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ECC Guidelines

Part 8: Advanced Challenges in Resuscitation

Section 2: Toxicology in ECC

▶ General Considerations

Poisoning is the third leading cause (11%) of injury-related mortality in the United States and is among the top 3 causes in most other countries. In the United States in 1995 there were an estimated 2.4 million exposures to poison and 1 million visits to the Emergency Department (1% of all visits), 215 000 admissions to the hospital, and 18 549 deaths due to poisoning.^{1 2 3} Although exposure to poison is common, life-threatening or fatal poisoning is not. Poisoning is a relatively infrequent cause of cardiac arrest overall but is a leading cause in victims <40 years old. The long-term survival rate among victims of cardiac arrest due to poisoning is good, averaging 24% in 6 studies.

The relatively small number of life-threatening poisonings and the lack of a prehospital triage protocol for severe poisonings in the United States are major obstacles to the performance of high-quality clinical research. Because the research in this area consists primarily of small case series (level of evidence [LOE] 5), animal studies (LOE 6), and case reports, the American Heart Association (AHA) class of recommendation for most recommendations for treating victims of poisoning is IIb. The following evidence- and consensus-based guidelines were developed by a toxicology work group of the AHA Advanced Cardiovascular Life Support (ACLS) Committee to provide guidance in the management of severe poisoning when standard ECC guidelines may not be optimal or appropriate. In addition to these guidelines we recommend consultation with a medical toxicologist or certified regional poison information center and use of a poison treatment center for unusual cases of poisoning.^{4 5} See Tables 1⁺ and 2⁺.

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View this table: **Table 1.** Sympathomimetic and Cardiotoxic Drugs
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▶ **Drug-Induced Emergencies: Prearrest**

Airway and Respiratory Management

Because poisoned patients can deteriorate rapidly, frequently assess their ability to protect their airway and breathe adequately. International guidelines recommend gastric lavage only for patients who have ingested a potentially lethal amount of drug or toxin and present within 1 hour of ingestion.⁶ In obtunded or comatose patients, perform rapid-sequence intubation before gastric lavage to prevent aspiration pneumonia. Because reversal of benzodiazepine intoxication with flumazenil is hazardous, we do not recommend routine inclusion of this practice in "coma cocktail" protocols.

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Opiate Poisoning

If a patient suspected of overdosing on an opiate has a pulse, try to reverse respiratory insufficiency with naloxone, an opiate antagonist, before inserting an endotracheal tube. Do not withhold naloxone until artificial ventilation is initiated. Heroin is the opiate taken in most cases of opiate overdose treated in emergency settings. Severe complications after opiate reversal are uncommon (<2%). Although the effects of naloxone do not last as long as those of heroin (45 to 70 minutes compared with 4 to 5 hours), naloxone is the preferred agent for reversal. Some EMS systems allow selected patients aroused with naloxone to refuse transport to the hospital against medical advice. Allowing selected patients to refuse observation against medical advice rarely leads to serious consequences such as severe re-narcotization or delayed pulmonary edema.^{7 8} Naloxone can be administered intramuscularly, subcutaneously, or intravenously. The IM and SC routes theoretically provide greater ease of administration, less risk of needle puncture, and less risk of severe withdrawal in patients addicted to opiates than the IV route.

The desired end points of opiate reversal are adequate airway reflexes and ventilations, not complete arousal. Acute, abrupt withdrawal from opiates may increase the frequency of severe complications such as pulmonary edema, ventricular arrhythmia, and severe agitation. In 2 studies of opiate reversal with naloxone, only small doses were required for opiate reversal. In emergency settings the recommended initial dose of naloxone is 0.4 to 0.8 mg IV or 0.8 mg IM or

SC. In communities in which abuse of naloxone-resistant opiates is prevalent, larger initial doses of naloxone may be needed. When opiate overdose is strongly suspected or in areas where abuse of "China white" is prevalent, titration to a total naloxone dose of 6 to 10 mg is recommended if needed.

Drug-Induced Hemodynamically Significant Bradycardia

In cases of drug-induced hemodynamically significant bradycardia (HSB), atropine is seldom helpful but is acceptable to administer because it is not harmful. The major exception is acute organophosphate or carbamate poisoning, in which case atropine may be lifesaving. The recommended starting dose of atropine for adults with insecticide poisoning is 2 to 4 mg. Avoid use of isoproterenol, which may induce or aggravate hypotension and ventricular arrhythmias.

In cases of massive β -blocker poisoning, however, isoproterenol given in very high doses is reportedly effective. Digoxin-specific Fab antibody fragments are extremely effective therapy for life-threatening ventricular arrhythmias or heart block due to poisoning with digoxin or cardiac glycosides.⁹ Electrical cardiac pacing is often effective in cases of mild to moderate drug-induced HSB. If external pacing is poorly tolerated or electrical capture is difficult to maintain, use transvenous pacing. When transcutaneous pacing is used, prophylactic transvenous placement of the pacer wire is not recommended because the tip of the catheter may trigger ventricular arrhythmias when the myocardium is irritable. In cases of very severe poisoning, capture may not occur despite proper location of the wire and use of the highest voltage settings. If HSB is resistant to atropine and pacing, use vasopressors with greater β -agonist activity. Management of more resistant drug-induced HSB is discussed in Drug-Induced Shock.

Drug-Induced Hemodynamically Significant Tachycardia

Drug-induced hemodynamically significant tachycardia (HST) may induce myocardial ischemia, myocardial infarction, or ventricular arrhythmias and lead to high-output heart failure and shock. Avoid use of routine measures such as adenosine therapy and synchronized cardioversion in patients with drug-induced HST because the tachycardia is likely to recur or to be refractory. In patients with borderline hypotension, diltiazem and verapamil are relatively contraindicated because they may precipitate more severe shock. Pharmacological measures are preferred when rate control is necessary.

Benzodiazepines such as diazepam or lorazepam are generally safe and effective in patients with drug-induced HST. Avoid using benzodiazepines in amounts that depress the level of consciousness and create the need for respiratory assistance. Physostigmine is a specific antidote that may be preferable for drug-induced HST and central anticholinergic syndrome due to *pure* anticholinergic poisoning. Very cautious use of a nonselective β -blocker such as propranolol may be effective in patients with drug-induced HST due to sympathomimetic poisoning.

Drug-Induced Hypertensive Emergencies

A drug-induced hypertensive emergency is often short-lived, and aggressive therapy is not needed. This is an important caution because hypotension may occur later in cases of severe

stimulant poisoning. Benzodiazepines are first-line therapy. In patients with a drug-induced hypertensive emergency refractory to benzodiazepines, use short-acting antihypertensive agents, such as nitroprusside, as second-line therapy. Labetalol (a nonselective β -blocker, α -blocker, and β_2 -agonist) in carefully titrated doses is a third-line agent, effective at times for drug-induced hypertensive emergencies associated with sympathomimetic poisoning. Propranolol (a nonselective β -blocker) is contraindicated because it may block the β_2 -receptors, leaving α -adrenergic stimulation unopposed and worsening hypertension.¹⁰

Drug-Induced Acute Coronary Syndromes

Treatment of drug-induced acute coronary syndromes is similar to the treatment recommended for drug-induced hypertensive emergencies. Catheterization studies have shown that nitroglycerin and phentolamine (an α -blocker) reverse cocaine-induced vasoconstriction, that labetalol has no significant effect, and that propranolol worsens it.^{11 12 13 14} Therefore, benzodiazepines and nitroglycerin are first-line agents, phentolamine is a second-line agent, and propranolol is contraindicated. Although labetalol has been reported to be effective in isolated cases, use of this agent is controversial because it is a nonselective β -blocker.^{15 16} Esmolol and metoprolol are selective β -blockers (β_1 but not β_2) that will not aggravate hypertension, but these agents can induce hypotension.¹⁷ Because esmolol has a very short half-life, the adverse effects of this agent should disappear a few minutes after the infusion is stopped.

Intracoronary administration of thrombolytics or coronary vasodilators is preferred to blind peripheral administration in cases of a drug-induced acute coronary syndrome resistant to the treatments described here. Thrombolytics are contraindicated if an uncontrolled, severe drug-induced hypertensive emergency is present.

Drug-Induced VT and VF

Drug-induced ventricular tachycardia (VT) may be difficult to distinguish from drug-induced impaired conduction (wide complex). When sudden conversion to a wider-complex rhythm occurs with hypotension, drug-induced VT is likely and cardioversion is indicated. Use of antiarrhythmics is indicated in cases of hemodynamically stable drug-induced VT, but there is scant evidence to guide the choice of agent. Procainamide is contraindicated in cases of poisoning with tricyclic antidepressants (TCAs) or poisonings with other drugs that have similar antiarrhythmic properties. In theory lidocaine should be contraindicated in cases of cocaine poisoning. The current consensus, however, based on extensive clinical experience, is that lidocaine is safe and effective.¹⁸

In the past phenytoin was recommended for TCA-induced VT, but more recently the efficacy and safety of this agent have been questioned.^{19 20} There is no acceptable published data on the use of bretylium tosylate for drug-induced VT or VF. Although magnesium has beneficial effects in certain cases of drug-induced VT, it may also aggravate drug-induced hypotension.^{21 22} In most cases of drug-induced monomorphic VT or VF, lidocaine is the antiarrhythmic of choice.

Torsades de pointes can occur with exposure to many drugs, either therapeutic or toxic. Correctable factors that increase the risk of torsades de pointes include hypoxemia, hypokalemia, and hypomagnesemia. Treatment of drug-induced torsades de pointes includes correction of risk factors and electrical and pharmacological therapy:

- Magnesium supplementation is recommended for patients with torsades de pointes even if the serum concentration is normal.
- Lidocaine has produced mixed results in studies of torsades de pointes and is Class Indeterminate.
- Electrical overdrive pacing at rates of 100 to 120 beats per minute will usually terminate torsades de pointes.
- Pharmacological overdrive pacing with isoproterenol has also been recommended.
- Some toxicologists recommend potassium supplementation even if the serum concentration is normal.

The safety and efficacy of these recommended therapies for drug-induced polymorphic VT has not been established by higher levels of research. From the perspective of evidence-based guidelines, we have only lower-level publications of case reports, case series, and extrapolated data. These recommendations, therefore, are Class Indeterminate. This class neither prohibits nor encourages clinical use. It merely acknowledges that many toxicology approaches are "best guesses."

Drug-Induced Impaired Conduction

Poisoning with membrane-stabilizing agents prolongs ventricular conduction (increases QRS interval). This predisposes the heart to monomorphic VT. Hypertonic saline and systemic alkalization often reverse the adverse electrophysiological effects. This prevents or terminates VT secondary to poisoning from many types of sodium channel blocking agents.²³ Hypertonic sodium bicarbonate is particularly valuable because it both provides hypertonic saline and induces systemic alkalization. This appears to benefit several types of poisonings caused by sodium channel blockers (eg, TCA). When hypertonic sodium bicarbonate is used to treat severe poisoning, the goal is an arterial pH of 7.50 to 7.55. Respiratory alkalosis can be used as a temporary measure until the appropriate degree of metabolic alkalosis can be attained with sodium bicarbonate. Establish systemic alkalization to the target arterial pH with repetitive boluses of 1 to 2 mEq/kg sodium bicarbonate. Maintain the alkalization via a titrated infusion of an alkaline solution consisting of 3 ampules of sodium bicarbonate (150 mEq) and KCl (30 mEq) in 850 mL of D₅W.

Drug-Induced Shock

Drug-induced shock usually results when the drug induces decreases in intravascular volume, falls in systemic vascular resistance (SVR), diminished myocardial contractility, or a combination of these factors.

Drug-Induced Hypovolemic Shock

Initial treatment of drug-induced shock usually includes a fluid challenge to correct hypovolemia and to optimize preload. If the offending agent is cardiotoxic, it will reduce the patient's ability to tolerate a high intravascular volume and may lead to iatrogenic congestive heart failure. If shock persists after an adequate fluid challenge, start a vasopressor. Evidence supports dopamine as the most effective pressor agent in mild to moderate poisoning.²⁴ Most patients with drug-induced shock have decreased contractility and low SVR. Empirical treatment of dopamine-resistant shock with more potent vasopressors is based on the assumption that decreased SVR is present.

When drug-induced shock is unresponsive to volume loading and conventional doses of vasopressors, high-dose vasopressors are indicated. If possible, establish central hemodynamic monitoring using a Swan-Ganz catheter before starting high-dose vasopressors. But do not delay vasopressor therapy to have a central monitoring line in place. Optimize cardiac preload quickly. Then use cardiac output (CO) and SVR to guide vasopressor and inotrope selection.

Drug-Induced Distributive Shock

When CO is normal or high and SVR is low (distributive shock), more potent vasoconstriction (ie, a greater α -adrenergic effect as produced by norepinephrine or phenylephrine) is needed. Dobutamine and isoproterenol decrease SVR and are contraindicated. The dose of an α -adrenergic-selective vasopressor should be increased until the shock is adequately treated or adverse effects such as ventricular arrhythmias are observed. Some patients require doses of vasopressors far above the usual doses. Use of powerful vasoconstrictors such as vasopressin or endothelin in cases of severe poisoning has not been well studied but may be considered if ventricular arrhythmia develops before the shock is adequately treated.

Drug-Induced Cardiogenic Shock

In cases of drug-induced shock characterized by low CO and high SVR (cardiogenic shock) or low SVR (typical drug-induced shock), inotropic agents are often required. Agents to choose from include calcium, amrinone, glucagon, insulin, isoproterenol, and dobutamine. Sometimes more than one agent is necessary.^{25 26} Although these agents may increase contractility and CO, they may also decrease SVR. A concomitant vasopressor is often required.²⁷

Drug-Induced Cardiac Arrest **Cardioversion/Defibrillation**

Electrical cardioversion or defibrillation is appropriate for pulseless patients with drug-induced VT or VF. In cases of sympathomimetic poisoning with refractory VF, the cost-benefit ratio of epinephrine in management is unknown. If epinephrine is used in such cases, increase the interval between doses and use only standard dose amounts (1 mg IV). Avoid high-dose epinephrine. Furthermore, propranolol is contraindicated in sympathomimetic poisoning, on the basis of limited human studies in the cardiac catheterization suite and limited animal survival studies.

Prolonged CPR and Resuscitation

In ACLS, cardiac resuscitation is usually terminated after 20 to 30 minutes unless there are signs that the central nervous system is viable. More prolonged CPR and resuscitation are warranted in poisoned patients. Cerebral blood flow drops dramatically with prolonged CPR in animal models

of cardiac arrest. Nevertheless, in cases of severe poisoning, recovery with good neurological outcomes has occasionally been reported in patients who received prolonged CPR, sometimes for up to 3 to 5 hours.^{28, 29} The marked vasodilatation associated with many types of severe poisonings may explain these observations.

Circulatory Assist Devices

Intra-aortic balloon pumps and cardiopulmonary bypass circuits are circulatory assist devices that have been used successfully in cases of critical poisoning. Because these techniques are expensive, personnel intensive, and associated with significant morbidity, they should be used only in cases refractory to *maximal* medical care. A disadvantage of the intra-aortic balloon pump is the need for an intrinsic cardiac rhythm for synchronization and diastolic augmentation. Emergency cardiopulmonary bypass does not require an intrinsic rhythm. Recent technological advances have made rapid application through peripheral vessels possible. To be effective, circulatory assist devices must be used rapidly (ie, before the irreversible effects of severe shock occur).

Brain Death and Organ Donation Criteria

Electroencephalographic and neurological criteria for brain death are not valid during acute toxic encephalopathy and can be applied only when drug concentrations are no longer toxic. In the presence of toxic drug concentrations the only valid criterion for brain death is the absence of cerebral blood flow. Successful transplantation of organs from victims of fatal poisoning with acetaminophen, cyanide, methanol, and carbon monoxide has been reported.³⁰ Transplantation of organs from victims poisoned with agents capable of severe end-organ damage (eg, carbon monoxide, cocaine, and iron) is controversial but may be appropriate if the donor is thoroughly evaluated.

▶ Summary

Use of standard ACLS protocols for all patients who are critically poisoned may not result in an optimal outcome. Care of severely poisoned patients can be enhanced by urgent consultation with a medical toxicologist. Alternative approaches required in severely poisoned patients include

- Higher doses than usual
- Drugs that are rarely used to treat cardiac arrest (amrinone, calcium, esmolol, glucagon, insulin, labetalol, phenylephrine, physostigmine, and sodium bicarbonate)
- Heroic measures, such as prolonged CPR and use of circulatory assist devices

When resuscitation is unsuccessful, organ donation may still be an option.

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