Advanced cardiac life support (ACLS) in adults

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INTRODUCTION — The field of resuscitation has been evolving for more than two centuries [1]. The Paris Academy of Science recommended mouth-to-mouth ventilation for drowning victims in 1740 [2]. In 1891, Dr. Friedrich Maass performed the first documented chest compressions on humans [3]. The American Heart Association (AHA) formally endorsed cardiopulmonary resuscitation (CPR) in 1963, and by 1966, they had adopted standardized CPR guidelines for instruction to lay-rescuers [2].

Advanced cardiac life support (ACLS) guidelines have evolved over the past several decades based on a combination of scientific evidence of variable strength and expert consensus. The American Heart Association (AHA) developed the most recent ACLS guidelines in 2010 using the comprehensive review of resuscitation literature performed by the International Liaison Committee on Resuscitation (ILCOR), and these were updated in 2015 [4-8]. Guidelines are reviewed continually but are formally released every five years, and published in the journals Circulation and Resuscitation.

This topic will discuss the management of cardiac arrhythmias in adults as generally described in the most recent iteration of the ACLS Guidelines. Where our suggestions differ or expand upon the published guidelines, we state this explicitly. The evidence supporting the published guidelines is presented separately, as are issues related to controversial treatments for cardiac arrest patients, basic life support (BLS), airway management, and post-cardiac arrest management. (See "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest" and "Therapies of uncertain benefit in basic and advanced cardiac life support" and "Basic life support (BLS) in adults" and "Basic airway management in adults" and "Advanced emergency airway management in adults" and "Post-cardiac arrest management in adults".)

EVIDENCE BASED GUIDELINES — Because of the nature of resuscitation research, few randomized controlled trials have been completed in humans. Many of the recommendations in the American Heart Association's 2010 Guidelines for advanced cardiac life support and the 2015 update (hereafter referred to as the ACLS Guidelines) are made based upon retrospective studies, animal studies, and expert consensus [5,7]. Guideline recommendations are classified according to the GRADE system [9]. The evidence supporting the ACLS Guidelines is reviewed in detail separately. (See "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest".)

PRINCIPLES OF MANAGEMENT

Excellent basic life support and its importance — Excellent cardiopulmonary resuscitation (CPR) and early defibrillation for treatable arrhythmias remain the cornerstones of basic and advanced cardiac life support (ACLS). Although the 2015 update for the American Heart Association (AHA) Guidelines for ACLS (ACLS Guidelines) suggest several revisions, including medications and monitoring, the emphasis on excellent CPR and its critical role in resuscitative efforts remains unchanged (algorithm 1 and algorithm 2 and figure 1) [5,7]. We emphasize the term "excellent CPR" because anything short of this standard does not achieve adequate cerebral and coronary perfusion, thereby compromising a patient's chances for neurologically intact survival. CPR is discussed in detail separately; key principles in the performance of ACLS are summarized in the following table (table 1). (See "Basic life support (BLS) in adults".)

In the past, clinicians frequently interrupted CPR to check for pulses, perform tracheal intubation, or obtain venous access. Current ACLS Guidelines strongly recommend that every effort be made **NOT** to interrupt CPR; other less vital interventions (eg, tracheal intubation or administration of medications to treat arrhythmias) are made either while CPR is performed or, if a required intervention cannot be performed while CPR is in progress, during the briefest possible addition to the 2 minute rhythm check (after the completion of a full cycle of CPR).

Studies in both the in-hospital and prehospital settings demonstrate that chest compressions are often performed incorrectly, inconsistently, and with excessive interruption [10-14]. Chest compressions must be of sufficient depth (5 to 6 cm, or 2 to 2.5 inches) and rate (between 100 and 120 per minute), and allow for complete recoil of the chest between compressions, to be effective.

A single biphasic defibrillation remains the recommended treatment for ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). CPR should be performed until the defibrillator is ready for immediate discharge and resumed immediately after the shock is given, without pausing to recheck a pulse [15,16]. Interruptions in CPR (eg, for subsequent attempts at defibrillation or pulse checks) should occur no more frequently than every 2 minutes, and for the shortest possible duration. Key elements in the performance of manual defibrillation are described in the following table (table 2).

Patients are often over-ventilated during resuscitations, which can compromise venous return resulting in reduced cardiac output and inadequate cerebral and cardiac perfusion. A 30:2 compression to ventilation ratio (one cycle) is recommended in patients without advanced airways. According to the ACLS Guidelines, asynchronous ventilations at 8 to 10 per minute are administered if an endotracheal tube or extraglottic airway is in place, while continuous chest compressions are performed simultaneously [17]. We believe that 6 to 8 ventilations per minute are sufficient in the low-flow state of cardiac arrest and help to prevent excessive intrathoracic pressure.

Resuscitation team management — The resuscitation of a sudden cardiac arrest (SCA), by its nature a low frequency, high acuity event, is often chaotic. A growing body of literature demonstrates that by employing the principles of Crisis Resource Management (CRM), adapted from the aviation industry and introduced into medical care by anesthesiologists, disorganization during resuscitation decreases and patient care improves [18-21]. A primary goal of CRM is to access the collective knowledge and experience of the entire team in order to provide the best care possible and to compensate for oversights or other problems that any individual is likely to experience during such stressful events. Training in these principles to improve the quality of ACLS performed by healthcare clinicians is feasible and recommended [22].

Two principles provide the foundation for CRM: leadership and communication [20]. Resuscitations usually involve a number of healthcare providers from different disciplines, sometimes from different areas of an institution, who may not have worked together previously. Under these circumstances, role clarity can be difficult to establish. In CRM, it is imperative that one person assumes the role of team leader [20]. This person is responsible for the global management of the resuscitation, including: ensuring that all required tasks are carried out competently; incorporating new information and coordinating communication among all team members; developing and implementing management strategies that will maximize patient outcome; and reassessing performance throughout the resuscitation.

The team leader should avoid performing technical procedures, as performance of a task inevitably shifts attention from the primary leadership responsibilities. In circumstances where staffing is limited (eg, small community hospital), the team leader may be required to perform certain critical procedures. In these situations, leadership may be temporarily transferred to another clinician or the team leader may be forced temporarily to perform both roles, although this compromises the ability to provide proficient leadership.

In CRM, communication is organized to provide effective and efficient care. All pertinent communication goes through the team leader and the team leader shares important information with the team. When the team leader determines the need to perform a task, the request is directed to a specific team member, ideally by name. That team member verbally acknowledges the request and performs the task or, if unable to do so, informs the team leader that someone else should be assigned. Specific emphasis is placed on the assigned team member repeating back medication doses and defibrillator energy settings to the team leader. This "closed-loop" communication leads to a more orderly transfer of information and is the appropriate standard for all communication during resuscitations.

Though most decisions emanate from the team leader, a good team leader enlists the collective wisdom and experience of the entire team as needed. Team members must be encouraged to speak up if they have a concern or a feasible suggestion. Efforts should be made to overcome the tendency to withhold potentially life-saving suggestions due to the fear of being incorrect or the nature of hierarchies that exist in many healthcare institutions. Extraneous personnel not directly involved with patient care are asked to leave in order to reduce noise and to ensure that orders from the leader and feedback from the resuscitation team can be heard clearly.

Initial management and ECG interpretation — In the 2010 ACLS Guidelines, circulation assumed a more prominent role in the initial management of cardiac arrest and this approach continues in the 2015 update. The "mantra" is: circulation, airway, breathing (C-A-B). Once unresponsiveness is recognized, resuscitation begins by addressing circulation (chest compressions), followed by airway opening, and then rescue breathing. The ACLS Guidelines emphasize the importance of excellently performed, uninterrupted chest compressions and early defibrillation. Rescue breathing is performed after the initiation of excellent chest compressions and definitive airway management may be delayed if there is adequate rescue breathing without an advanced airway in place. (See 'Excellent basic life support and its importance' above and "Basic life support (BLS) in adults", section on 'Recognition of cardiac arrest'.)

In the non-cardiac arrest situation, the other initial interventions for ACLS include administering oxygen (if the patient's oxygen saturation is below 94 percent), establishing vascular access, placing the patient on a cardiac and oxygen saturation monitor, and obtaining an electrocardiogram (ECG) [5,7]. Unstable patients must receive immediate care, even when data are incomplete or presumptive (algorithm 1 and algorithm 2). Patients with clear evidence of STEMI on ECG should be prepared for rapid transfer to the catheterization laboratory, receive a thrombolytic (if not contraindicated), or be transferred to a center with PCI capabilities. These decisions are made based on local resources and protocols.

Stable patients require an assessment of their electrocardiogram in order to provide appropriate treatment consistent with ACLS guidelines. Although it is best to make a definitive interpretation of the ECG prior to making management decisions, the settings in which ACLS guidelines are commonly employed require a modified, empirical approach. Such an approach is guided by the following questions:

- Is the rhythm fast or slow?
- Are the QRS complexes wide or narrow?
- Is the rhythm regular or irregular?

The answers to these questions often enable the clinician to make a provisional diagnosis and initiate appropriate therapy.

AIRWAY MANAGEMENT WHILE PERFORMING ACLS — Ventilation is performed during CPR to maintain adequate oxygenation. The elimination of carbon dioxide is less important, and normalization of pH through hyperventilation is both dangerous and unattainable until there is return of spontaneous circulation (ROSC). However, during the first few minutes following sudden cardiac arrest (SCA), oxygen delivery to the brain is limited primarily by reduced blood flow [23,24]. Therefore, in adults, the performance of excellent chest compressions takes priority over ventilation during the initial period of basic life support. In settings with multiple rescuers or clinicians, ventilations and chest compressions are performed simultaneously.

The ventilation rate is determined by whether the patient is intubated. If the patient is not intubated, the compression to ventilation ratio is 30:2. If the patient is intubated, we suggest performing no more than six non-synchronized ventilations per minute. (See "Basic life support (BLS) in adults".)

Although research has yet to identify the preferred parameters for ventilation (eg, respiratory rate, tidal volume, inspired oxygen concentration), it is widely believed that a lower minute ventilation is needed for patients in cardiac arrest. Therefore, lower respiratory rates are used (the ACLS Guidelines recommend 10 breaths per minute with an advanced airway in place; we believe 6 breaths are adequate). In addition, we know that hyperventilation is harmful, as it leads to increased intrathoracic pressure, which decreases venous return and compromises cardiac output. Tidal volumes of approximately 600 mL delivered in a controlled fashion such that chest rise occurs over no more than one second is recommended in the ACLS Guidelines. (See "Basic life support (BLS) in adults", section on 'Ventilations'.)

Taking these principles into account, the ACLS Guidelines support the use of a bag-mask device or a blindly placed supraglottic airway for ventilation during the initial management of SCA, deferring placement of an endotracheal tube, unless intubation can be competently performed without interrupting chest compressions or one cannot ventilate the patient by other less invasive means. The performance of bag-mask ventilation (BMV) is described separately. (See "Basic airway management in adults".)

A blindly inserted supraglottic airway (eg, laryngeal mask airway, Combitube, laryngeal tube) can be placed without interrupting chest compressions, provides adequate ventilation in most cases, and reduces the risk of aspiration compared to bag-mask ventilation. Therefore, clinicians may prefer to ventilate with a supraglottic device while CPR is ongoing, rather than performing tracheal intubation. Supraglottic airways and tracheal intubation are discussed separately. (See "Devices for difficult emergency airway management in adults", section on 'Extraglottic devices' and "Direct laryngoscopy and tracheal intubation in adults".)

Advanced airway management using a supraglottic airway or tracheal intubation may not be the best approach for managing cardiac arrest patients in the prehospital setting. This view is supported by a prospective observational nationwide Japanese study involving 649,359 patients with sudden out-of-hospital cardiac arrest [25]. In this study, the rate of survival with a favorable neurologic outcome was significantly lower among those managed with advanced airway techniques compared with bag-mask ventilation (1.1 versus 2.9 percent; odds ratio [OR] 0.38, 95% CI 0.36-0.39). Higher rates of survival with a favorable neurologic outcome persisted across all analyzed subgroups, including adjustments for initial rhythm, return of spontaneous circulation, bystander CPR, and additional treatments. Although this study has limitations due to its observational approach and was performed in a single country and so may not be generalizable to all settings, its size and consistent findings across all subgroup analyses support its conclusions.

Other evidence supporting a more basic approach to airway management during resuscitation of cardiac arrest includes a retrospective review of registry data involving 10,691 cardiac arrest patients, which reported that patients managed in the prehospital setting with bag-mask ventilation had significantly higher rates of neurologically intact survival to hospital discharge than patients managed with either a supraglottic airway or intubation (18.6 versus 5.2 percent and 5.4 percent, respectively) [26]. However, randomized trials of airway management in the setting of cardiac arrest are lacking and some observational studies have reached different conclusions [27].

If advanced airway management is to be performed at all in prehospital cardiac arrest patients, it must be done by competent providers, require less than 10 seconds to complete without interruption of excellent chest compressions, and be used only after all other more essential resuscitative maneuvers have been initiated. Once performed, rescuers must avoid hyperventilation. In addition, unless adequate BMV cannot be performed, placement of an advanced airway should be attempted only during active chest compressions or deferred to the 2 minute interval (after a complete cycle of CPR) when defibrillation or patient reassessment is performed.

The ACLS Guidelines include the following additional recommendations about airway management during the performance of ACLS [23]:

- Although evidence is lacking, it is reasonable to provide 100 percent oxygen during CPR. In patients who are successfully resuscitated (ie, spontaneous circulation returns), it is important to avoid hyperoxia while maintaining oxygen saturation above 94 percent. (See "Post-cardiac arrest management in adults", section on 'Mechanical ventilation'.)
- Cricoid pressure is controversial and is no longer routinely recommended during intubation. It may be useful for preventing gastric insufflation during bag-mask ventilation. These issues are discussed separately. (See "Rapid sequence intubation in adults", section on 'Protection (cricoid pressure) and positioning'.)
- Oropharyngeal and nasopharyngeal airways can be useful adjuncts. We encourage their use when performing bag-mask ventilation. (See "Basic airway management in adults", section on 'Airway adjuncts'.)
- Continuous waveform capnography (performed in addition to clinical assessment) is recommended for both confirming and monitoring correct tracheal tube placement, and for monitoring the quality of CPR and the return of spontaneous circulation. If waveform capnography is not available, a non-waveform CO_2 detector may be used, in addition to clinical assessment. (See "Carbon dioxide monitoring (capnography)", section on 'Clinical applications for intubated patients'.)

MANAGEMENT OF SPECIFIC ARRHYTHMIAS

Sudden cardiac arrest

Ventricular fibrillation and pulseless ventricular tachycardia — Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) are non-perfusing rhythms emanating from the ventricles, for which early rhythm identification and defibrillation, are the mainstays of treatment. The most recent version of the AHA algorithm for the management of cardiac arrest can be accessed here (AHA cardiac arrest algorithm) (algorithm 2 and figure 1). Excellent cardiopulmonary resuscitation (CPR) is performed until the rescuer is ready to perform early defibrillation and is continued until adequate spontaneous circulation is achieved. Treatable underlying causes should be identified and managed as quickly as possible (table 3) [8,23].

Begin performing excellent chest compressions as soon as sudden cardiac arrest (SCA) is recognized and continue while the defibrillator is being attached. If a defibrillator is not immediately available, continue CPR until one is obtained. As soon as a defibrillator is available, attach it to the patient (figure 2) and charge it while continuing CPR, then stop compressions to assess the rhythm and defibrillate if appropriate (eg, VF or pulseless VT is present). If asystole or pulseless electrical activity (PEA) is present, continue CPR. Resume CPR immediately after any shock is given. (See "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest", section on 'VF and pulseless VT'.)

In the case of a witnessed cardiac arrest, perform defibrillation as quickly as possible. Decreased time to defibrillation improves the likelihood of successful conversion to a perfusing rhythm and of patient survival.

Biphasic defibrillators are recommended because of their increased efficacy at lower energy levels [28-30]. The ACLS Guidelines recommend that when employing a biphasic defibrillator clinicians use the initial dose of energy recommended by the manufacturer (120 to 200 J). If this dose is not known, the maximal dose may be used. We suggest a first defibrillation at maximal energy for VF or pulseless VT.

The ACLS Guidelines recommend the resumption of CPR immediately after defibrillation without rechecking for a pulse. CPR should not be interrupted to assess the rhythm, and rhythm checks and additional shocks should be performed no more frequently than every 2 minutes. (See "Basic life support (BLS) in adults", section on 'Phases of resuscitation' and "Basic life support (BLS) in adults", section on 'Defibrillation'.)

If VF or pulseless VT persists after at least one attempt at defibrillation and 2 minutes of CPR, give epinephrine (1 mg IV every 3 to 5 minutes) while CPR is performed continuously. In the 2015 ACLS update, vasopressin was removed from the treatment algorithm for cardiac arrest.

Some study results have raised doubts about the benefit of epinephrine [31,32]. Other researchers theorize that high concentrations of circulating catecholamines may be harmful in patients who experience a return of spontaneous circulation (ROSC), and that lower doses of epinephrine or longer dosing intervals may be prudent when treating VF or pulseless VT [31,33,34]. However, pending more conclusive data or a formal change in ACLS protocols, we suggest giving epinephrine in accordance with the existing Guidelines. (See "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest", section on 'VF or VT arrest and vasopressors'.)

Evidence suggests that antiarrhythmic drugs provide little survival benefit in refractory VF or pulseless VT. Nevertheless, the current ACLS Guidelines state that they may be used in certain situations. The

timing of antiarrhythmic use is not specified. We suggest that antiarrhythmic drugs be considered after a second unsuccessful defibrillation attempt in anticipation of a third shock.

- Amiodarone (300 mg IV with a repeat dose of 150 mg IV as indicated) may be administered in VF or pulseless VT unresponsive to defibrillation, CPR, and epinephrine.
- Lidocaine (1 to 1.5 mg/kg IV, then 0.5 to 0.75 mg/kg every 5 to 10 minutes) may be used if amiodarone is unavailable.
- Magnesium sulfate (2 g IV, followed by a maintenance infusion) may be used to treat polymorphic ventricular tachycardia consistent with torsade de pointes, but is not recommended for routine use in adult cardiac arrest patients. (See 'Irregular wide complex' below.)

Refractory VF or pulseless VT may be caused by an acute coronary syndrome (ACS), in which case percutaneous coronary intervention can be performed if the patient is successfully resuscitated and the procedure is feasible. Note that following cardiac arrest the ECG may be insensitive for ACS; cardiology consultation is needed for patients with return of spontaneous circulation (ROSC) [4]. Causes other than ACS can lead to SCA (table 3).

In summary, the ROSC in VF and pulseless VT hinges on early defibrillation and excellent CPR. Although, the ACLS Guidelines advocate the appropriate use of advanced airway management and treatment with specific medications, these interventions have not been shown to improve survival in SCA. Therefore, such interventions must never be initiated at the expense of performing excellent CPR and early defibrillation.

Asystole and pulseless electrical activity — Asystole is defined as a complete absence of demonstrable electrical and mechanical cardiac activity. Pulseless electrical activity (PEA) is defined as any one of a heterogeneous group of organized electrocardiographic rhythms without sufficient mechanical contraction of the heart to produce a palpable pulse or measurable blood pressure. By definition, asystole and PEA are non-perfusing rhythms requiring the initiation of excellent CPR immediately when either is present. The most recent version of the AHA algorithm for the management of cardiac arrest can be accessed here (AHA cardiac arrest algorithm) (algorithm 2).

In the ACLS Guidelines, asystole and PEA are addressed together because successful management for both depends on excellent CPR, vasopressor treatment (ie, epinephrine), and rapid reversal of underlying causes, such as hypoxia, hyperkalemia, poisoning, and hemorrhage [8,23]. Asystole may be the result of a primary or secondary cardiac conduction abnormality, possibly from end-stage tissue hypoxia and metabolic acidosis, or, rarely, the result of excessive vagal stimulation. It is crucial to identify and treat potential secondary causes of asystole or PEA as rapidly as possible. Some causes (eg, tension pneumothorax, cardiac tamponade) result in ineffective CPR. Do not hesitate to perform invasive procedures to treat suspected secondary causes; if the patient is receiving CPR, there is little chance the intervention will make the situation worse. The accompanying tables describe important secondary causes of cardiac arrest (table 3).

After initiating CPR, treat reversible causes as appropriate and administer epinephrine (1 mg IV every 3 to 5 minutes). Vasopressin was removed from the treatment algorithm in the 2015 update. As with VF and pulseless VT, evidence supporting the benefit of epinephrine in patients with asystole or PEA is limited and further study is needed. Neither asystole nor PEA responds to defibrillation. Atropine is no longer recommended for the treatment of asystole or PEA. Cardiac pacing is ineffective for cardiac arrest and not recommended. (See "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest".)

In summary, treatment for asystole and PEA consists of early identification and treatment of reversible causes and excellent CPR with epinephrine administration until either ROSC or a shockable rhythm occurs.

Monitoring — The ACLS Guidelines encourage the use of clinical and physiologic monitoring to optimize the performance of CPR and to detect the return of spontaneous circulation (ROSC) [5]. Assessment and immediate feedback about important clinical parameters, such as the rate and depth of chest compressions, adequacy of chest recoil between compressions, and rate and force of ventilations, can improve CPR. End-tidal carbon dioxide ($EtCO_2$) measurements from continuous waveform capnography (using nasal sampling if performing bag-mask ventilation (BMV) or tracheal tube sampling in intubated patients) accurately reflect cardiac output and cerebral perfusion pressure, and therefore the quality of CPR. Sudden, sustained increases in $EtCO_2$ during CPR indicate a ROSC while decreasing $EtCO_2$ during CPR may indicate inadequate compressions. Except for the initial pulse check to determine pulselessness, the need for checking a pulse during resuscitation is obviated by the use of $EtCO_2$ which enables determination of both ROSC and CPR quality. (See "Carbon dioxide monitoring (capnography)", section on 'Effectiveness of CPR' and "Carbon dioxide monitoring (capnography)", section on 'Return of spontaneous circulation'.)

Data from other physiologic monitors is less likely to be available in patients with SCA, but measurements obtained from arterial and central venous catheters provide useful feedback about the quality of CPR and ROSC [23]. Measurements of arterial relaxation provide a reasonable approximation of coronary perfusion pressure. During CPR, a reasonable goal is to maintain the arterial relaxation (or "diastole") pressure above 20 mmHg. Central venous oxygen saturation ($SCVO_2$) provides information about oxygen delivery and cardiac output. During CPR, a reasonable goal is to maintain $SCVO_2$ above 30 percent.

Bradycardia — Bradycardia is defined conservatively as a heart rate below 60 beats per minute, but symptomatic bradycardia generally entails rates below 50 beats per minute. The ACLS Guidelines recommend that clinicians not intervene unless the patient exhibits evidence of inadequate tissue perfusion thought to result from the slow heart rate [8,23]. The most recent version of the AHA algorithm for the management of bradycardia can be accessed here (AHA cardiac arrest algorithm) (algorithm 3). Signs and symptoms of inadequate perfusion include hypotension, altered mental status, signs of shock, ongoing ischemic chest pain, and evidence of acute pulmonary edema. Hypoxemia is a common cause of bradycardia; look for signs of labored breathing (eg, increased respiratory rate, retractions, paradoxical abdominal breathing) and low oxygen saturation. Mild symptoms may not warrant treatment other than supplemental oxygen.

If any significant symptoms are present in the setting of bradycardia, administer atropine (if easily done) and immediately prepare to treat the patient with transcutaneous pacing or an infusion of a chronotropic agent (dopamine or epinephrine). Do not delay treatment with transcutaneous pacing or a chronotropic agent in order to give atropine.

The initial dose of atropine is 0.5 mg IV. This dose may be repeated every 3 to 5 minutes to a total dose of 3 mg. Do not give atropine if there is evidence of a high degree (second degree [Mobitz] type II or third degree) atrioventricular (AV) block [35]. Atropine exerts its antibradycardiac effects at the AV node and is unlikely to be effective if a conduction block exists at or below the Bundle of His, or in transplanted hearts, which lack vagal innervation. Atropine may be harmful in the setting of cardiac ischemia. (See "Second degree atrioventricular block: Mobitz type II" and "Third degree (complete) atrioventricular block".)

Before using transcutaneous pacing, assess whether the patient can perceive the pain associated with this procedure and if so provide appropriate sedation and analgesia whenever possible. Infusions of dopamine are dosed at 2 to 10 mcg/kg per minute, while epinephrine is given at 2 to 10 mcg per minute. Each is titrated to the patient's response. (See "Procedural sedation in adults".)

If neither transcutaneous pacing nor infusion of a chronotropic agent resolves the patient's symptoms, prepare for transvenous pacing and obtain expert consultation if available. Patients requiring transcutaneous or transvenous pacing also require cardiology consultation, and admission for evaluation for permanent pacemaker placement.

Common toxicologic causes of symptomatic bradycardia include supratherapeutic levels of beta-blockers, calcium channel blockers, and Digoxin. These poisonings are discussed separately. (See "Beta blocker poisoning" and "Calcium channel blocker poisoning" and "Digitalis (cardiac glycoside) poisoning".)

Tachycardia

Approach — Tachycardia is defined as a heart rate above 100 beats per minute, but symptomatic tachycardia generally involves rates over 150 beats per minute, unless underlying ventricular dysfunction exists [8,23]. Management of tachyarrhythmias is governed by the presence of clinical symptoms and signs caused by the rapid heart rate. The most recent version of the AHA algorithm for the management of tachycardia can be accessed here (AHA cardiac arrest algorithm) (algorithm 4).

The fundamental approach is as follows: First determine if the patient is unstable (eg, manifests ongoing ischemic chest pain, acute mental status changes, hypotension, signs of shock, or evidence of acute pulmonary edema). Hypoxemia is a common cause of tachycardia; look for signs of labored breathing (eg, increased respiratory rate, retractions, paradoxical abdominal breathing) and low oxygen saturation.

If instability is present and appears related to the tachycardia, treat immediately with synchronized cardioversion, unless the rhythm is sinus tachycardia [36]. Some cases of supraventricular tachycardia may respond to immediate treatment with a bolus of adenosine (6 to 12 mg IV) without the need of cardioversion. Whenever possible, assess whether the patient can perceive the pain associated with cardioversion, and if so provide appropriate sedation and analgesia. (See "Procedural sedation in adults".)

In the stable patient, use the electrocardiogram (ECG) to determine the nature of the arrhythmia. In the urgent settings in which ACLS algorithms are most often employed, specific rhythm identification may not be possible. Nevertheless, by performing an orderly review of the ECG, one can determine appropriate management. Three questions provide the basis for assessing the electrocardiogram in this setting:

- Is the patient in a sinus rhythm?
- Is the QRS complex wide or narrow?
- Is the rhythm regular or irregular?

More detailed approaches to rhythm determination in tachycardia are discussed separately. (See "Clinical manifestations, diagnosis, and evaluation of narrow QRS complex tachycardias" and "Approach to the diagnosis of wide QRS complex tachycardias" and "Overview of the acute management of tachyarrhythmias".)

Regular narrow complex — Sinus tachycardia and supraventricular tachycardia are the major causes of a regular narrow complex arrhythmia [8,23]. Sinus tachycardia is a common response to fever, anemia, shock, sepsis, pain, heart failure, or any other physiologic stress. No medication is needed to treat sinus tachycardia; care is focused on treating the underlying cause. (See "Sinus tachycardia:

Evaluation and management".)

Supraventricular tachycardia (SVT) is a regular tachycardia most often caused by a reentrant mechanism within the conduction system (algorithm 4). The QRS interval is usually narrow but can be longer than 120 ms if a bundle branch block (ie, SVT with aberrancy or fixed bundle branch block) is present. Vagal maneuvers, which may block conduction through the AV node and result in interruption of the reentrant circuit, may be employed on appropriate patients while other therapies are prepared. Vagal maneuvers alone (eg, Valsalva, carotid sinus massage), convert up to 25 percent of SVTs to sinus rhythm, while Valsalva followed immediately by supine repositioning with a passive leg raise has been shown to be even more effective. SVT refractory to vagal maneuvers is treated with adenosine [37,38]. (See "Overview of the acute management of tachyarrhythmias" and "Clinical manifestations, diagnosis, and evaluation of narrow QRS complex tachycardias" and "Reentry and the development of cardiac arrhythmias" and "Vagal maneuvers".)

Because of its extremely short half-life, adenosine (6 to 12 mg IV) is injected as rapidly as possible into a large proximal vein, followed immediately by a 20 mL saline flush and elevation of the extremity to ensure the drug enters the central circulation before it is metabolized. If the first dose of adenosine does not convert the rhythm, a second and third dose of 12 mg IV may be given. Larger doses (eg, 18 mg IV) may be needed in patients taking theophylline or theobromine, or who consume large amounts of caffeine; smaller doses (eg, 3 mg IV) should be given to patients taking dipyridamole or carbamazepine, those with transplanted hearts, or when injecting via a central vein.

Prior to injection, warn the patient about transient side effects from adenosine, including chest discomfort, dyspnea, and flushing, and give reassurance that these effects are very brief. Perform continuous ECG recording during administration. If adenosine fails to convert the SVT, consider other etiologies for this rhythm, including atrial flutter or a non-reentrant SVT, which may become apparent on the ECG when AV nodal conduction is slowed.

If conversion attempts fail, initiate rate control with either an intravenous nondihydropyridine calcium channel blocker or a beta blocker. Agents to choose from include: diltiazem, verapamil, and a number of beta blockers, including metoprolol, atenolol, esmolol, and labetalol. (See "Control of ventricular rate in atrial fibrillation: Pharmacologic therapy", section on 'Pharmacologic treatment'.)

Irregular narrow complex — Irregular narrow-complex tachycardias may be caused by atrial fibrillation, atrial flutter with variable atrioventricular (AV) nodal conduction, multifocal atrial tachycardia (MAT), or sinus tachycardia with frequent premature atrial beats (algorithm 4). Of these, atrial fibrillation is most common [8,23].

The initial goal of treatment in stable patients is to control the heart rate using either a nondihydropyridine calcium channel blocker (diltiazem 15 to 20 mg IV over 2 minutes, repeat at 20 to 25 mg IV after 15 minutes, or verapamil 2.5 to 5 mg IV over 2 minutes followed by 5 to 10 mg IV every 15 to 30 minutes) or a beta blocker (eg, metoprolol 5 mg IV for 3 doses every 2 to 5 minutes; then up to 200 mg PO every 12 hours). The management of atrial fibrillation and SVT is discussed in detail separately. (See "Overview of atrial fibrillation" and "Rhythm control versus rate control in atrial fibrillation" and "Control of ventricular rate in atrial fibrillation: Pharmacologic therapy" and "Clinical manifestations, diagnosis, and evaluation of narrow QRS complex tachycardias" and "Multifocal atrial tachycardia".)

Calcium channel blockers and beta-blockers may cause or worsen hypotension. Patients should be closely monitored while the drug is given, and patients at greater risk of developing severe hypotension (eg, elders) often require loading doses that are below the usual range. Combination therapy with a beta blocker and calcium channel blocker increases the risk of severe heart block.

Diltiazem is suggested in most instances for the management of acute atrial fibrillation with rapid ventricular response. Beta-blockers may also be used and may be preferred in the setting of an acute coronary syndrome. Beta-blockers are more effective for chronic rate control. For atrial fibrillation associated with hypotension, amiodarone may be used (150 mg IV over 10 minutes, followed by 1 mg/min drip for six hours, and then 0.5 mg/min), but the possibility of conversion to sinus rhythm must be considered [39]. For atrial fibrillation associated with acute heart failure, amiodarone or digoxin may be used for rate control. Treatment of MAT includes correction of possible precipitants, such as hypokalemia and hypomagnesemia. The ACLS Guidelines recommend consultation with a cardiologist for these arrhythmias.

Cardioversion of stable patients with irregular narrow complex tachycardias should **NOT** be undertaken without considering the risk of embolic stroke. If the duration of atrial fibrillation is known to be less than 48 hours, the risk of embolic stroke is low, and the clinician may consider electrical or chemical cardioversion [40]. A number of medications can be used for chemical cardioversion and the best drug varies according to clinical circumstance. The questions of whether chemical cardioversion is appropriate and which agent to select are reviewed separately.

Regular wide complex — A regular, wide-complex tachycardia is generally ventricular in etiology (algorithm 4). Aberrantly conducted supraventricular tachycardias may also be seen. Because differentiation between ventricular tachycardia (VT) and SVT with aberrancy can be difficult, assume VT is present. Treat clinically stable undifferentiated wide-complex tachycardia with antiarrhythmics or elective synchronized cardioversion [8,23].

In cases of regular, wide-complex tachycardia with a monomorphic QRS complex, adenosine may be used for diagnosis and treatment. Do **NOT** give adenosine to patients who are unstable or manifest wide-complex tachycardia with an irregular rhythm or a polymorphic QRS complex. Adenosine is unlikely to affect ventricular tachycardia but is likely to slow or convert SVT with aberrancy. Dosing is identical to that used for SVT. (See 'Regular narrow complex' above.)

Other antiarrhythmics that may be used to treat stable patients with regular, wide-complex tachycardia include procainamide (20 mg/min IV), amiodarone (150 mg IV given over 10 minutes, repeated as needed to a total of 2.2 g IV over the first 24 hours), and sotalol (100 mg IV over 5 minutes). A procainamide infusion continues until the arrhythmia is suppressed, the patient becomes hypotensive, the QRS widens 50 percent beyond baseline, or a maximum dose of 17 mg/kg is administered. Procainamide and sotalol should be avoided in patients with a prolonged QT interval. If the wide-complex tachycardia persists, in spite of pharmacologic therapy, elective cardioversion may be needed. The ACLS Guidelines recommend expert consultation for all patients with wide complex tachycardia.

SVT with aberrancy, if **DEFINITIVELY** identified (eg, old ECG demonstrates bundle branch block), may be treated in the same manner as narrow-complex SVT, with vagal maneuvers, adenosine, or rate control. (See 'Irregular narrow complex' above.)

Irregular wide complex — A wide complex, irregular tachycardia may be atrial fibrillation with preexcitation (eg, Wolf Parkinson White syndrome), atrial fibrillation with aberrancy (bundle branch block), or polymorphic ventricular tachycardia (VT)/torsades de pointes (algorithm 4) [8,23]. Use of atrioventricular (AV) nodal blockers in wide complex, irregular tachycardia of unclear etiology may precipitate ventricular fibrillation (VF) and patient death, and is contraindicated. Such medications include beta blockers, calcium channel blockers, digoxin, and adenosine. To avoid inappropriate and possibly dangerous treatment, the ACLS Guidelines suggest assuming that any wide complex, irregular tachycardia is caused by preexcited atrial fibrillation.

Patients with a wide complex, irregular tachycardia caused by preexcited atrial fibrillation usually manifest extremely fast heart rates (generally over 200 beats per minute) and require immediate electric cardioversion. In cases where electric cardioversion is ineffective or unfeasible, or atrial fibrillation recurs, antiarrhythmic therapy with procainamide, amiodarone, or sotalol may be given. The ACLS Guidelines recommend expert consultation for all patients with wide complex tachycardia. Dosing for antiarrhythmic medications is described above. (See 'Regular wide complex' above.)

Treat polymorphic VT with emergent defibrillation. Interventions to prevent recurrent polymorphic VT include correcting underlying electrolyte abnormalities (eg, hypokalemia, hypomagnesemia) and, if a prolonged QT interval is observed or thought to exist, stopping all medications that increase the QT interval. Magnesium sulfate (2 g IV, followed by a maintenance infusion) can be given to prevent polymorphic VT associated with familial or acquired prolonged QT syndrome [41].

A clinically stable patient with atrial fibrillation and a wide QRS interval **KNOWN** to stem from a preexisting bundle branch block (ie, old ECG demonstrates preexisting block) may be treated in the same manner as a narrow-complex atrial fibrillation.

Alternative methods for medication administration — Whenever possible, ACLS medications should be given intravenously. When IV access cannot be established, intraosseous (IO) lines are safe, effective, and can be placed efficiently [8,23]. Medication doses for IO administration are identical to those for IV therapy. If neither IV nor IO access can be established, some medications may be given via the tracheal tube. (See "Intraosseous infusion".)

Multiple studies have demonstrated that lidocaine, epinephrine, atropine, vasopressin, and naloxone are absorbed via the trachea [23]; however, the serum drug concentrations achieved using this route are unpredictable. If the patient already has peripheral, intraosseous, or central venous access, these are always the preferred routes for drug administration. When unable to obtain such access expeditiously, one may use the endotracheal tube while attempting to establish vascular or intraosseous access. At no point should excellent CPR be interrupted to obtain vascular access.

Doses for tracheal administration are 2 to 2.5 times the standard IV doses and medications should be diluted in 5 to 10 mL of sterile water or normal saline before injection down the tracheal tube.

POST-RESUSCITATION CARE — The ACLS Guidelines recommend a combination of goal-oriented interventions provided by an experienced multidisciplinary team for all cardiac arrest patients with return of spontaneous circulation [8,23,42]. Important objectives for such care include:

- Optimizing cardiopulmonary function and perfusion of vital organs
- Managing acute coronary syndromes
- Implementing strategies to prevent and manage organ system dysfunction and injury

Management of the post-cardiac arrest patient is reviewed separately. (See "Post-cardiac arrest management in adults".)

TERMINATION OF RESUSCITATIVE EFFORTS — Determining when to stop resuscitation efforts in cardiac arrest patients is difficult, and little data exist to guide decision-making. Factors associated with poor and good outcomes are discussed in detail separately. (See "Prognosis and outcomes following sudden cardiac arrest in adults".)

Physician survey data and clinical practice guidelines suggest that factors influencing the decision to stop resuscitative efforts include [43-47]:

- Duration of resuscitative effort >30 minutes without a sustained perfusing rhythm
- Initial electrocardiographic rhythm of asystole
- Prolonged interval between estimated time of arrest and initiation of resuscitation
- Patient age and severity of comorbid disease
- Absent brainstem reflexes
- Normothermia

More objective endpoints of resuscitation have been proposed. Of these, the best predictor of outcome may be the end-tidal carbon dioxide ($EtCO_2$) level following 20 minutes of resuscitation [48-50]. $EtCO_2$ values are a function of CO_2 production and venous return to the right heart and pulmonary circulation. A very low $EtCO_2$ (<10 mmHg) following prolonged resuscitation (>20 minutes) is a sign of absent circulation and a strong predictor of acute mortality [48-50]. It is crucial to note that low $EtCO_2$ levels may also be caused by a misplaced (esophageal) endotracheal tube, and this possibility needs to be excluded before the decision is made to terminate resuscitative efforts. (See "Carbon dioxide monitoring (capnography)".)

Resuscitation in the emergency department does not appear to be superior to field resuscitation by emergency medical services (EMS) personnel. Therefore, EMS personnel should not be required to transport all victims of sudden cardiac arrest to the hospital, if further resuscitation is deemed futile [51,52].

Large, retrospective cohort studies have assessed criteria (BLS and ALS) for the prehospital termination of resuscitative efforts in cardiac arrest, initially described in the OPALS study [53,54]. Both BLS and ALS criteria demonstrated high specificity for identifying out-of-hospital cardiac arrest patients with little or no chance of survival. Studies of another clinical decision rule suggest that it too accurately predicts survival and would reduce unnecessary transports substantially if implemented [51,55].

According to a systematic review of 12 small trials, most of which studied convenience samples of patients with sudden cardiac arrest (n = 568), bedside echocardiography may be helpful for assessing prognosis [56]. In this review, the pooled sensitivity and specificity of echocardiography to predict the return of spontaneous circulation (ROSC) were 91.6 and 80 percent respectively (95% CI for sensitivity 84.6 to 96.1%; 95% CI for specificity 76.1 to 83.6%). Of the 190 patients found to have cardiac wall motion, 98 (51.6 percent) achieved ROSC, whereas only 9 (2.4 percent) of the 378 without cardiac wall motion did so. Limitations of the individual studies prevented the authors from assessing survival to discharge or survival with good neurologic function.

The authors of this review emphasize that echocardiography results should not be the sole basis for terminating resuscitative efforts but may serve as an adjunct to clinical assessment. Bedside echocardiography must never interfere with resuscitation efforts, and should not interrupt or delay resumption of CPR, except in cases where the ultrasound is being obtained strictly to confirm absence of wall motion when a decision to terminate resuscitative efforts is imminent.

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient information: Ventricular fibrillation (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Cardiopulmonary resuscitation (CPR) and early defibrillation for treatable arrhythmias remain the cornerstones of basic and advanced cardiac life support (ACLS). Excellent chest compressions without interruption are the key to successful CPR (table 1). (See 'Excellent basic life support and its importance' above.)
- The performance of teams providing ACLS improves when there is a single designated leader who asks for and accepts helpful suggestions from members of the team, and when the team practices clear, closed-loop communication. (See 'Resuscitation team management' above.)
- Begin properly performed CPR immediately for any patient with suspected cardiac arrest. Other initial interventions for ACLS include administering oxygen when needed, establishing intravenous access, placing the patient on a cardiac and oxygen saturation monitor, and obtaining an electrocardiogram (ECG). (See 'Initial management and ECG interpretation' above.)
- In adults, properly performed chest compressions take priority over ventilation during the initial period of basic life support. When ventilating the patient in cardiac arrest, give 100 percent oxygen, use low respiratory rates (approximately 6 breaths per minute), and avoid hyperventilation, which is harmful. (See 'Airway management while performing ACLS' above.)
- For the purposes of ACLS, ECG interpretation is guided by three questions:
  - Is the rhythm fast or slow?
  - Are the QRS complexes wide or narrow?
  - Is the rhythm regular or irregular?
- The basic approach and important aspects of management for each arrhythmia covered by the ACLS Guidelines are discussed in the text and summarized in the accompanying algorithms (see 'Management of specific arrhythmias' above):
  - Cardiac arrest (ventricular fibrillation; pulseless ventricular tachycardia; asystole; pulseless electrical activity): (algorithm 2 and figure 1)
  - Bradycardia with pulse: (algorithm 3)
  - Tachycardia with pulse: (algorithm 4)

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