

specific problems in the poisoned patient

renal failure

- in the poisoned patient, acute renal failure (ARF) is most frequently the result of a decrease in extracellular fluid volume and renal hypoperfusion caused by drug- or chemical-induced vasodilation, drug-induced myocardial depression, or rhabdomyolysis.
- Attempts to prevent ARF are crucial, because there is no specific therapy once ARF has occurred.
- Studies evaluating the efficacy of low-dose dopamine (0.5 to 3.0 mg/kg/min) in preventing ARF have not demonstrated any benefit, but the efficacy of administration of low-dose dopamine after periods of hypotension in poisoned patients, who unlike trial patients are typically younger and without chronic disease, has not been evaluated.

seizures

- Blood pH may be as low as 7.17 at 30 minutes and 7.20 at 60 minutes after resolution of a 30- to 60-second seizure.
- Acidosis decreases cardiac output, oxygen extraction, and left ventricular end-diastolic pressure and impairs myocardial contractility. If a patient has ingested a cardiotoxic drug (e.g., a TCA) that causes significant myocardial depression, the consequences of acidosis can increase the toxicity of the drug.
- Ictal increases in plasma epinephrine levels may add to the potential risk for cardiac arrhythmias.
- airway reflexes are inhibited post-ictally, which adds to the potential for aspiration.
- an aggressive approach toward terminating seizures in the poisoned patient is warranted.
- Benzodiazepines are the drugs of choice to quickly terminate seizures, because they are lipophilic and rapidly enter the CNS.

specific investigations

- Urine drug screens are usually obtained in poisoned patients; however, there is no standardized screen. Interpretation of urine drug screen results depends on the clinician's knowledge of which toxins have been screened and whether confirmatory testing (ideally performed by a different analytic method) will follow.
- Quantitative serum drug testing is done when quantitation of a toxin is clinically relevant, as is the case for:
 - (i) paracetamol,
 - (ii) anticonvulsant agents,
 - (iii) salicylates,
 - (iv) digoxin ,
 - (v) ethanol,
 - (vi) ethylene glycol,
 - (vii) methanol,
 - (viii) iron,
 - (ix) lithium , and
 - (x) theophylline .

hypotension

general points

- Hypotension in the poisoned patient is most frequently caused by receptor blockade, drug-induced myocardial depression, or drug-induced vasodilatation.
- It is reflex to treat initial periods of hypotension with fluids; however, unless the poisoned patient is hypovolemic, large volumes of fluid may predispose the patient to the development of acute respiratory failure.
- The poisoned patient who is young and healthy responds to hypotension with an outpouring of endogenous catecholamines. Adrenergic receptors are sensitive in the young patient. Administration of further catecholamines may not be of benefit, because all catecholamine receptors are stimulated by endogenous catecholamines.

specific treatments

- Agents that must be considered for the treatment of hypotension in the poisoned patient are:
 - (i) sodium bicarbonate (for a sodium channel-blocking agent),
 - (ii) glucagon, and
 - (iii) insulin/glucose.
- Glucagon:
 - The cardiovascular effects of glucagon are mediated by myocardial glucagon receptors, which are catecholamine independent. Stimulation activates adenylate cyclase, leading to increased intracellular levels of the second messenger cAMP which increases myocardial calcium uptake.
 - Glucagon increases heart rate and stroke volume, thereby increasing cardiac output.
 - After intravenous administration, augmented inotropy is seen within 1 to 3 minutes, with a peak effect in 5 to 7 minutes.
 - An acceptable regimen is 10 mg of glucagon given over 10 minutes (rapid administration causes vomiting), followed by 1 to 3 mg/hour.
- Insulin & glucose:
 - Insulin improves contractility in anoxic rat hearts and improves the cardiac index after cardiopulmonary bypass surgery.
 - During drug-induced shock, insulin shifts myocardial fatty acid oxidation to carbohydrate oxidation which increases contractility
 - In the hypotensive poisoned patient, a reasonable dose of insulin is 10 units of regular insulin and 50 mL of 50% dextrose solution. This should be followed by infusion of 6 units of insulin per hour, with concurrent administration of sufficient glucose to maintain euglycemia.

cardiac arrhythmias

- Treatment of the arrhythmia must take into consideration the pharmacology of the toxin causing the arrhythmia.