Supportive data for advanced cardiac life support in adults with sudden cardiac arrest

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INTRODUCTION

Sudden cardiac arrest (SCA) and sudden cardiac death (SCD) refer to the sudden cessation of cardiac activity with hemodynamic collapse, often due to sustained ventricular tachycardia/ventricular fibrillation. Other causes of SCA and SCD are asystole and pulseless electrical activity. These events most commonly occur in patients with structural heart disease (that may not have been previously diagnosed), particularly coronary heart disease. (See "Pathophysiology and etiology of sudden cardiac arrest".)

The event is referred to as SCA (or aborted SCD) if an intervention (eg, defibrillation) results in the return of spontaneous circulation (ROSC) and restored circulation. The event is called SCD if the patient dies. However, the use of SCD to describe both fatal and nonfatal cardiac arrest persists by convention. (See "Overview of sudden cardiac arrest and sudden cardiac death", section on 'Definitions'.)

The treatment of SCA consists of emergency resuscitation followed, in survivors, by immediate post-resuscitative care and attempted long-term prevention of recurrence using pharmacologic and nonpharmacologic interventions. Over time, the frequency of cardiopulmonary resuscitation (CPR) performed by bystanders has increased, and the interval between collapse and defibrillation has decreased, both of which are particularly important in survival [1,2]. Despite these improvements as well as advances in the treatment of heart disease, the outcome

of patients experiencing SCA remains poor. (See "Prognosis and outcomes following sudden cardiac arrest in adults".)

In reality, there are only two out of hospital therapies of resuscitation that have been shown to be associated with improved survival: excellent, prompt chest compressions and early defibrillation. Resuscitation should focus on providing these two elements with the highest quality. This is not to say that advanced cardiac life support (ACLS) therapies should be withheld if indicated; however, shockable rhythms should first be defibrillated, and excellent CPR (either compressions only, or compressions and mouth to mouth breathing) should be performed. Any other interventions should be delayed until these first-line therapies are implemented. In general, the performance of the second-line interventions (as part of ACLS) should rarely interfere with defibrillation and excellent CPR.

The only other therapy has shown to be associated with a neurologically favorable survival advantage is targeted temperature management (TTM) in the post-arrest care. This is discussed in greater detail separately. (See 'Targeted temperature management' below and "Initial assessment and management of the adult post-cardiac arrest patient", section on 'Temperature management'.)

The data supporting advanced cardiac life support (ACLS) recommendations for the management of SCA will be reviewed here. The performance of ACLS, controversies surrounding cardiopulmonary resuscitation, and issues related to the prevention of recurrent SCA in survivors are discussed separately. (See "Advanced cardiac life support (ACLS) in adults" and "Adult basic life support (BLS) for health care providers" and "Therapies of uncertain benefit in basic and advanced cardiac life support" and "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy" and "Pharmacologic therapy in survivors of sudden cardiac arrest".)

VF AND PULSELESS VT

The management of ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) is the same (algorithm 1) [3]. Survival depends upon prompt, high-quality cardiopulmonary resuscitation (CPR) with chest compressions and the earliest defibrillation possible to reestablish organized electrical activity with a stable sinus or supraventricular rhythm. Excellent CPR is a priority and should be maintained throughout the resuscitative effort; pauses should only occur during rhythm analysis and defibrillation (as soon as possible for the first defibrillation and then at two-minute intervals, and when required to deliver ventilations in an un-intubated patient by bag-mask ventilation at a ratio of 30:2). Other procedures, such as

vascular access, vasopressor administration, and intubation, should not be performed in the initial resuscitation efforts (ie, first few minutes) unless they can be performed without interrupting CPR and defibrillation. Multiple studies have reported that intubation should not be attempted in adults until return of spontaneous circulation (ROSC) unless bag-mask ventilation is ineffective or there has been a prolonged period of resuscitation [4-6]. Any adjunctive procedure requiring discontinuation of CPR should occur during rhythm checks, as this minimizes interruptions in perfusion and the build-up necessary to reach threshold perfusion pressures once compressions are restarted. Interruptions should be brief and should never create prolonged delays in resumption of CPR. (See "Advanced cardiac life support (ACLS) in adults".)

Specific issues relating to the management of VF and pVT, including technical aspects of defibrillation, are discussed below. The use of automated external defibrillators (AED) for the treatment of cardiac arrest is presented separately. (See "Automated external defibrillators".)

Defibrillation — The only effective approach for the treatment of VF and pVT is defibrillation, with earlier efforts yielding better outcomes. VF rarely, if ever, terminates spontaneously or after delivery of an antiarrhythmic drug. (See "Cardioversion for specific arrhythmias", section on 'Ventricular fibrillation'.)

Timing — The success of defibrillation and patient survival depends upon the duration of the arrhythmia and the promptness of defibrillation [2,7-9]:

- When VF has been present for seconds to a few minutes and the fibrillatory waves are coarse, the success rate for terminating VF with defibrillation is high.
- As VF continues, the fibrillatory waves become finer (waveform 1) [10].
- When VF continues for more than four minutes, especially if not accompanied by excellent CPR, irreversible damage to the central nervous system and other organs begins, which can reduce survival even if defibrillation is successful [11-13].

There is strong evidence that longer interruptions in chest compressions reduce the likelihood of successful defibrillation and lower the odds of survival [14-18]. In a prospective observational cohort of 506 adult patients with out-of-hospital cardiac arrest, patients with the highest chest compression fractions (61 to 80 percent and 81 to 100 percent), defined as the proportion of time in cardiac arrest without spontaneous circulation during which chest compressions were being performed, had the greatest chance of survival to hospital discharge (adjusted odds ratios [OR] 3.01, 95% CI 1.37-6.58 and 2.33, 95% CI 0.96-5.63, respectively). As there are data indicating that shortening the duration between stopping compressions and delivering the

defibrillatory shock has a survival advantage, defibrillators should be fully charged prior to cessation of compressions, after which immediate rhythm identification and shock delivery is performed as indicated (this should typically occur in no more than three to five seconds) [18,19].

In a series of over 12,000 patients treated by Emergency Medical Services, 4546 had witnessed VF [2]. For these patients with witnessed VF, a shorter defibrillation response interval was significantly correlated with an increased chance of survival to hospital discharge. The analysis yielded an odds ratio for survival of 0.88 for every one-minute increase in response time, which corresponds to a 3 to 5 percent drop in survival for each minute of delay to defibrillation [2].

In a trial of 9933 patients with out-of-hospital cardiac arrest who were randomly assigned to either 30 to 60 seconds of CPR or 180 seconds of CPR prior to initial cardiac rhythm analysis, there was no significant difference in survival to hospital discharge [20]. Although there has been debate about whether CPR should be performed before defibrillation, practically, in nearly all cardiac arrests, rescuers should perform excellent compressions as they await the arrival and deployment of an AED or while a monitor/defibrillator is being placed and charged. Thus, CPR should generally be performed prior to defibrillation in these situations.

Early defibrillation of VT/VF in patients with in-hospital cardiac arrest has also been shown to improve survival, in both the short term and long term. Among a cohort of 8119 with in-hospital cardiac arrest with VT/VF, patients who were defibrillated within two minutes of cardiac arrest had significantly better survival (compared with initial defibrillation at >2 minutes) at one year (26 versus 16 percent), three years (19 versus 11 percent), and five years (15 versus 9 percent) [21].

Based upon the above studies, the 2020 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation and emergency cardiovascular care suggest the initiation of CPR while awaiting the arrival of a defibrillator [22-25]. Once the defibrillator is attached to the patient, CPR should be briefly stopped to allow for a rhythm check and shock, if indicated (cessation of compressions should not occur until the defibrillator is fully charged), followed by immediate resumption of CPR for two minutes prior to any additional rhythm checks.

Defibrillatory waveforms — Defibrillators manufactured prior to 2000 deliver a monophasic wave of direct electrical current. Since then, "biphasic" devices have been developed, which reverse current polarity 5 to 10 milliseconds after discharge begins (figure 1).

Biphasic waveforms defibrillate more effectively and at lower energies than monophasic waveforms. They have a much higher likelihood of first shock efficacy, which is the basis for the current recommendation to deliver a single shock compared with earlier recommendations that

three shocks be delivered in rapid succession [3]. Data comparing the use of monophasic and biphasic waveforms in the treatment of VF are presented separately. (See "Basic principles and technique of external electrical cardioversion and defibrillation", section on 'Monophasic versus biphasic waveforms'.)

There have been case reports in the literature on the successful use of double sequence defibrillation for VF not terminated by maximal energy from a single defibrillator [26,27]. Importantly, this procedure is not warranted for those whose VF terminates with shock and then reinitiates. This procedure employs two external defibrillators used simultaneously or sequentially (one right after the other) to deliver higher energy with the intent to convert VF to a perfusing rhythm. Despite these case reports, two retrospective studies with small patient populations of OHCA showed disparate results; one reported no difference [26,27]. A recent cluster randomized study comparing dual-sequential defibrillation with vector change defibrillation versus standard defibrillation in patients with refractory VF demonstrated that dual-sequential defibrillation was superior in survival to hospital discharge and had a good neurologic outcome [28]. These data suggest that dual-sequential defibrillation should be performed for those with refractory VF.

There are accumulating data that show VF waveforms can be analyzed to predict the need for >3 shocks [29]. These individuals may particularly benefit from dual-sequential defibrillation.

VF or VT arrest and vasopressors — Despite conflicting data on survival, the 2020 guidelines concluded that it is appropriate to administer epinephrine (1 mg IV/IO every three to five minutes) for patients presenting with cardiac arrest [3,25,30]. Routine use of high-dose epinephrine is not supported by data and is not recommended. Additionally, the 2020 guidelines state that vasopressin alone or in combination with epinephrine may be considered during resuscitation from cardiac arrest but offers no advantage over epinephrine alone. Accordingly, we recommend that epinephrine be used as the sole vasopressor during resuscitation from cardiac arrest and that vasopressin no longer be used [3].

The totality of the evidence assessing the efficacy of epinephrine for out-of-hospital cardiac arrest suggest that, although ROSC and even overall survival may be increased with the use of epinephrine, meaningful survival with favorable neurologic outcome is not improved [31-34]. Whether improvement in post-ROSC care will be able to transform those without meaningful neurologic outcome is not clear. Thus, as the data are quite mixed in outcome, they do not support the routine use of epinephrine in the treatment of all patients, but should serve as an impetus for additional randomized controlled trials of this issue targeting subgroups who are more likely to benefit. Pending review and updating of ACLS protocols, it is reasonable to

include epinephrine in resuscitation protocols as an option for resuscitation personnel, and the decision whether to administer it can occur on a case-by-case basis.

Epinephrine has been studied in two randomized, double-blind, placebo-controlled trials, with mixed survival results [31,32].

- In the trial of 8014 patients in the United Kingdom with out-of-hospital cardiac arrest (PARAMEDIC-2) who were randomized to either epinephrine (1 mg doses, mean total dose 4.9 mg) or placebo, the primary outcome (30-day survival) was significantly higher in the group who received epinephrine (130 patients [3.2 percent] versus 94 patients [2.4 percent]; adjusted OR 1.47; 95% CI 1.09-1.97) [31]. Subgroup analysis showed a significant survival benefit for patients with a nonshockable initial rhythm (OR 2.10; 95% CI 1.11-3.98) that was not seen in patients with an initial shockable rhythm. However, patients receiving epinephrine had no significant improvement in survival with favorable neurologic outcome (score between 0 and 3 on modified Rankin scale) but among survivors were more likely to have severe neurologic impairment (modified Rankin scale score 4 or 5; 31 versus 18 percent of survivors).

- In a trial of 601 patients with out-of-hospital cardiac arrest who were randomized to either epinephrine (1 mg doses, mean total dose 5 mg) or placebo, patients who received epinephrine were more likely to have ROSC (24 versus 8 percent, OR 3.4, 95% CI 2.0-5.6) and survival to hospital admission, but there was no significant improvement in survival to hospital discharge (4 versus 1.9 percent, OR 2.2, 95% CI 0.7-6.3) [32].

- A 2019 meta-analysis, which combined the data from these two randomized trials, reported increased survival to hospital discharge for patients with nonshockable initial rhythms receiving epinephrine, with the results largely driven by the PARAMEDIC-2 data [33]. No significant improvement in survival to hospital discharge was seen in patients with an initial shockable rhythm.

The benefits of epinephrine (and other vasopressors) in the treatment of cardiac arrest have been questioned in numerous nonrandomized studies:

- In a prospective observational study of 417,188 adults with out-of-hospital cardiac arrest, investigators compared ROSC, survival, and neurologic outcome in patients who received epinephrine (15,030 patients, 3.6 percent) with those who did not receive epinephrine (402,158 patients, 96.4 percent) [35]. While there was a significantly greater ROSC in the epinephrine group (18.5 versus 5.7 percent using raw data, 18.3 versus 10.5 percent using propensity analysis), this did not translate into improved outcomes. In fact, using propensity analysis, patients receiving epinephrine had significantly lower rates of survival

at one month (5.1 versus 7.0 percent); survival with moderate or good cerebral performance (1.3 versus 3.1 percent); and survival with no, mild, or moderate neurological disability (1.3 versus 3.1 percent).

- Similar results were reported from a study of 1646 patients presenting to a single center between 2000 and 2012 following out-of-hospital cardiac arrest with ROSC, in which the chance of survival was significantly lower among patients who received epinephrine and decreased further as the cumulative dose of epinephrine during resuscitation increased [36].

- The early administration of epinephrine within two minutes following the initial defibrillation for VF/VT may be detrimental. In a prospective cohort study of 2978 patients with in-hospital cardiac arrest and a shockable rhythm (1510 patients with epinephrine administered within two minutes of defibrillation and 1468 propensity score matched patients without early epinephrine administration), patients who received early epinephrine had a significantly decreased likelihood of survival (OR 0.70, 95% CI 0.59-0.82) [37]. (See "Advanced cardiac life support (ACLS) in adults", section on 'Pulseless ventricular tachycardia and ventricular fibrillation'.)

In a 2019 Cochrane Database systematic review which looked only at randomized controlled trials (26 trials with 21,704 patients) in which standard-dose epinephrine was compared to placebo, high-dose epinephrine, or vasopressin (alone or in combination with epinephrine), the following findings were reported among patients receiving epinephrine compared with placebo [38]:

- Moderate quality evidence of ROSC (relative risk [RR] 2.86; 95% CI 2.21-3.71)
- Moderate quality evidence of survival to hospital discharge (RR 1.44; 95% CI 1.11-1.86)
- No significant difference in survival with favorable neurologic outcomes (RR 1.21; 95% CI 0.90-1.62)

Although the 2020 guidelines still allow vasopressin to be "considered" (ie, 2b) alone or in combination with epinephrine, we recommend that vasopressin no longer be used for SCA29. Although a single randomized controlled trial found higher rates of successful resuscitation and 24-hour survival with vasopressin compared with epinephrine, subsequent randomized trials and a meta-analysis failed to show an improvement in initial ROSC or survival to hospital discharge [39-42]. Furthermore, in a multicenter trial of 1442 patients, treatment of out-of-hospital cardiac arrest with a combination of epinephrine and vasopressin did not improve outcomes compared with treatment with epinephrine alone [43]. Because of this lack of benefit of vasopressin over epinephrine, the 2015 AHA guidelines removed vasopressin from the

treatment protocol (allowing for EMS and hospitals to remove it from resuscitation supplies) for patients with VF or pVT [3].

Because vasopressors given in the setting of ROSC may be detrimental, it is now strongly suggested that resuscitation be performed using quantitative waveform capnography so that ROSC may be more readily identified during ongoing compressions [44-46]. As soon as ROSC is obtained, no further vasopressors should generally be administered. (See "Advanced cardiac life support (ACLS) in adults", section on 'Airway management' and "Carbon dioxide monitoring (capnography)".)

Antiarrhythmic drugs — VF or VT may persist despite electrical countershock or recur after successful electrical countershock. The 2020 AHA guidelines state that IV/IO antiarrhythmic drug therapy may be considered in such cases, in particular for witnessed cardiac arrest in which the time to drug administration is shorter, although benefit from this therapy remains uncertain [3,25,47-49]. Antiarrhythmic drugs that may be used include amiodarone and lidocaine, with no guideline-based preference of a preferred agent. Some post-hoc nonrandomized data suggest that outcomes are better for both amiodarone and lidocaine administered intravenously (compared with intraosseously), but this has not been assessed in a randomized controlled trial [50,51].

The performance of ACLS, including the details of antiarrhythmic drug therapy, is discussed elsewhere. (See "Advanced cardiac life support (ACLS) in adults".)

Amiodarone — Intravenous amiodarone increases ROSC and survival to hospital admission compared with lidocaine in patients with refractory VF and pVT, However, there is no significant difference between amiodarone and lidocaine in terms of the patient-important outcomes of survival to hospital discharge and neurologically intact survival [47,52].

- In the ALIVE trial of 347 patients with out-of-hospital sudden cardiac arrest (SCA) and persistent or recurrent VF despite three defibrillation shocks, IV/IO epinephrine, and a further attempt at defibrillation, survival to hospital admission was significantly higher in patients receiving amiodarone (23 versus 12 percent for lidocaine, OR 2.17, 95% CI 1.21-3.83) [53]. Among patients who had the ROSC prior to antiarrhythmic administration, amiodarone led to an even higher rate of survival to admission (42 versus 27 percent with lidocaine).

  Despite these benefits, the in-hospital death rates in the two groups were 78 and 75 percent, similar to those in other studies, and there was **no significant difference in survival to hospital discharge** (nine and five patients in the amiodarone and lidocaine groups, respectively).

- In the ARREST trial of 504 patients with SCA due to VF or pVT who were not resuscitated after at least three defibrillation shocks, patients were randomly assigned to intravenous bolus amiodarone (300 mg) or placebo [54]. While the mean time to resuscitation and the total number of shocks delivered were similar in the two groups, survival to hospital admission was significantly greater in the amiodarone group (44 versus 34 percent with placebo), especially in patients who had the ROSC during defibrillation prior to receiving amiodarone (64 versus 41 percent placebo). Amiodarone, however, was associated with more adverse effects, including hypotension (59 versus 48 percent) and bradycardia requiring therapy (41 versus 25 percent). (See "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

  The ARREST trial was not sufficiently powered to detect differences in survival to hospital discharge, which did not differ significantly between the two groups, similar to the findings in the ALIVE trial.

Intravenous amiodarone is also effective for the acute suppression of life-threatening, hemodynamically significant ventricular tachyarrhythmias that recur despite therapy with other agents. Amiodarone can prevent recurrence of sustained spontaneous VT or VF in more than 50 percent of patients, and it has been approved for the acute treatment and prevention of VF and for hemodynamically significant VT that is refractory to other agents [55,56].

Lidocaine — There is no significant difference between lidocaine and amiodarone in terms of the patient-important outcomes of survival to hospital discharge and neurologically intact survival. The 2020 AHA guidelines state that either lidocaine or amiodarone can be administered during cardiac arrest for VT/VF refractory to shocks and epinephrine [25,48,49]. (See 'Amiodarone' above and "Advanced cardiac life support (ACLS) in adults", section on 'Pulseless patient in sudden cardiac arrest'.)

Comparison of amiodarone and lidocaine — In 2016, the first randomized trial (the ALPS study) was published that compared amiodarone, lidocaine, and placebo in patients with pVT or VF refractory to defibrillation and initial vasopressor therapy [57]. This study was the first to evaluate the effect of a new preparation of amiodarone that employs a solvent that is not associated with hypotension. In this trial of 3026 patients, there was no significant difference in the primary outcome (survival to hospital discharge) among the three groups, with no statistically significant improvement in survival for either the amiodarone or lidocaine groups compared with placebo (24.4 versus 23.7 versus 21.0 percent, respectively). Similarly, there was no significant improvement in the prespecified secondary outcome of favorable neurologic function at discharge among those who received amiodarone or lidocaine. Subgroup analysis identified a nonsignificant mortality decrease of 5 percent for either drug therapy in patients

with bystander-witnessed arrest (in whom CPR and activation of emergency medical systems likely occur earlier than in non-witnessed patients).

A prespecified analysis from the ALPS study was performed on a cohort of 1063 patients with cardiac arrest initially due to a nonshockable rhythm (ie, pulseless electrical activity or asystole) that subsequently evolved to a shockable rhythm during resuscitation efforts [58]. As with the primary cohort from the ALPS study (patients presenting with cardiac arrest due to pVT or VF), patients were randomized to amiodarone, lidocaine, or placebo. Similar to the findings among patients presenting with an initial shockable rhythm, for patients who evolved from a nonshockable to a shockable rhythm during resuscitation, there was no significant difference in the primary outcome (survival to hospital discharge) among the amiodarone, lidocaine, and placebo groups (4.1 versus 3.1 versus 1.9 percent, respectively).

These randomized trial data further support the approach as stated in the 2018 AHA focused update to the 2015 AHA guidelines, in which amiodarone and lidocaine "may be considered" for patients with VF or pVT which is refractory to initial treatments, especially when arrest is witnessed [48,49].

Magnesium sulfate — Data from randomized trials do NOT support the routine use of magnesium sulfate for the treatment of cardiac arrest [48,49]. However, observational data from a small number of patients suggest that intravenous magnesium sulfate is beneficial for the treatment of a VF or pVT arrest due to drug-induced prolonged QT interval associated with torsades de pointes [59]. Magnesium's primary benefit is in the prevention of recurrent episodes of VF, not in the termination of such. Defibrillation is generally necessary for termination. The 2020 AHA guidelines state that torsades de pointes is the only arrhythmia for which administration of magnesium sulfate should be considered [25,48,49]. (See "Acquired long QT syndrome: Clinical manifestations, diagnosis, and management".)

Targeted temperature management — The induction of mild to moderate hypothermia (target temperature 32 to 34ºC for 24 hours) has been shown to improve survival in patients successfully resuscitated after a cardiac arrest who are comatose. Improved neurologic outcome and reduced mortality have been demonstrated in multiple series of patients with VF arrest in whom spontaneous circulation was restored, even when the patient remained comatose after resuscitation. More recently, a large randomized controlled trial, which involved 939 patients with ROSC following cardiac arrest and compared target temperatures of 32 to 34 degrees with targeted temperatures of 34 to 36 degrees, showed no difference in survival between the groups [60]. A more recent and larger randomized controlled trial of 1900 patients with cardiac arrest showed no difference in outcomes between those randomized to hypothermia (33 degrees C) versus normothermia [61]. It is still unclear whether the benefits of

targeted temperature management are related to actual cooling or the prevention of post-ROSC fever. Targeted temperature management is discussed in greater detail elsewhere. (See "Advanced cardiac life support (ACLS) in adults", section on 'Post-resuscitation care' and "Initial assessment and management of the adult post-cardiac arrest patient", section on 'Temperature management'.)

PULSELESS ELECTRICAL ACTIVITY

In contrast to ventricular fibrillation or pulseless ventricular tachycardia, there is no role for defibrillation in the management of pulseless electrical activity (PEA) or asystole (algorithm 1). Excellent cardiopulmonary resuscitation (CPR), vasopressor therapy, and rapid treatment of reversible causes are the mainstays of PEA management. However, because CPR is ineffective in cardiac arrest due to cardiac tamponade and tension pneumothorax, the rapid identification and treatment of these eminently reversible causes must be of primary importance. Reversible causes include the five H's and T's (hypoxia, hypovolemia, hydrogen ion (acidosis), hypo-/hyperkalemia, hypothermia, toxins [especially narcotics and benzodiazepines], tamponade [cardiac], tension pneumothorax, thrombosis [pulmonary], thrombosis [coronary]) as defined by the 2015 AHA guidelines (table 1) [3]. While CPR with excellent chest compressions is performed, other rescuers should not be afraid to perform "heroic" procedures, even if no definitive confirmatory studies are available, for plausibly suspected causes of PEA.

CPR should be provided throughout the resuscitative effort, except during brief pauses at two-minute intervals to analyze the rhythm and assess for return of spontaneous circulation (ROSC). In a cohort of 3960 patients with out-of-hospital cardiac arrest and a nonshockable cardiac rhythm, 1774 patients were treated with ACLS before and 2186 patients were treated with ACLS after the revised 2005 guidelines, which increased the emphasis on chest compressions during resuscitation [62]. Patients treated after 2005 with an increased emphasis on chest compressions had significantly greater ROSC (34 versus 27 percent), one month survival (6.2 versus 4.1 percent), and favorable neurologic outcomes (5.1 versus 3.4 percent). (See "Advanced cardiac life support (ACLS) in adults", section on 'Asystole and pulseless electrical activity'.)

PEA and vasopressors — The efficacy of epinephrine (1 mg IV/IO push every three to five minutes) for PEA remains uncertain, but it remains a part of the 2015 AHA guidelines for the treatment of cardiac arrest with PEA [3,35]. In a secondary analysis of a cardiac arrest registry, among a cohort of 32,101 patients with out-of-hospital cardiac arrest and a nonshockable initial rhythm (published after the 2015 AHA guidelines), earlier administration of epinephrine was associated with improved survival, with a 4 percent decreased likelihood of survival for each

additional minute of delay in epinephrine administration [63]. Improvements in one-year survival following in-hospital cardiac arrest have also been reported with early administration of epinephrine [21]. (See 'VF or VT arrest and vasopressors' above.)

PEA and atropine — Atropine is not recommended in the 2020 guidelines for PEA because of its lack of therapeutic benefit [3,25]. The administration of atropine (1 mg IV/IO every three to five minutes) may be considered for PEA when the rate is slow, ie, absolute bradycardia with a rate <50 beats/minute or a relative bradycardia (rate less than expected relative to the underlying condition) [22,23]. Atropine's effect is on vagally-mediated bradycardia, which is rarely a cause of cardiac arrest, and treatment successes are reportable.

In a cohort study of 1029 patients with PEA published after the 2010 AHA guidelines, 30-day neurologic outcomes were no different in the groups treated with epinephrine and atropine versus epinephrine alone [64]. In addition, survival was significantly lower in the group treated with epinephrine and atropine (3.2 percent versus 7.1 percent in epinephrine only group; OR 0.43, 95% CI 0.19 to 0.91), suggesting that atropine may actually be harmful when used to treat PEA. Registry data on over 20,000 in-hospital cardiac arrests with a non-shockable rhythm, collected between 2006 and 2015, showed no difference in survival before and after atropine was removed from the treatment protocol in the 2010 AHA guidelines [65].

ASYSTOLE

Sudden cardiac arrest in which asystole is the initial rhythm is associated with an extremely poor prognosis (0 to 2 percent survival to hospital discharge). Asystole is usually a secondary event, resulting from prolonged ventricular fibrillation or pulseless electrical activity (PEA) with subsequent loss of all electrical activity. It may also occur as a result of prolonged hypoxia, acidosis, and death of myocardial tissue (waveform 1). (See "Prognosis and outcomes following sudden cardiac arrest in adults", section on 'Asystole'.)

True asystole should be confirmed by checking the ECG and defibrillator cable connections, making certain that the gain is turned up and the monitor is working. Confirmation of asystole in another lead is recommended. The 2015 AHA guidelines recommend that asystole be treated in the same manner as PEA, with excellent cardiopulmonary resuscitation (CPR), vasopressors, and the consideration of possible reversible causes (table 1) [3]. Atropine is no longer recommended as therapy in asystole. CPR, with an emphasis on excellent chest compressions, should be provided throughout the resuscitative effort, stopping only at two-minute intervals during which brief rhythm analysis and defibrillation is provided if the patient has reverted to a shockable rhythm [62]. There is no benefit to defibrillation in patients with asystole. (See

'Pulseless electrical activity' above and "Advanced cardiac life support (ACLS) in adults", section on 'Asystole and pulseless electrical activity'.)

Because randomized controlled trials and other data have not shown a survival benefit with the use of temporary pacing in asystole, its routine use is not recommended by the 2020 AHA guidelines [3,25,66-70].

Prolonged resuscitative efforts of patients in asystole are generally futile. Termination of resuscitation is discussed elsewhere. (See "Advanced cardiac life support (ACLS) in adults", section on 'Termination of resuscitative efforts'.)

Asystole and vasopressors

Epinephrine — Epinephrine (1 mg IV/IO push every three to five minutes) is recommended in patients with asystole, although its benefit has not been clearly demonstrated in prospective trials [3,35]. In a secondary analysis of a cardiac arrest registry, among a cohort of 32,101 patients with out-of-hospital cardiac arrest and a nonshockable initial rhythm (published after the 2015 AHA guidelines), earlier administration of epinephrine was associated with improved survival, with a 4 percent decreased likelihood of survival for each additional minute of delay in epinephrine administration [63]. Improvements in one-year survival following in-hospital cardiac arrest have also been reported with early administration of epinephrine [21]. In a separate registry study that included more than 41,000 patients (not all of whom were in asystole), delay to epinephrine administration was also associated reduced survival [71]. (See 'VF or VT arrest and vasopressors' above.)

Routine use of high-dose epinephrine is not supported by data and is not recommended [72,73]. In a randomized trial comparing repeated high doses and standard doses of epinephrine for out-of-hospital cardiac arrest, survival to hospital discharge was similar with high-dose and standard dose epinephrine [73]. Although high-dose epinephrine improved the rate of successful resuscitation in patients with asystole, it did not improve survival to hospital discharge.

Vasopressin — Vasopressin has been studied as a potential alternative to epinephrine. In one trial comparing vasopressin with epinephrine in 1186 patients with out-of-hospital arrest, the 528 patients with asystole who received vasopressin were significantly more likely to survive until hospital admission (29 versus 20 percent with epinephrine, odds ratio [OR] 0.6, 95% CI 0.4-0.9) and hospital discharge (4.7 versus 1.5 percent, OR 0.3, 95% CI 0.1-1.0) [41]. However, a subsequent meta-analysis, which included data from this and four smaller studies (1519 patients overall), found no significant benefit of vasopressin compared with epinephrine among patients with asystole, or any other initial cardiac rhythm [42].

Based upon these data, the 2020 AHA guidelines removed vasopressin as an option for the treatment of patients with asystole [3,25].

A more recent placebo-controlled randomized trial of 501 patients with in-hospital cardiac arrest showed that vasopressin given with methylprednisolone after epinephrine administration was associated with a higher rate of return of spontaneous circulation (42 versus 33 percent) but no difference in 30-day mortality or neurologic recovery [74]. These new data are unlikely to modify the 2020 guideline recommendation regarding vasopressin.

Asystole and atropine — Due to a paucity of data supporting the use of atropine for asystole, the 2020 AHA guidelines no longer recommend the routine use of atropine for the management of asystole [3,25]. In a cohort study of 6419 patients with asystole, return of spontaneous circulation was significantly greater in patients receiving epinephrine and atropine (33 versus 19 percent with epinephrine alone) [64]. However, overall survival and 30-day neurologic outcomes were no different in the two groups. Registry data on over 20,000 in-hospital cardiac arrests with a non-shockable rhythm, collected between 2006 and 2015, showed no difference in survival before and after atropine was removed from the treatment protocol in the 2010 AHA guidelines [65].

INEFFECTIVE THERAPIES

A number of therapies have been shown to be generally ineffective in patients who present with sudden cardiac arrest, and therefore are not part of routine management:

- Sodium bicarbonate, except in patients with hyperkalemia (calcium is first-line therapy) or tricyclic antidepressant overdose
- Fibrinolytic therapy
- Cardiac pacing for asystole or pulseless electrical activity (PEA)
- Magnesium sulfate, except in patients who have drug-induced QT prolongation and develop torsades de pointes (see 'Magnesium sulfate' above)
- Vasopressin for SCA of any cause
- Atropine for PEA

The relevant data regarding these therapies are presented separately. (See "Therapies of uncertain benefit in basic and advanced cardiac life support".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Basic and advanced cardiac life support in adults".)

SUMMARY AND RECOMMENDATIONS

- **Introduction** – In patients with sudden cardiac arrest (SCA), survival depends upon prompt resuscitative efforts, including excellent cardiopulmonary resuscitation (CPR) with chest compressions and, when indicated, defibrillation to reestablish organized electrical activity with a stable rhythm. Chest compressions should not be stopped until the defibrillator is fully charged and the patient is ready for defibrillation. (See 'Introduction' above.)

- **Advanced cardiac life support** – Excellent CPR should be provided throughout the resuscitative effort until return of spontaneous circulation or termination of resuscitative efforts. Brief (10 second) pauses in CPR should only occur at two-minute intervals for rhythm analysis and defibrillation, if indicated (algorithm 1). (See "Advanced cardiac life support (ACLS) in adults" and 'Introduction' above.)

- **Ventricular fibrillation and pulseless ventricular tachycardia** – The only definitively effective treatment of ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT) is defibrillation, as VF and VT rarely, if ever, terminate spontaneously or after delivery of an antiarrhythmic drug. The chances of successful defibrillation, and survival, are greatest when defibrillation is performed early after the onset of VF or VT. However, while awaiting the arrival and setup of a defibrillator, CPR should be performed. (See "Advanced cardiac life support (ACLS) in adults" and 'VF and pulseless VT' above and 'Defibrillation' above.)

- **Vasopressors** – In shockable rhythms, the priority remains early defibrillation and excellent CPR. If return of spontaneous circulation is not achieved after the second defibrillation, epinephrine 1 mg IV/IO should be administered every three to five minutes while excellent CPR and defibrillation are continued. (See "Advanced cardiac life support (ACLS) in adults" and 'VF or VT arrest and vasopressors' above.)

- **Antiarrhythmic drugs** – VF and VT often persist despite defibrillation, or recur promptly after successful defibrillation. In such cases, intravenous antiarrhythmic drug therapy should be used, with amiodarone or lidocaine being the preferred antiarrhythmic drugs.

Notably in the management of cardiac arrest, amiodarone did not provide any benefit when assessing the clinically relevant outcome of neurologically favorable survival as compared with lidocaine or placebo. (See "Advanced cardiac life support (ACLS) in adults" and 'Antiarrhythmic drugs' above.)

- Magnesium sulfate – In patients with recurrent VF or pVT thought to be due to torsades de pointes with a prolonged QT interval, the administration of intravenous magnesium sulfate should be considered. (See 'Magnesium sulfate' above.)

- **Pulseless electrical activity and asystole** – In contrast to SCA caused by VF or pVT, there is no role for defibrillation in the management of pulseless electrical activity (PEA) or asystole. While CPR is ongoing, epinephrine should be administered as soon as possible (and repeated every three to five minutes). However, before vascular access and epinephrine administration are considered, excellent CPR must be in progress, and epinephrine should only administered after early identification and management of other etiologies of non-shockable rhythms (table 1). (See "Advanced cardiac life support (ACLS) in adults" and 'Pulseless electrical activity' above and 'Asystole' above.)

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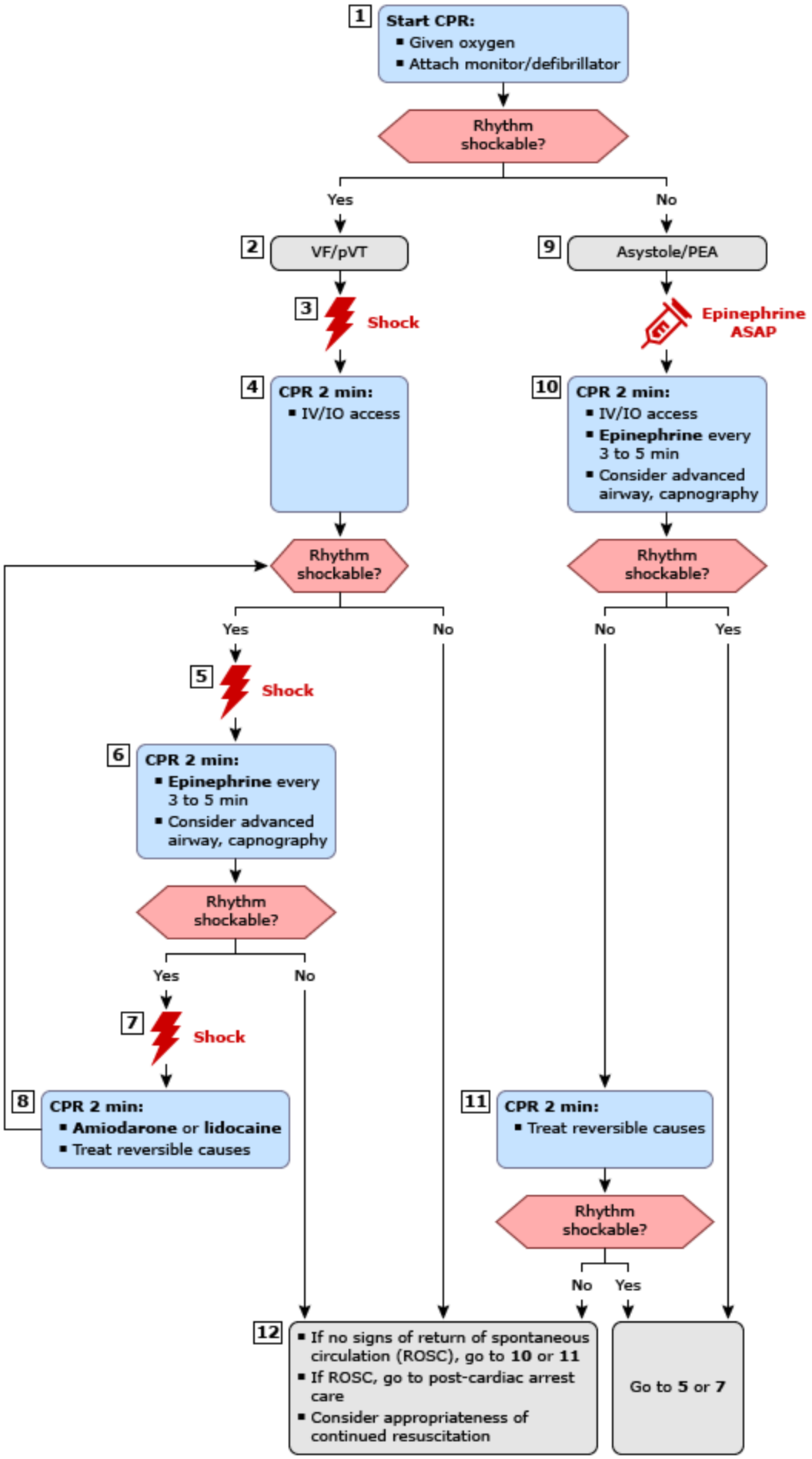
Topic 1028 Version 46.0

GRAPHICS

Adult cardiac arrest algorithm



CPR quali

- Push hard (at least 2 inch
fast (100 to 120/min) and
chest recoil.
- Minimize interruptions in
- Avoid excessive ventilatio
- Change compressor every
sooner if fatigued.
- If no advanced airway, 30
ventilation ratio.
- Quantitative waveform ca
  - If $PETCO_2$ is low or decr
CPR quality.

Shock energy for d

- **Biphasic:** Manufacturer r
(eg, initial dose of 120 to
unknown, use maximum
and subsequent doses sh
and higher doses may be
- **Monophasic:** 360 J.

Drug thera

- **Epinephrine IV/IO dos**
1 mg every 3 to 5 minute
- **Amiodarone IV/IO dos**
First dose: 300 mg bolus.
Second dose: 150 mg.
or
- **Lidocaine IV/IO dose:**
First dose: 1 to 1.5 mg/kg
Second dose: 0.5 to 0.75

Advanced ai

- Endotracheal intubation o
advanced airway.
- Waveform capnography o
confirm and monitor ET tu
- Once advanced airway in
breath every 6 seconds (1
with continuous chest com

Return of spontaneous c

- Pulse and blood pressure.
- Abrupt sustained increase
(typically ≥40 mmHg).
- Spontaneous arterial pres
intra-arterial monitoring.

Reversible ca

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

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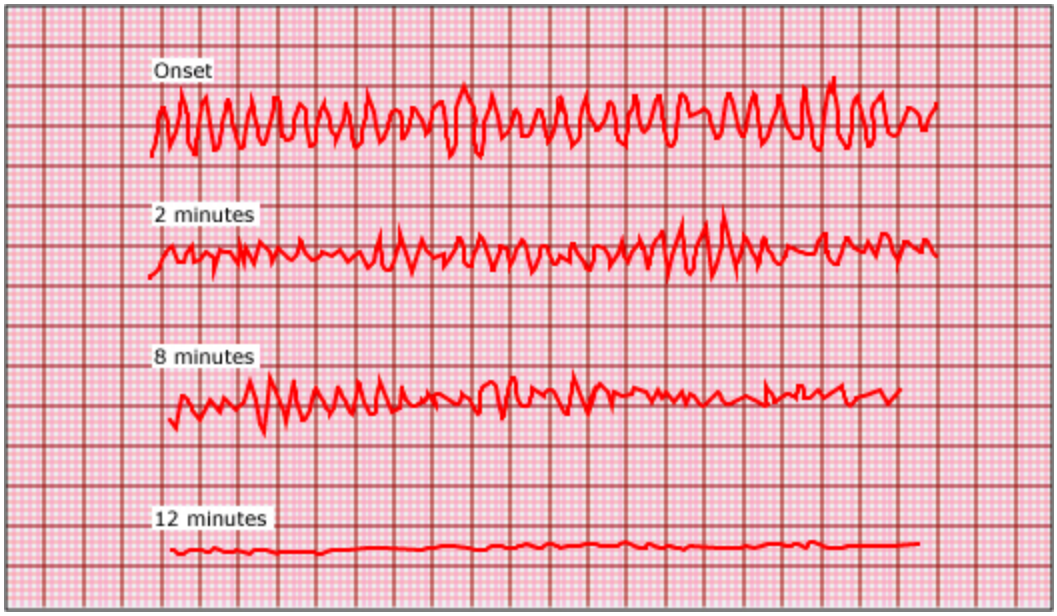
Graphic 129983 Version 10.0

Continuous electrocardigraphic (ECG) strip during an episode of ventricular fibrillation (VF) that progresses to fine VF and then asystole



At the onset of ventricular fibrillation (VF), the QRS complexes are regular, widened, and of tall amplitude, suggesting a more organized ventricular tachyarrhythmia. Over a brief period of time, the rhythm becomes more disorganized with high amplitude fibrillatory waves; this is coarse VF. After a longer period of time, the fibrillatory waves become fine, culminating in asystole.

Graphic 67777 Version 3.0

Defibrillation waveforms in implantable cardioverter-defibrillators

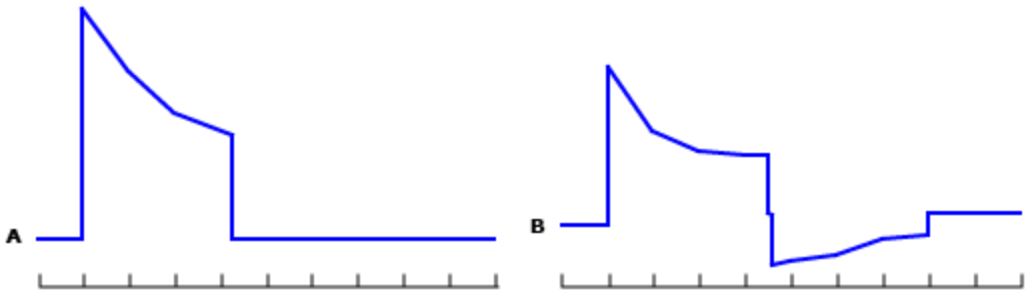


Figure A shows the monophasic, exponentially decaying pulse was the waveform used in the first generation of ICDs. Figure B shows the biphasic waveform, which is generated with a single capacitor by switching the output polarity during discharge. Each division is 2 milliseconds.

ICD: implantable cardioverter-defibrillator.

Graphic 51842 Version 3.0

Treatable conditions associated with cardiac arrest

Condition	Common associated clinical settings
Acidosis	Diabetes, diarrhea, drug overdose, renal dysfunction, sepsis, shock
Anemia	Gastrointestinal bleeding, nutritional deficiencies, recent trauma
Cardiac tamponade	Post-cardiac surgery, malignancy, post-myocardial infarction, pericarditis, trauma
Hyperkalemia	Drug overdose, renal dysfunction, hemolysis, excessive potassium intake, rhabdomyolysis, major soft tissue injury, tumor lysis syndrome
Hypokalemia*	Alcohol abuse, diabetes mellitus, diuretics, drug overdose, profound gastrointestinal losses
Hypothermia	Alcohol intoxication, significant burns, drowning, drug overdose, elder patient, endocrine disease, environmental exposure, spinal cord disease, trauma
Hypovolemia	Significant burns, diabetes, gastrointestinal losses, hemorrhage, malignancy, sepsis, trauma
Hypoxia	Upper airway obstruction, hypoventilation (CNS dysfunction, neuromuscular disease) pulmonary disease
Myocardial infarction	Cardiac arrest
Poisoning	History of alcohol or drug abuse, altered mental status, classic toxidrome (eg, sympathomimetic), occupational exposure, psychiatric disease
Pulmonary embolism	Immobilized patient, recent surgical procedure (eg, orthopedic), peripartum, risk factors for thromboembolic disease, recent trauma, presentation consistent with acute pulmonary embolism
Tension pneumothorax	Central venous catheter, mechanical ventilation, pulmonary disease (eg, asthma, chronic obstructive pulmonary disease), thoracentesis, thoracic trauma

CNS: central nervous system.

* Hypomagnesemia should be assumed in the setting of hypokalemia, and both should be treated.

Adapted from: Eisenberg MS, Mengert TJ. Cardiac resuscitation. N Engl J Med 2001; 344:1304.

Graphic 52416 Version 8.0