more definitive care. Know the Hs and the Ts, and hunt for the root cause of PEA in every patient.

2. **"I was busy intubating – it's not my fault that chest compressions were too slow."**
   Chest compressions are the single most important intervention in cardiac arrest regardless of etiology, whereas intubation is not necessary in the majority of cases. Physicians must be meticulous about every aspect of compression quality. Compression rate can be monitored using the CPR quality feedback features available on many modern defibrillators, a metronome or timer, or even by singing ("Stayin' Alive" has just the right tempo).

3. **"I couldn't compress any deeper – I was too tired!"**
   Achieving adequate compression depth is physically challenging for many rescuers, and as fatigue worsens, compressions suffer. Physicians must monitor their team members for signs of fatigue and change compressors as needed to ensure quality CPR.

4. **"I had no idea we stopped compressions for so long – I was having trouble getting a decent image on the ultrasound."**
   Low chest compression fraction contributes to poor survival outcomes, and needless pauses in CPR are not to be tolerated. In cardiac arrest, nothing is more important than CPR, and compressions should be halted only for scheduled breaths and rhythm checks/defibrillation. The guidelines specifically stipulate that ultrasound should not be allowed to interfere with CPR.

5. **"That monitor tracing was really weird – I didn't realize it was V-tach."**
   Defibrillation is the only effective treatment for VF/VT arrest, and it is essential for clinicians to recognize these rhythms rapidly and reliably. While manual defibrillation mode is preferred for clinicians, automated mode may be used in the face of uncertainty about the rhythm.

6. **"I knew it was V-fib, but I couldn't get the blasted defibrillator hooked up! It's the tech's job anyway."**
   It is common practice to delegate defibrillation to nursing staff, and physicians may run dozens of resuscitations without ever touching the defibrillator, leading to skill decay. As leaders, physicians must be the most competent members of the resuscitation team, and should thoroughly understand operation of lifesaving equipment like the defibrillator.

7. **"Sure, I didn't ventilate – but everyone knows that chest compressions are the only thing that matters!"**
   Emphasis on the importance of CPR and defibrillation has led to a misconception that respiratory support is irrelevant. While "hands-only" CPR is acceptable for lay rescuers, health professionals must provide ventilation along with chest compression in all resuscitations. Advanced airway placement is not mandatory, but effective ventilation is. Every breath should produce visible chest rise, and compressions should be coordinated with ventilations in a 30:2 ratio prior to advanced airway placement.

8. **"So I gave a few doses of sodium bicarbonate – what's wrong with that?"**
   The only drugs that are included in resuscitation algorithms are epinephrine (all rhythms) and amiodarone (VF/VT – lidocaine is an acceptable alternative). Unless the patient has a specific indication for bicarbonate, such as hyperkalemia or tricyclic antidepressant overdose, this drug is not warranted and may be harmful.

9. **"I got him back – it's not my fault that he's febrile."**
   Targeted temperature management in the range of 32ºC to 36ºC is required for a minimum of 24 hours following successful resuscitation from cardiac arrest. Fever is common in the postresuscitation period, and is known to be deleterious to outcomes, so temperature control must be meticulous.

10. **"Her ECG looked OK – how was I supposed to know this was a STEMI?"**
    Acute coronary syndromes are common among cardiac arrest patients, and the ECG is not reliable for diagnosing coronary ischemia in the postarrest period. Cardiac catheterization should be initiated for all successfully resuscitated patients with a suspected cardiac etiology of arrest.

not.[110] The only prospective study demonstrated improved neurologic outcome among patients treated with ECPR, but this study bundled ECPR with therapeutic hypothermia and aortic balloon pump placement, making it impossible to discern the independent contribution of ECPR.[111] There is currently insufficient evidence to recommend routine use of ECPR, but it may be considered for select patients, particularly in cases with reversible etiologies of arrest where time is needed to bridge the patient to definitive management.[78]

Steroids

Inflammation may contribute to adverse outcome in cardiac arrest, and steroids have been studied as adjunctive treatment in arrest patients. There is no current evidence that steroids produce independent benefit, but 2 recent studies in which steroids were bundled with vasopressin and epinephrine suggest benefit of this combination over epinephrine alone.[112,113] While provocative, these studies were not designed to address the independent effects of steroids, and the guidelines do not recommend routine use of steroids at present.[22]

Disposition

All patients who survive cardiac arrest should be managed in intensive care units.

Summary

Cardiac arrest is a common problem that requires prompt and effective intervention on the part of healthcare providers. High-quality CPR and rapid defibrillation (when indicated) are, by far, the most important interventions, and take priority over all other treatments. Airway management and respiratory support should be provided as soon as feasible. Advanced interventions such as intubation, vascular access, and parenteral medications are included in cardiac arrest algorithms, though their importance is minimal compared to basic interventions. Identification of the underlying cause of cardiac arrest is important for guiding therapy both during the arrest and after spontaneous circulation is restored. Targeted temperature management and coronary angiography should be considered for all successfully resuscitated arrest patients.

Time- And Cost-Effective Strategies

- Know when to terminate resuscitation: Futile resuscitation consumes considerable time and resources. While there are no guidelines regarding termination of resuscitation, common sense and medical judgment can be applied to decide when further efforts are likely to be futile. If patients do not respond despite high-quality CPR and effective management of underlying disease processes, then cessation of resuscitation should be considered. $ETCO_2$ and ultrasound may be used as adjuncts in making this decision.
- Disposition successfully resuscitated patients effectively. Cardiac arrest patients with return of spontaneous circulation require considerable time and resources from the care team, and are best managed in an intensive care setting. Patients with suspected cardiac causes of arrest should be expeditiously transported to the cardiac catheterization lab, and others should be sent to the ICU for targeted temperature management and other physiologic support.

Case Conclusions

In consultation with cardiology, the decision was made to send your first patient to the cath lab. In the meantime, your young patient from the night club is a dilemma – why does a young man go into cardiac arrest? As CPR continues, you hear sobbing behind you, and turn around to see a tearful young woman standing in the hallway. "It's his girlfriend," says the medic. You look at your watch – you have 90 seconds before the next shock. You ask what happened and if she knows anything that might help. "He had some weird thing with his heart," she says. "He was supposed to get some kind of study, some electricity thing, and they said he might need one of those boxes that can shock you. He was totally freaked out about it, and he kept putting it off." You turn to your student, "Take a look in his record and see what you can figure out." It's almost time for the next rhythm check when you notice his hand move. "Stop!" you say. The team pauses CPR and you see an organized rhythm on the monitor. It wasn't just your imagination – he moves his hand again, this time bringing it up to his chest and moaning a bit. "We've got him back," you say. "Let's cycle a blood pressure and grab a 12-lead."

"I've got his record!" cries your student. "You won't believe it! Brugada syndrome! Picked up on a routine ECG, totally asymptomatic. He's due in electrophysiology lab this week." You smile at your student. "Well I hope you were paying attention – you won't see this every day!" You go back to the bedside and find your patient with stable blood pressure and oxygenation, now opening his eyes. "Please get cardiology on the phone right away," you say.

"Don't need to – they're calling you," says the nurse, handing you a phone. "Hey, great timing!" you say. "Not sure what you mean," says the cardiologist, "I was just calling to tell you about that V-fib patient we cathed. You wouldn't believe it – a 99% LAD stenosis, with a totally normal ECG!" You smile. "The way my night is going, I'll believe anything!" "He's totally stable now," says the cardiologist, "normal vitals, following commands – what a great save!" "I'm so glad to hear it," you reply, "You won't believe this, but I've got another one for you…"

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study is included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. You are attending a sporting event when you hear someone cry for help. You rush over and find a young woman unresponsive on the ground. She has no palpable carotid pulse and no obvious respiratory effort. You begin chest compressions and call for an automated external defibrillator (AED) while a bystander calls EMS. Once the AED arrives, you apply it and it does not advise a shock. What is the most likely cause of this patient's arrest?

a. Congenital cardiomyopathy
b. Congenital conduction disturbance
c. Acute myocardial infarction
d. Pulmonary embolism

2. **You rush to the bedside of a patient who has collapsed suddenly after presenting with mild dyspnea. The patient, a 72-year-old man, had previously been stable on supplemental oxygen. He is now apneic and unresponsive. Your intern palpates the carotid and says, "I think there might be a weak pulse – I'm just not sure." What is the most appropriate course of action?**
   a. Palpate the carotid yourself for 30 seconds to determine whether there is a pulse
   b. Perform ultrasound examination to see if there is myocardial movement
   c. Immediately begin chest compressions
   d. Immediately begin bag-valve mask ventilation

3. **You are supervising the resuscitation of a 52-year-old man in pulseless electrical activity (PEA) of unknown etiology. You want to ensure optimal CPR quality, and you notice that your new defibrillator has a CPR feedback feature. You check the readings on the monitor, and see the following information regarding chest compressions: rate: 110; depth: 2.2; recoil: complete; and chest compression fraction: 56%. What advice should you give your CPR provider?**
   a. Push faster! Rate should be above 120.
   b. Don't push so hard! Depth should only be 2 inches.
   c. Lean more! You shouldn't release pressure on the chest wall.
   d. Get back on the chest! Decrease the frequency and length of pauses.

4. **You are resuscitating a morbidly obese 48-year-old woman in PEA. Your intern goes to the head of the bed to manage the airway. What instructions should you give her?**
   a. Don't begin ventilations right now – chest compressions are the top priority and we don't want to interrupt them for respiration.
   b. Use the bag-valve mask to give 2 breaths for every 30 compressions – and be sure to check for chest rise with each breath.
   c. Get set up to intubate right away – we'll pause chest compressions so you can get a clear shot at the airway.

5. **EMS transports a 63-year-old man in ventricular fibrillation (VF). Chest compressions and bag-valve mask ventilation are in progress, and 1 shock was administered prior to arrival. The patient does not yet have vascular access. Which statement regarding vascular access in this patient is TRUE?**
   a. A central line is the recommended form of access, as this approach ensures rapid delivery of drugs to the heart.
   b. Tibial intraosseous access is inappropriate, as this approach is technically difficult and less likely to succeed than other options.
   c. Vascular access is not necessary, as pharmacologic interventions are not indicated for this patient.
   d. A peripheral IV is acceptable, as this approach allows rapid administration of drugs, fluids, and other needed interventions.

6. **You are debriefing following the unsuccessful resuscitation of an elderly patient who presented in asystole. A team member asks why epinephrine was given to this patient, stating that he had read that it doesn't make any difference and might even be dangerous in resuscitation. Which is the best response?**
   a. That's correct – epinephrine has no proven benefit and may actually cause harm, which is why it has been removed from arrest algorithms.
   b. That's incorrect – epinephrine improves both short- and long-term survival outcome, which is why it is a mandatory element of all arrest algorithms.
   c. That's partly correct – epinephrine may worsen ischemia and arrhythmias, but there is evidence that it increases return of spontaneous circulation, so it is still recommended in arrest algorithms.

7. **You are resuscitating a 57-year-old man who had a witnessed cardiac arrest with immediate bystander CPR. The first rhythm was ventricular tachycardia (VT), and the patient has remained in VT despite continuous high-quality CPR, defibrillation, effective bag-valve mask ventilation, and epinephrine. What is the next most appropriate course of action?**
   a. Administer amiodarone
   b. Administer lidocaine
   c. Administer magnesium
   d. Perform intubation

8. **You are providing medical direction for EMS in a rural area, and you receive a radio consult for a patient with witnessed VF arrest. The patient has been successfully resuscitated and now has stable vital signs, but remains comatose. His current rectal temperature is 36°C. The EMT states that she still has an hour in transit to the nearest hospital, and she'd like to begin "cooling him down." What is the most appropriate response?**
   a. No – there is no proven benefit for targeted temperature management for patients with out-of-hospital cardiac arrest.
   b. No – his core temperature is already in the acceptable range, so we can use targeted temperature management to keep him at 36°.
   c. Yes – targeted temperature management is only beneficial when core temperature is lowered to 32°, so we must begin cooling immediately.
   d. Yes – targeted temperature management requires boluses of chilled intravenous fluids to keep body temperature between 32° and 36°.

9. **A 68-year-old woman presents to the ED with dyspnea and chest pain 2 days after undergoing knee replacement surgery. She is tachycardic, tachypneic, hypoxemic, and mildly hypotensive on arrival. Pulmonary embolism is strongly suspected, and CT angiography of the chest confirms this diagnosis. A heparin drip is initiated promptly, but the patient deteriorates and has a PEA arrest. Which statement regarding this patient's care is TRUE?**
   a. Fibrinolysis is absolutely contraindicated due to her recent major surgery.
   b. Heparin would be expected to adequately address her PE.
   c. Fibrinolysis or other clot-directed therapy should be performed promptly.
   d. Intracranial hemorrhage is not associated with appropriate treatment of cardiac arrest due to PE.

10. **You have successfully resuscitated a 59-year-old woman with pulseless VT who had a sudden, witnessed collapse in a shopping mall. She has a prior history of coronary artery disease with a single coronary stent placed 3 years ago. She now has stable vital signs but remains comatose. Her 12-lead ECG shows ST segment depressions, but no STEMI. Which statement regarding the role of cardiac catheterization in her care is TRUE?**
   a. She should not undergo catheterization because of her neurologic status.
   b. She should not undergo catheterization because her ECG is nondiagnostic.
   c. She should undergo catheterization because of her ischemic ECG changes.
   d. She should undergo catheterization because of her history and presentation.

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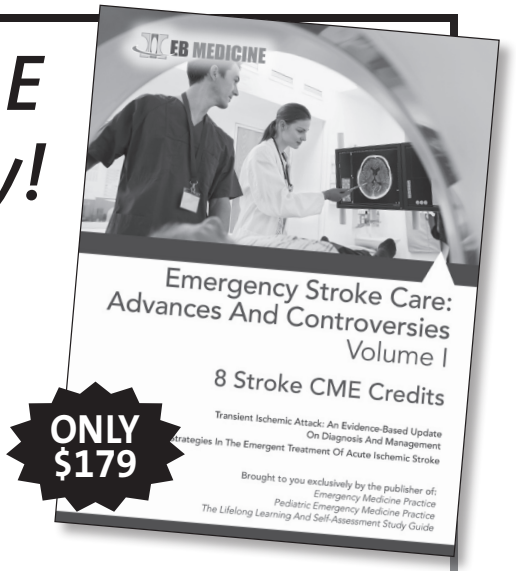

Emergency Stroke Care: Advances And Controversies, Volume I is a brand-new resource that reviews the latest research, recommendations, and guidelines for the diagnosis and management of stroke.

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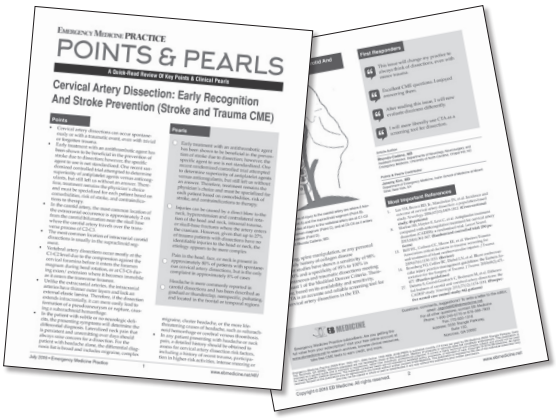

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