American Heart Association.

# HIGHLIGHTS

## of the **2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning**: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

**The American Heart Association thanks the following people for their contributions to the development of this publication:** Eric J. Lavonas, MD, MS; Amber V. Hoover, RN, MSN; Ian R. Drennan, ACP, PhD; and the AHA Resuscitation From Critical Poisoning Guidelines Writing Group.

These Highlights summarize the key recommendations addressed in the "2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning,"[1] which revises the *American Heart Association* (AHA) *Guidelines for Cardiopulmonary Resuscitation* (CPR) *and Emergency Cardiovascular Care*. These guidelines have been developed for resuscitation providers and for AHA instructors. They are based on evidence reviews and provide the rationale for the recommendations.

## Process Overview for Developing Guidelines Focused Updates

In developing these guidelines, the writing group produced clinical questions in the population, intervention, comparison, outcome format; performed structured literature reviews; synthesized the evidence; and developed treatment recommendations by using standardized methodology. Recommendations about opioid poisoning were updated from the *2020 AHA Guidelines for CPR and Emergency Cardiovascular Care*,[2,3] while the other recommendations were developed *de novo*. Each recommendation was assigned a Class of Recommendation and Level of Evidence using standard AHA definitions (Table 1). Conflicts of interest of the writing group members were disclosed and managed by using AHA processes. The completed guidelines were reviewed by the Emergency Cardiovascular Care Science Subcommittee, AHA Science Advisory and Coordinating Committee, the AHA Executive Committee, and peer reviewers nominated by the American Academy of Pediatrics, the American College of Medical Toxicology, the American Academy of Clinical Toxicology, America's Poison Centers, and the editors of *Circulation*.

**Table 1. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care (Updated May 2019)***

| CLASS (STRENGTH) OF RECOMMENDATION | |
|---|---|
| **CLASS 1 (STRONG)** | **Benefit >>> Risk** |
| **Suggested phrases for writing recommendations:**<br>• Is recommended<br>• Is indicated/useful/effective/beneficial<br>• Should be performed/administered/other<br>• Comparative-Effectiveness Phrases†:<br>– Treatment/strategy A is recommended/indicated in preference to treatment B<br>– Treatment A should be chosen over treatment B | |
| **CLASS 2a (MODERATE)** | **Benefit >> Risk** |
| **Suggested phrases for writing recommendations:**<br>• Is reasonable<br>• Can be useful/effective/beneficial<br>• Comparative-Effectiveness Phrases†:<br>– Treatment/strategy A is probably recommended/indicated in preference to treatment B<br>– It is reasonable to choose treatment A over treatment B | |
| **CLASS 2b (WEAK)** | **Benefit ≥ Risk** |
| **Suggested phrases for writing recommendations:**<br>• May/might be reasonable<br>• May/might be considered<br>• Usefulness/effectiveness is unknown/unclear/uncertain or not well-established | |
| **CLASS 3: No Benefit (MODERATE)**<br>**(Generally, LOE A or B use only)** | **Benefit = Risk** |
| **Suggested phrases for writing recommendations:**<br>• Is not recommended<br>• Is not indicated/useful/effective/beneficial<br>• Should not be performed/administered/other | |
| **CLASS 3: Harm (STRONG)** | **Risk > Benefit** |
| **Suggested phrases for writing recommendations:**<br>• Potentially harmful<br>• Causes harm<br>• Associated with excess morbidity/mortality<br>• Should not be performed/administered/other | |

| LEVEL (QUALITY) OF EVIDENCE‡ | |
|---|---|
| **LEVEL A** | |
| • High-quality evidence‡ from more than 1 RCT<br>• Meta-analyses of high-quality RCTs<br>• One or more RCTs corroborated by high-quality registry studies | |
| **LEVEL B-R** | **(Randomized)** |
| • Moderate-quality evidence‡ from 1 or more RCTs<br>• Meta-analyses of moderate-quality RCTs | |
| **LEVEL B-NR** | **(Nonrandomized)** |
| • Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies<br>• Meta-analyses of such studies | |
| **LEVEL C-LD** | **(Limited Data)** |
| • Randomized or nonrandomized observational or registry studies with limitations of design or execution<br>• Meta-analyses of such studies<br>• Physiological or mechanistic studies in human subjects | |
| **LEVEL C-EO** | **(Expert Opinion)** |
| • Consensus of expert opinion based on clinical experience | |

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR 1 and 2a; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely-used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

## Highlights of the "2023 Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning"

In the 12-month period ending in April 2021, more than 100 000 people in the United States died of poisoning and drug overdose, an increase of 28.5% from the prior year. Ninety percent of these deaths were unintentional. Although the majority of these deaths (75 673) were attributed to opioid overdose, poisoning from other toxins continues to claim a significant number of lives.

In addition to standard basic and advanced life support, treatment of cardiac arrest and life-threatening toxicity due to poisoning often requires specialized treatments that most clinicians do not use frequently. Expert consultation with a medical toxicologist, clinical toxicologist, or regional poison center facilitates rapid and effective therapy. The 2023 guidelines focused update[1] centered on the treatment of 12 critical poisoning scenarios and on the role of venoarterial extracorporeal membrane oxygenation (VA-ECMO) in the treatment of poisoning. In almost all cases, the available evidence is more directly relevant to the management of prearrest states (eg, hypotension and arrhythmias that are refractory to standard care) than to cardiac arrest itself. Table 2 provides a list of selected antidotes used in resuscitation from critical poisoning, along with dosing regimens commonly used in the literature.

**Table 2. Commonly Used Doses of Antidotes for Resuscitation in Critical Poisoning**

| Antidote | Indication | Initial dose (adult)* | Initial dose (pediatric)* | Maintenance infusion | Notes |
|---|---|---|---|---|---|
| Atropine | β-Blockers<br>CCBs<br>Digoxin<br>Local anesthetics | 0.5-1.0 mg every 3-5 min up to 3 mg | 0.02 mg/kg | None | |
| Atropine | Organophosphates<br>Carbamates | 1-2 mg, doubled every 5 min | 0.02 mg/kg, doubled every 5 min | 10%-20% of the total loading dose per hour up to 2 mg/h (adults) | Titrate to reversal of bronchorrhea, bronchospasm, bradycardia, and hypotension. |
| Calcium chloride | CCBs | 2000 mg<br>28 mEq $Ca^{2+}$<br>20 mL 100 mg/mL solution | 20 mg/kg<br>0.28 mEq $Ca^{2+}$/kg<br>0.2 mL/kg 100 mg/mL solution | 20-40 mg·kg$^{-1}$·h$^{-1}$<br>0.28-0.56 mEq $Ca^{2+}$kg$^{-1}$·h$^{-1}$<br>0.2-0.4 mL·kg$^{-1}$·h$^{-1}$<br>100 mg/mL solution | Titrate to blood pressure.<br>Do not exceed serum ionized calcium concentration 1.5-2 times the upper limits of normal.<br>Administer through central line, especially in children. |
| Calcium gluconate | CCBs | 6000 mg<br>28 mEq $Ca^{2+}$<br>60 mL 100 mg/mL solution | 60 mg/kg<br>0.28 mEq/kg $Ca^{2+}$<br>0.6 mL/kg 100 mg/mL solution | 60-120 mg·kg$^{-1}$·h$^{-1}$<br>0.28-0.56 mEq $Ca^{2+}$·kg$^{-1}$·h$^{-1}$<br>0.6-1.2 mL·kg$^{-1}$·h$^{-1}$<br>100 mg/mL solution | Titrate to blood pressure.<br>Do not exceed serum ionized calcium concentration 1.5-2 times the upper limits of normal. |

*(continued)*

| Antidote | Indication | Initial dose (adult)* | Initial dose (pediatric)* | Maintenance infusion | Notes |
|---|---|---|---|---|---|
| Digoxin immune Fab | Digoxin | Acute overdose: 1 vial for every 0.5 mg digoxin ingested<br>Chronic poisoning: Use formula: dose in vials=serum digoxin concentration (ng/mL)×weight (kg)/100<br>Acute overdose, critically ill, ingested dose unknown: 10-20 vials | Same as adult | None | 1 vial contains 40 mg Fab.<br>Lower doses may be equally effective.[4] |
| Digoxin immune Fab | Yellow oleander<br>*Bufo* toad venom | 1200 mg (30 vials) | Unknown | None | |
| Flumazenil | Benzodiazepines | 0.2 mg, titrated up to 1 mg | 0.01 mg/kg | None | Many contraindications |
| Glucagon | β-Blockers<br>CCBs | 2-10 mg | 0.05-0.15 mg/kg | 1-15 mg/h (adult) | Anticipate vomiting. |
| Hydroxocobalamin | Cyanide | 5 g | 70 mg/kg | None | |
| Insulin | β-Blockers<br>CCBs | 1 unit/kg | Same as adult | 1-10 units·kg$^{-1}$·h$^{-1}$ | Regular human insulin. Monitor for hypoglycemia, hypokalemia, volume overload. |
| ILE | Local anesthetics | 1.5 mL/kg up to 100 mL | Same as adult | 0.25 mL·kg$^{-1}$·min$^{-1}$ for up to 30 min | All studies use 20% lipid emulsion. |
| Methylene blue | CCBs<br>Methemoglobinemia | 1-2 mg/kg, repeated every hour if needed | Same as adult | 1 mg·kg$^{-1}$·h$^{-1}$ (for vasodilatory shock) | Maximum 5-7 mg/kg |
| Naloxone | Opioids | 0.2-2 mg IV/IO/IM<br>2-4 mg intranasal<br>Repeat every 2-3 min as needed | 0.1 mg/kg | Two thirds of the waking dose per hour | Titrate to reversal of respiratory depression and restoration of protective airway reflexes. |
| Pralidoxime | Organophosphates | 1-2 g | 20-50 mg/kg | 400-600 mg/h (adult)<br>10-20 mg·kg$^{-1}$·h$^{-1}$ (pediatric) | |
| Sodium bicarbonate† | Sodium channel blockers<br>Cocaine | 50-150 mEq | 1-3 mEq/kg | Prepare 150 mEq/L solution, infuse at 1-3 mL·kg$^{-1}$·h$^{-1}$ | Monitor for hypernatremia, alkalemia, hypokalemia, hypochloremia. |
| Sodium nitrite | Cyanide | 300 mg | 6 mg/kg | None | Monitor for hypotension. |
| Sodium thiosulfate | Cyanide | 12.5 g | 250 mg/kg | None | |

Abbreviations: β-Blocker, β-adrenergic receptor antagonist; CCB, calcium channel blocker; Fab, fragment antigen binding; ILE, intravenous lipid emulsion; IM, intramuscular; IO, intraosseous; and IV, intravenous.

*Unless otherwise stated, the route of administration should be intravenous or intraosseous. Maximum pediatric dose should not exceed adult dose. Most antidotes should be repeated frequently and titrated to achieve control of critical signs and symptoms. The ideal dose of most antidotes is not known and is often controversial. Large doses are sometimes required to overcome competitive inhibition of molecular targets such as adrenergic receptors and ion channels. Consult a medical or clinical toxicologist, regional poison center, or topic-specific reference for detailed dosing and administration instructions.

†Different sodium bicarbonate solutions are typically used for adults (1 mEq/mL) and children (0.5 mEq/mL). Both formulations are hypertonic.

## Benzodiazepines

Benzodiazepines are commonly used sedative-hypnotics used to treat anxiety, insomnia, seizures, and withdrawal syndromes and as a component of general anesthesia and procedural sedation. Benzodiazepine overdose causes central nervous system and respiratory depression. These effects are more pronounced when benzodiazepines are used with other sedatives, such as opioids or alcohol.

Although flumazenil can be effective in select patients with respiratory depression caused by pure benzodiazepine poisoning (eg, patients who develop respiratory depression during procedural sedation, in whom past medical and medication use histories are known), flumazenil administration may cause harm in patients who are at increased risk for seizures or dysrhythmias, such as people who have seizure disorders, are dependent on benzodiazepines, or who have coingested other medications (such as tricyclic antidepressants). For this reason, the AHA does not recommend flumazenil administration in patients with undifferentiated drug overdose. Because patients receiving CPR with mechanical ventilation are already being treated for apnea, flumazenil is not recommended for the treatment of cardiac arrest from benzodiazepine poisoning. Combined poisoning with opioids and benzodiazepines is common; in this scenario, naloxone, which has a better safety profile, should be given before flumazenil administration is considered.

## β-Blockers and Calcium Channel Blockers

β-Adrenergic receptor antagonists (commonly called *β-blockers*) and antagonists of the L-type calcium channel (commonly called *calcium channel blockers*) cause hypotension and bradycardia, which can be refractory to treatment with vasopressors and other critical care modalities. High-dose insulin therapy (eg, 1 unit/kg intravenous [IV] bolus, followed by an infusion of 1 to 10 units/kg per hour, with treatment to avoid hypoglycemia, severe hypokalemia, or severe volume overload) can be lifesaving for patients who fail to respond to vasopressors alone. Using glucagon (for β-blocker overdose) and calcium (for calcium channel blocker overdose) is supported by limited data, and is reasonable to use. Patients with severe bradycardia may benefit from atropine administration or pacing. Dialysis may be used to remove atenolol or sotalol, but it is not thought to be effective for other β-blocker and calcium channel blocker poisoning. For patients with life-threatening shock refractory to pharmacologic interventions, it is reasonable to use VA-ECMO to support the patient until the poison can be eliminated.

## Cocaine

Cocaine poisoning causes central nervous system stimulation, agitation, tachycardia, hypertension, hyperthermia, diaphoresis, and coronary vasospasm. Cocaine acts on cardiac ion channels like a Vaughan-Williams Ia or Ic medication, causing QRS and QT interval prolongation and wide-complex tachycardia, which may progress to cardiac arrest. Benzodiazepines remain the mainstay of initial management of blood pressure and psychomotor agitation for patients with acute cocaine poisoning. Vasodilators, such as nitrates, phentolamine, or calcium channel blockers, may be used for patients with cocaine-induced coronary vasospasm or hypertensive emergencies. Sodium bicarbonate may be used to treat wide-complex tachycardia or cardiac arrest from cocaine poisoning; lidocaine, a Vaughan-Williams Ib drug, also can be used to treat wide-complex tachycardia. Patients with cocaine-induced hyperthermia should have rapid external cooling, such as with ice water immersion.

## Cyanide

Cyanide is commonly used in jewelry cleaning, electroplating, metallurgy, and other industrial and laboratory processes. It is also found in fire smoke and certain plant compounds. Rarely, cyanide poisoning is used in criminal poisoning or suicide attempts. In the body, cyanide blocks the electron transport chain, inhibiting aerobic respiration and adenosine triphosphate formation. Clinically, this causes hypotension, lactic acidemia, altered mental status, seizures, and death.

Hydroxocobalamin (vitamin $B12_a$) scavenges cyanide to form nontoxic cyanocobalamin. Alternatively, sodium nitrite oxidizes hemoglobin to form methemoglobin, which has a high affinity for cyanide. Sodium thiosulfate acts as a cofactor for cyanide metabolism, forming minimally toxic thiocyanate.

The initial treatment for patients with cyanide poisoning is hydroxocobalamin administration; if hydroxocobalamin is not available, then sodium nitrite is an alternative. Sodium thiosulfate, which is slower-acting than these other agents but may have synergistic benefit, is then administered to speed cyanide elimination.

## Digoxin

Digoxin and related cardiac glycosides are found in medications, plants such as foxglove and oleander, and certain toad venoms. Poisoning caused by overdosage, unintentional ingestion, drug-drug interaction, or drug accumulation due to reduced renal clearance may occur. Patients with cardiac glycoside poisoning may develop gastrointestinal symptoms, confusion, hyperkalemia, bradycardia, and cardiac conduction abnormalities including atrioventricular nodal block, ventricular tachycardia, ventricular fibrillation, and asystole.

Digoxin-specific immune antibody fragments (digoxin-Fab), which bind to and inactivate digoxin and structurally similar cardiac glycosides, should be administered to patients with life-threatening poisoning. Atropine and/or cardiac pacing may be necessary while waiting for the antidote to take effect, and limited data support the use of lidocaine, phenytoin, or bretylium for patients with ventricular dysrhythmias while awaiting digoxin-Fab effect. Recommended doses for digoxin-Fab vary widely worldwide; the strongest data exist for digoxin, digitoxin, *Bufo* toad venom, and yellow oleander poisoning.

## Local Anesthetics

Local anesthetic systemic toxicity occurs as a complication of regional (or, rarely, local) anesthesia, when enough local anesthetic enters the circulation to cause seizures, agitation, dysarthria, confusion, arrhythmias, or cardiovascular collapse. Bupivacaine is the best studied and most dangerous form of local anesthetic systemic toxicity.

Both hypoxemia and acidemia worsen bupivacaine toxicity, so ventilation and treatment of acidemia are critical. An IV infusion of lipid emulsion can effectively treat both neurotoxicity and cardiotoxicity from bupivacaine. Although the ideal dose and formulation are not known, most studies used a 20% solution of long-chain fatty acids, infused as an IV bolus of 1.5 mL/kg over 1 minute, followed by an infusion of 0.25 mL/kg per minute for up to 30 minutes. Benzodiazepines should be used to treat seizures, and sodium bicarbonate may be useful for patients with wide-complex tachycardia. Some patients with refractory cardiogenic shock require VA-ECMO.

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## Methemoglobinemia

Acquired methemoglobinemia occurs after exposure to an oxidant stressor that oxidizes iron in the hemoglobin molecule from the ferrous ($Fe^{2+}$) to the ferric ($Fe^{3+}$) state. In the ferric state, hemoglobin no longer effectively binds and delivers oxygen to the tissues. Common sources of oxidant stress that can cause methemoglobinemia include nitrates, nitrites, and many pharmaceuticals (eg, dapsone, benzocaine, phenazopyridine). Patients with methemoglobinemia can appear cyanotic and dusky and report shortness of breath and fatigue. Although moderate methemoglobinemia is generally well tolerated, severe methemoglobinemia can lead to cardiovascular collapse and death.

The most widely accepted treatment for methemoglobinemia is methylene blue, which acts as a cofactor to reduce methemoglobin to hemoglobin. In rare cases, exchange transfusion and/or hyperbaric oxygen therapy may be useful.

## Opioids

The epidemic of opioid poisoning continues to worsen in the United States and internationally, with more than 75 000 deaths reported in the United States in the year ending in April 2021. Most deaths are unintentional.

In this guidelines focused update,[1] the AHA reaffirms the recommendations in the *2020 AHA Guidelines for CPR and Emergency Cardiovascular Care*[2,3] and the observations in the AHA's 2021 scientific statement on opioid-associated out-of-hospital cardiac arrest.[5] Minor changes were made to evidence grading and supportive text, and new references were added.

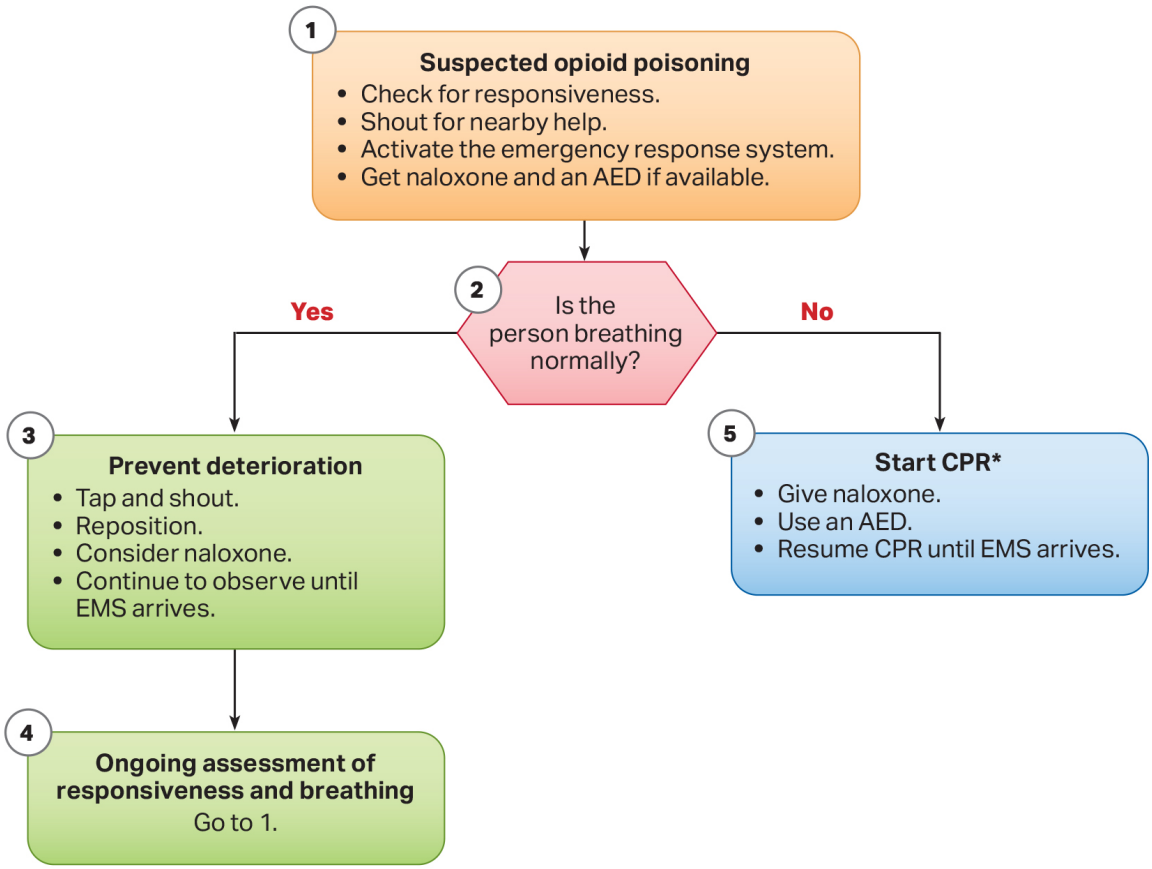
Widespread availability and use of naloxone is an important tool to reduce deaths from opioid overdose. Many studies have shown that lay rescuers who receive overdose education and naloxone training, ideally including skills practice, can effectively identify opioid overdose and administer naloxone.

It can be difficult in the hospital setting, and may be impossible in the out-of-hospital setting, to accurately differentiate opioid-associated resuscitative emergencies from other causes of cardiac and respiratory arrest. Therefore, the cornerstone of treatment is highly effective CPR, preferably with both chest compressions and ventilation. The emergency response system should be activated as soon as possible (Figures 1 and 2). For patients who definitely have a pulse (ie, respiratory arrest), rescue breathing or bag-mask ventilation should be performed until effective spontaneous breathing occurs. Naloxone administration, by either lay rescuers or health care professionals, may restore spontaneous respirations, protective airway reflexes, and normal mental status. People with opioid overdose who are breathing normally with intact airway reflexes can be carefully observed without naloxone, regardless of somnolence.

While naloxone administration has no known or theoretical benefit in cardiac arrest, if there is uncertainty whether the patient is in cardiac or only respiratory arrest, naloxone should be given.

Because the effect of opioids can be longer than the effect of naloxone, people who require naloxone should be observed in a health care setting until their level of consciousness and vital signs have normalized and the risk of recurrent opioid toxicity is low.

**Figure 1. Opioid-Associated Emergency for Lay Responders Algorithm.[2]**

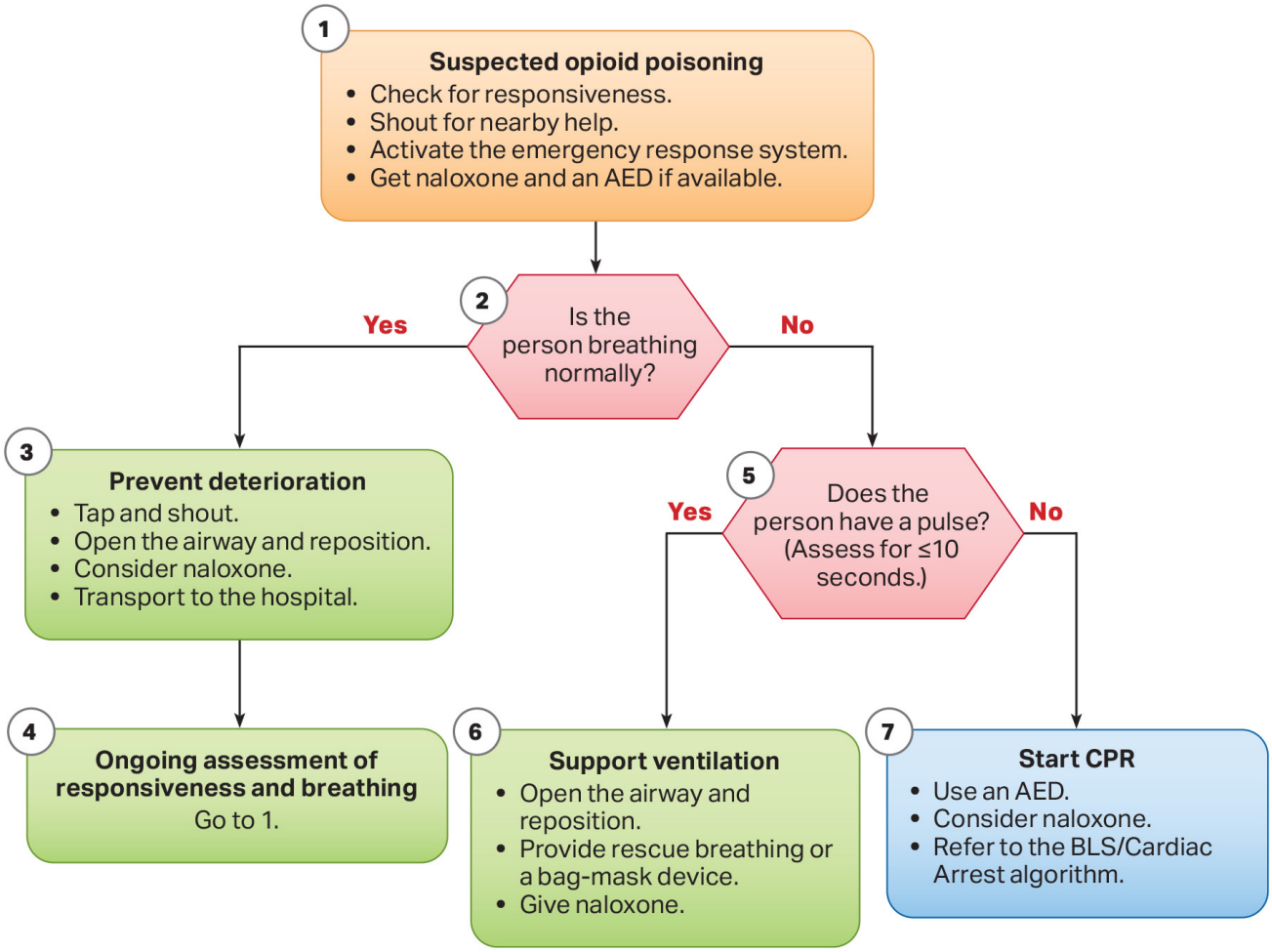



*For adult and adolescent victims, responders should perform compressions and rescue breaths for opioid-associated emergencies if they are trained and perform Hands-Only CPR if not trained to perform rescue breaths. For infants and children, CPR should include compressions with rescue breaths.



Abbreviations: AED, automated external defibrillator; EMS, emergency medical services.

**Figure 2. Opioid-Associated Emergency for Healthcare Providers Algorithm.[2]**






Abbreviations: AED, automated external defibrillator; BLS, basic life support.

## Organophosphates and Carbamates

Organophosphates and carbamates are found in pesticides, nerve agents, and some medications. These chemicals inhibit acetylcholinesterase, causing parasympathetic effects (bradycardia, bronchospasm, bronchorrhea, miosis, hypersalivation, lacrimation, urination, diarrhea, vomiting, diaphoresis), nicotinic effects (tachycardia, mydriasis, fasciculations progressing to paralysis), and central nervous system effects (altered mental status, apnea, seizures). Early and effective treatment may prevent deterioration to respiratory and cardiac arrest.

The cornerstones of treatment include decontamination, atropine, benzodiazepines, and oximes. Dermal decontamination through removal of contaminated clothing and copious irrigation with soap and water, performed by people wearing protective barriers, helps prevent further absorption of chemicals and protects people in the care environment. Atropine blocks parasympathetic overstimulation, treating bronchorrhea, bradycardia, bronchospasm, and central nervous system effects, but does not reverse paralysis. Early endotracheal intubation may be necessary. Benzodiazepines are used to prevent and treat seizures. When administered early in people with organophosphate poisoning, oximes (such as pralidoxime) reactivate the acetylcholinesterase enzyme, reversing nicotinic effects to slowly improve respiratory and skeletal muscle strength.

## Sodium Channel Blockers

Many poisons, such as tricyclic antidepressants, block cardiac sodium channels with properties similar to Vaughan-Williams class Ia or Ic antidysrhythmics. Sodium channel blocker poisoning causes QRS prolongation, hypotension, seizures, ventricular dysrhythmias, and cardiovascular collapse. The mainstay of treatment is IV administration of sodium bicarbonate. Vaughan-Williams class Ib medications (eg, lidocaine) may also be beneficial. IV lipid emulsion may be used to treat life-threatening sodium channel blocker poisoning that fails to respond to other therapies.

## Sympathomimetics

The hallmark of sympathomimetic poisoning is increased activity of the adrenergic nervous system. Amphetamines, cathinones, and some synthetic cannabinoid receptor agonists produce sympathomimetic poisoning. Clinicians are rarely able to determine which specific substance was used, and treatment must be based on presenting signs and symptoms and limited available history. Sympathomimetic poisoning may cause tachycardia, hypertension, agitation, seizures, hyperthermia, rhabdomyolysis, metabolic acidosis, myocardial infarction, stress (takotsubo) cardiomyopathy, and cardiac arrest. Immediate treatment for life-threatening sympathomimetic

poisoning is sedation to treat agitation. Although temporary application of physical restraints is often required, the prolonged use of physical restraints without sedation is potentially harmful.

Patients with life-threatening hyperthermia should receive rapid external cooling; immersion and evaporative cooling are more effective than cooling blankets, the application of cold packs, or endovascular cooling devices are. Vasodilators (eg, phentolamine and/or nitrites) can be used to treat coronary vasospasm that persists after sedation. Patients with refractory cardiogenic shock may require mechanical circulatory support, such as intra-aortic balloon pump or VA-ECMO.

### Venoarterial Extracorporeal Membrane Oxygenation

VA-ECMO is a resuscitative measure providing both cardiac and pulmonary support. In the setting of poisoning, VA-ECMO treats refractory cardiogenic shock by providing mechanical circulatory support while the offending poison is eliminated. Poisoned patients may make ideal candidates for temporary VA-ECMO support, because, in the absence of permanent end-organ damage, the natural course of drug overdose is recovery due to renal, hepatic, or extracorporeal clearance of the toxin.

The use of VA-ECMO in the poisoned patient is limited by availability, logistics of transport, patient comorbidities, and the significant risks inherent to the procedure. The pathophysiology of the specific poisoning and the clinical features of the patient must both be considered in the decision to initiate VA-ECMO. In particular, VA-ECMO does not generally correct distributive shock or reverse cellular injury. A multidisciplinary approach, including consultation from a poison center or medical toxicologist, is helpful to determine the appropriateness of VA-ECMO in specific cases.

## References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