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ISCCM
INDIAN SOCIETY OF CRITICAL CARE MEDICINE

ISCCM Textbook of Toxicology

Treating the Critically Ill Poisoned Patient



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1.4 Circulation in Poisoning

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INTRODUCTION

The circulatory system, comprising the cardiovascular and lymphatic systems, constitutes a sophisticated anatomical and physiological framework responsible for the orchestrated transport of oxygen, nutrients, and metabolic by-products. Poisons and toxins can cause disruption in the circulatory homeostasis and early recognition of a poison's circulatory impact is crucial, given that these disturbances may rapidly result in hemodynamic instability, shock, and multiorgan dysfunction.[1] A structured approach is essential to tailor interventions to the specific toxicological insult and the individual patient's hemodynamic profile.

India, with its diverse population, poses unique challenges in the management of critically sick poisoned patients. Factors such as availability of poisons over the counter, varied toxic exposures, resource limitations, and sociocultural influences necessitate a tailored approach. Acknowledging these challenges is vital for developing effective strategies to optimize circulatory support in poisoned patients in the Indian healthcare setting.

NORMAL CIRCULATION

Cardiac Cycle

The cardiac cycle, a rhythmic sequence, commences with atrial contraction, initiating ventricular filling, succeeded by ventricular contraction propelling blood into circulations. Diastole, the relaxation phase, allows ventricular filling, creating a cyclic repetition of events.[2]

The cardiac impulse, originating in the sinoatrial (SA) node, coordinates the cycle. Sodium ions (Na^+) move through funny channels, establishing a pacemaker potential. Calcium ion (Ca^{2+}) influx prompts depolarization, modulated by autonomic stimulation. Repolarization involves potassium efflux. The atrioventricular (AV) node introduces a controlled delay, and the bundle of His and Purkinje fibers transmit the impulse, ensuring ventricular contraction.[3]

The cardiac myocyte action potential consists of phases involving sodium and calcium movements, ensuring the heart's coordinated contraction and relaxation.[4] Excitation–contraction coupling involves L-type calcium channels, calcium-induced calcium release (CICR), and active calcium reuptake, ensuring effective cardiac muscle contractions.[5] Regulation of cardiac function involves inotropy, chronotropy, and factors affecting stroke volume (SV). Autonomic control, hormonal influence, and ion concentrations modulate inotropy and chronotropy. SV depends on preload, afterload, contractility, heart rate (HR), venous return, and ejection fraction, orchestrating optimal circulation and hemodynamic stability.[6]

Oxygen Delivery to Tissues

Tissue oxygen delivery is a dynamic process influenced by cardiac output, hemoglobin concentration (Hb), arterial oxygen saturation (SaO_2), and the efficiency of oxygen extraction. The equation for cardiac output is as follows: Cardiac output = heart rate (HR) × stroke volume (SV),

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highlighting the importance of HR and SV in determining the volume of oxygenated blood reaching tissues. Hb significantly impacts oxygen-carrying capacity, and the oxygen content (CaO_2) is calculated using the following equation: $CaO_2 = (Hb \times 1.34 \times SaO_2) + (0.003 \times PaO_2)$, where 1.34 is the oxygen-carrying capacity of hemoglobin, SaO_2 is arterial oxygen saturation, and PaO_2 is arterial oxygen partial pressure.[7]

Arterial oxygen saturation influences the oxygen–hemoglobin dissociation curve, impacting oxygen binding and release. Oxygen extraction ratio (O_2ER) is a critical factor, calculated as follows: $[(CaO_2 - CvO_2)/CaO_2] \times 100$, representing the proportion of oxygen delivered and utilized by tissues.[8]

Mechanisms of Circulatory Alterations in Poisoned Patients

See **Table 1**.

TABLE 1: Mechanisms of circulatory alterations in poisoned patients.

Mechanisms of circulatory alterations in poisoned patients	***Specific toxins and effects***
Myocardial toxicity	
Direct cardiotoxic agents	*Digitalis (e.g., foxglove):* Increased intracellular calcium, arrhythmias, impaired contractility
Ischemic injury	*Cocaine:* Coronary vasospasm, elevated risk of myocardial infarction
Arrhythmogenic effects	• *Certain antiarrhythmics:* Disruption of normal cardiac conduction system, potentially fatal arrhythmia • *Sympathomimetics (e.g., cocaine, amphetamines):* Precipitation of tachycardia due to sympathetic stimulation
Delayed effects	*Heavy metals and certain toxins:* Delayed cardiac effects, requiring prolonged monitoring and follow-up
Vascular effects	
Vasospasm	• *Ergot alkaloids:* Vasoconstriction, impaired tissue perfusion • *Sympathomimetics (e.g., cocaine):* Vasoconstriction, contributing to tissue ischemia and organ dysfunction
Vasodilation	• *Calcium channel blockers, beta-blockers:* Widespread vasodilation, drop in systemic vascular resistance • *Peripheral pooling of blood:* Exacerbates hypotension, necessitating aggressive interventions
Capillary leak syndrome	*Toxins and venoms:* Induce capillary leak syndrome, leading to fluid and protein extravasation, resulting in edema, hypovolemia, and compromised tissue perfusion
Coagulation abnormalities	*Warfarin, certain rodenticides:* Inhibit vitamin K–dependent clotting factors, increasing the risk of bleeding or thrombosis

CLINICAL EVALUATION OF CIRCULATION IN ACUTE CARE

Clinical assessment involves understanding the nature and dose of the poison, time since exposure, and any coingestions. Like any other critically sick patient in the emergency, poisoned patients must be evaluated in a structured template of primary survey (ABCDE approach). The assessment of circulatory status in critically ill poisoned patients is a cornerstone of emergency and critical care medicine.[9]

This includes vital signs, physical examination, and a focused bedside assessment by ultrasound and point-of-care testing (POCT) labs.

Heart Rate

Alterations in HR may be an early pointer to potential hemodynamic instability in poisoned patients. Tachycardia reflects sympathetic activation. However, bradycardia may also occur **(Table 2)**. Unstable tachyarrhythmias/bradyarrhythmias will require immediate stabilization. Serial monitoring of HR provides valuable insights into the evolving clinical course.

Blood Pressure

Hypotension is a red flag for circulatory compromise and may indicate severe poisoning. Elevated blood pressure (BP) may be observed **(Table 2)**. BP trends guide fluid resuscitation, vasopressor therapy, and antidote response, when administered.

Respiratory Rate

Respiratory compromise may occur due to direct respiratory depressant effects or secondary to cardiovascular collapse. Tachypnea may result from acid–base

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TABLE 2: Change in ABG parameters and their significance.

ABG parameter	*Significance*
Anion gap calculation	Elevated anion gap points toward specific toxins (MUDPILES)
Lactate levels	Monitoring aids in assessing shock severity and guiding resuscitation; elevated levels suggest certain toxins (e.g., cyanide, CO poisoning)
Pattern recognition	Identifying acid–base patterns inform about underlying toxins (e.g., respiratory alkalosis and metabolic acidosis in salicylate toxicity)
Other pearls and pitfalls	• Tricyclic antidepressants have increased toxicity in acidic milieu and hence acidosis must be aggressively treated • Patients with severe metabolic acidosis may land into peri-intubation cardiac arrest if minute ventilation is not maintained after paralysis

(ABG: arterial blood gas; CO: carbon monoxide)

disturbances or hypoxia, underscoring the need for respiratory support.

Temperature

Hyperthermia or hypothermia may accompany certain poisonings, influencing metabolic demands and exacerbating cardiovascular instability **(Table 2)**.

Oxygen Saturation

Continuous monitoring of oxygen saturation (SpO_2) is essential, particularly in cases where respiratory compromise is anticipated. It guides the need for supplemental oxygen and provides insights into the adequacy of tissue oxygenation.

Capillary Refill Time

Prolonged capillary refill time (CRT) is indicative of impaired peripheral perfusion. This simple yet informative bedside test aids in assessing the adequacy of cardiac output and vascular tone.

Skin and Mucous Membranes

Evaluation of skin color, temperature, and moisture provides insights into perfusion status and type of poison. Sympathomimetic and cholinergic toxidromes manifest as sweaty skin, whereas anticholinergic agents lead to dry and flushed skin. Cyanosis, mottling, or cool extremities may indicate compromised tissue perfusion.

Point-of-Care Ultrasound

Point-of-care ultrasound (POCUS) is a pivotal tool in rapidly assessing the cause of shock at the patient's bedside.[10-13]

Point-of-care ultrasound facilitates the identification of hypovolemia by assessing the inferior vena cava, detects pericardial effusion, and evaluates cardiac chambers and valves. Additionally, it assesses pulmonary causes, identifies deep venous thrombosis, and performs focused abdominal assessments.[14]

Various well-established ultrasound protocols like RUSH (Rapid Ultrasound in Shock and Hypotension) and FALLS (Fluid Administration Limited by Lung Sonography) may be leveraged for assessment and emergency management of poisoned patients who are in shock.[15-17]

Point-of-care ultrasound also aids in monitoring fluid responsiveness, guiding procedural interventions, and providing real-time insights for intervention assessment. However, its effective use requires clinician expertise and integration with clinical context for accurate shock diagnosis and management.[18]

ACID–BASE ALTERATIONS

Poisoned patients presenting with shock often exhibit complex acid–base disturbances, providing valuable insights into the underlying poison and guiding resuscitation strategies.[19-23]

ELECTROCARDIOGRAM CHANGES

Electrocardiogram (ECG) changes in poisoned patients can provide critical insights into the toxic effects on the cardiovascular system. These alterations may range from mild abnormalities to life-threatening arrhythmias. Understanding the mechanisms behind these ECG changes is crucial for prompt recognition and appropriate management. **Table 3** mentions the various ECG changes in poisoned patients.[24-26]

PRINCIPLES OF RESUSCITATION IN ACUTELY POISONED PATIENTS

The overall schematic for the circulatory resuscitation in poisoned patients is shown in **Flowchart 1**.

Fluids

Fluids have conventionally been the first treatment of choice for the management of shock. There is no

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high-quality evidence to guide the fluid therapy in toxin-related shock. Evidence from Surviving Sepsis Campaign guidelines of 2021 for patients with septic shock suggests the use of 30-mL/kg intravenous bolus.[27]

TABLE 3: Important ECG changes in poisoning.

ECG change	*Mechanism*	*Examples of toxins*
QT prolongation	• Inhibition of cardiac repolarization (K^+ blockade) • Risk of TdP	• Antiarrhythmics (quinidine) • Antibiotics (macrolides) • Tricyclic antidepressants
Bradyarrhythmias	Blockade of the cardiac conduction system	• Beta-blockers • Calcium channel blockers • Digoxin
Tachyarrhythmias	Increased sympathetic stimulation	• Cocaine • Amphetamines • Theophylline
• QRS widening • Right-axis deviation • Tachyarrhythmias	Sodium channel blockade	• Tricyclic antidepressants • Carbamazepine • Antiarrhythmics • Cocaine • Insecticides
ST-segment changes	Induction of ischemic or nonischemic conditions affecting myocardium	• Cocaine • Digitalis toxicity
Osborn (J) waves	Hypothermia, hypercalcemia	Sedatives, alcohols

(ECG: electrocardiogram; TdP: torsades de pointes)

This recommendation of 30-mL/kg fluids is based on PROCESS, PROMISE, and ARISE trials, which used the 30-mL/kg fluids for resuscitation.[28-30]

Our primary goals are to increase the preload to the heart, increase the ventricular filling, and improve the tissue perfusion. Aggressive fluid resuscitation may result in an increased risk of pulmonary edema and poor outcomes.[31,32] Using pulse index continuous cardiac output (PiCCO)–based fluid resuscitation does not improve outcomes as compared with central venous pressure–based management of sepsis and patients with septic shock with fluid resuscitation.[33] Thus, we recommend giving fluid boluses and continuous assessment of multiple end points of tissue perfusion and POCUS guidance. The choice of intravenous fluids has been a matter of debate for decades. In general, crystalloids are favored over colloids based on the literature from other patient populations. There is some evidence to favor balanced salt solution over normal saline;[34,35] however, some evidence suggests no such benefit.[36]

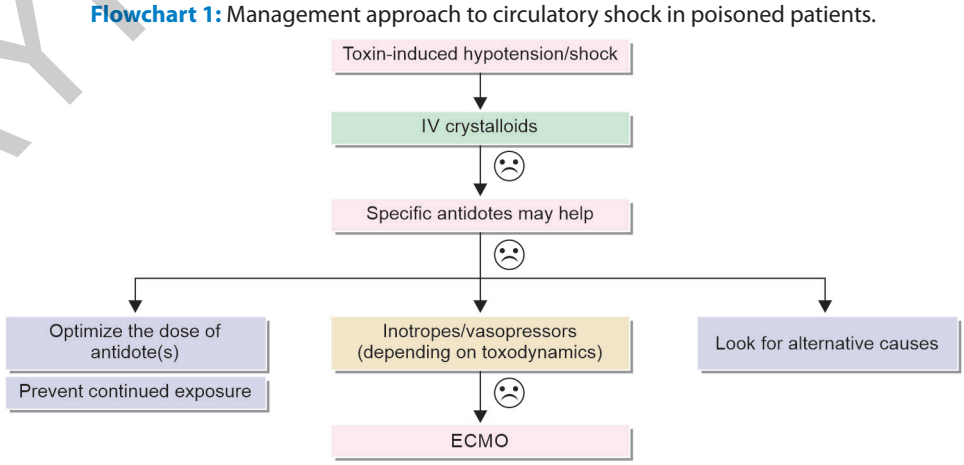
Antidotes

Most poisoned patients will require supportive management with fluids, vasopressors, etc. However, some poisons respond promptly to the administration of their antidotes. These agents (remembered as ABCDEN) must be readily available to the treating physician if the clinical condition demands **(Table 4)**.

Vasopressors

Vasopressors are useful for patients who have persistent shock after fluid resuscitation, or those patients who are not hypovolemic. The usual initial vasopressor of choice

Flowchart 1: Management approach to circulatory shock in poisoned patients.



(ECMO: extracorporeal membrane oxygenation; IV: intraventricular)

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TABLE 4: Specific antidotes in poisoning.

Resuscitation antidotes	*Useful in poisoning of*
Atropine	• Organophosphates • Beta-blocker, calcium channel blocker overdose
Antivenom (polyvalent)	Hemotoxic/vasculotoxic snake bite
Bicarbonate	Sodium channel blocking agents (tricyclic antidepressants, carbamazepine, etc.)
Calcium gluconate	• Life-threatening hyperkalemia • Hydrofluoric acid
Cyanide kit	Cyanide poisoning
DigiFab	• Digoxin overdose • Oleander poisoning
Dextrose	Hypoglycemic agents (insulin, sulfonylureas, valproate, salicylates, etc.)
Emulsion (intralipids)	Local anesthetic systemic toxicity (LAST)
Naloxone	Opioid overdose

is norepinephrine (0.1–2 µg/kg/min), epinephrine (2–10 µg/min) being the second-line vasopressor. Dopamine is out of favor as a preliminary choice of vasopressor as it has no role in renal protection and has an increased risk of tachyarrhythmias.[27] Dobutamine can be used where there is myocardial dysfunction, or low cardiac output despite fluid therapy. The evidence for the use of vasopressors is extrapolated from patients with septic shock as the data on poisoned patients are lacking. Phenylephrine is usually not used due to a greater risk of reduced cardiac output and reduced tissue perfusion.[37]

The role of inodilators like milrinone in patients with cardiac failure due to toxicological causes is weak and has no robust evidence.[38] Levosimendan is a calcium channel sensitizer with additional phosphodiesterase 3 (PDE3) inhibitory properties; its use in calcium channel blocker toxicity with circulatory failure is supported by case reports but robust evidence is lacking.[39] Similarly, the role of methylene blue in calcium channel blocker toxicity is supported by a few case reports of improving the mean arterial pressure when given along with conventional therapies and vasopressors.

There are data available that favor the use of high-dose insulin dextrose therapy (1–10 U/kg/h) doses in beta-blockers, and calcium channel blocker toxicities.[40-42] Insulin is an ionotropic agent and is a potent vasodilator, thereby reducing systemic vascular resistance and increasing the cardiac output in poisoned patients.

Extracorporeal Interventions

There is building evidence that poisoned patients with refractory shock managed on venoarterial (VA) extracorporeal membrane oxygenator have a lower mortality compared with patients managed on antidotal therapy and supportive medication because the multiorgan dysfunction is prevented in the extracorporeal membrane oxygenation (ECMO) subset and the kidneys and liver recover over time to flush the toxins out of the system.[43] So VA-ECMO may be used in patients with refractory shock and refractory dysrhythmias leading to cardiovascular collapse in poisoned patients.

MONITORING THE ACUTELY ILL POISONED PATIENT

The resuscitation must continue till the microcirculatory end points are reached. **Table 5** includes common clinical monitoring parameters to be considered. However, the monitoring must be individualized to the patient's clinical condition. End points of resuscitation are mentioned in **Table 6**.

COMMON CAUSES OF HEMODYNAMIC COLLAPSE IN TOXICOLOGY

Calcium Channel Blockers

Calcium channel blockers are a leading cause of mortality in poisoning patients.[44] There are two major types of calcium channel blockers—the dihydropyridines (DHPs) and non-DHPs. These act on the slow voltage-gated L-type calcium channels; however, at supratherapeutic doses, the difference of action between the non-DHP and DHP varieties is not maintained. Sinus bradycardia is the common presentation of calcium channel blocker toxicity, although all types of heart blocks can occur with no particular pattern or predilection. Calcium channel blockade can also lead to sodium channel blockade leading to a delay in phase 0 depolarization, resulting in rhythms like ventricular premature contractions and sometimes asystole.[24] Class 1 recommendation is to start vasopressors in conjunction with high-dose insulin therapy. Administering calcium is a class 2a recommendation and VA-ECMO is a class 2a recommendation. The role of intravenous lipid emulsion is placed at class 3 recommendation with no role in the management of calcium channel blocker toxicity.[45]

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TABLE 5: Clinical monitoring parameters in poisoned patients.

Parameters	*Monitoring strategy*	*Clinical significance*
Vital signs	Continuous monitoring of BP, HR, and RR	Early indicators of shock, guide fluid resuscitation
Continuous cardiac monitoring	Continuous ECG monitoring	Detects arrhythmias, guides targeted interventions
Hemodynamic monitoring	CVP monitoring, arterial catheterization	Provides insights into intravascular volume status, guides vasopressor use
Oxygenation and ventilation	Pulse oximetry, end-tidal CO_2 monitoring	Assess oxygenation status, detect changes in respiratory status
Temperature monitoring	Continuous temperature monitoring	Identifies hyperthermia or hypothermia
Neurological monitoring	GCS	Evaluates neurological status, detects changes in mental status
Blood gases	Serial blood gas analysis, lactate levels	Provides insights into acid–base status, assesses tissue perfusion
Fluid balance and urine output	Hourly urine output monitoring, fluid input and output monitoring	Assesses renal perfusion, prevents fluid overload
Clinical assessment	Serial clinical examination	Detects subtle changes indicative of ongoing shock

(BP: blood pressure; CVP: central venous pressure; ECG: electrocardiogram; GCS: Glasgow Coma Scale; HR: heart rate; RR: respiratory rate)

TABLE 6: End points of resuscitation in poisoned patients.

Category	*Parameter*	*End points*
Peripheral perfusion	• Capillary refill time • Skin mottling • Peripheral temperature	• <3 seconds • Absent • Warm
Venous oxygen saturation	• Central • Mixed	• ≥65–70% • ≥60–65%
Lactate	• Absolute value • Lactate clearance	• <2 mmol/L • >2 mmol/h
Urine output		>0.5 mL/kg/h

Cocaine Toxicity

It produces a sympathomimetic syndrome because of the effect cocaine has on multiple channels across the spectrum. Tachycardia is because by postsynaptic beta-receptor agonism with hypertension due to postsynaptic alpha-adrenergic agonism, it blocks the reuptake of norepinephrine and serotonin in the central nervous system (CNS) leading to neuropsychiatric manifestation.[46,47] Cocaine blocks sodium channel receptors leading to the prolongation of phase 0 of the cardiac cycle increasing the risk of wide complex tachycardias; the potassium efflux blockade leads to QT prolongation. Class 1 recommendation is to start external cooling if there is hyperthermia; class 2a recommendations are to give sodium bicarbonate if there are wide complex tachycardias and also lidocaine. Given the vasospasm and hypertensive emergency it produces, calcium channel blockers and nitrates are class 2a recommendations. Beta-blockers are not recommended.[45]

Sympathomimetic Toxicity

Sympathomimetics result in tachycardia, hypertension, agitation, seizures, hyperpyrexia, rhabdomyolysis, and acidosis.[48,49] Cardiac rhythm anomalies like ventricular tachycardia, fibrillation, pulseless electrical activity,[49,50] or myocardial infarction with normal coronaries can occur; takotsubo cardiomyopathy is a dreaded complication, although it resolves spontaneously within weeks and requires supportive circulatory care.[51]

Class 1 recommendation is controlling agitation and external cooling in cases of hyperthermia.[45]

Organophosphate and Carbamate Toxicity

In organophosphate and carbamate (OPC) poisoning, class 1[45] recommendation will be early administration of atropine at doses of 1–2 mg intravenous dose with doubling the dose every 5 minutes and, once atropinization is reached, then starting 10–20% of the atropinization dose as infusion till 48 hours of symptom resolution.[52] Early use of atropine is indicated in suspected OPC poisoning leading to bronchorrhea, bradycardia, cardiac arrest, and bronchospasm. Atropinization is defined by a clear chest on auscultation, a HR greater than 80, and systolic BP greater than 80 mm Hg.

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Sodium Channel Blocker Toxicity

These agents cause prolongation of phase 0 of the cardiac cycle leading to QRS prolongation, ventricular arrhythmias, and, in extreme poisoning, sine wave and asystole. The patient is also prone to developing ventricular arrhythmias due to intraventricular conduction delays and conduction blocks. Bradyarrhythmias are not common but are possible due to their effects on the pacemaker cells. Intraventricular conduction delays with a right-terminal R deviation in aVR lead are characteristic ECG changes in tricyclic antidepressant (TCA) toxicity **Figure 1**.[53] The class 1 indication would be using sodium bicarbonate at a dose of 100–150 mEq bolus followed by infusions at 1–3 mL/kg/h of 150 mEq/L sodium bicarbonate solutions. Management is continued till hypotension improves and the dysrhythmia ceases. pH should be maintained between 7.50 and 7.55, and serum sodium between 150 and 155 mEq/L. Lidocaine as a class 2 recommendation for ventricular dysrhythmia is recommended. VA-ECMO can be tried if there is refractory cardiogenic shock despite the best medical management.[45] The role of lipid emulsions is questionable and is suggested if all treatment modalities fail.

Cardiac Glycosides

Cardiac glycosides include a variety of compounds like digoxin, digitoxin, yellow oleander, and toad toxins. They are versatile in causing any type of dysrhythmia. Digitalis effect is a flattened T wave with a scooped ST-segment depression. The toxic effects of digitalis are because of increased automaticity with prolonged AV conduction. Premature ventricular contractions are very commonly found in digitalis toxicity.[24] Apart from that, AV blocks, tachyarrhythmias like paroxysmal atrial tachycardia with associated AV blocks, accelerated junction tachycardia, or atrial fibrillation with a slow ventricular rate are characteristic ECG findings of cardiac glycoside toxicity. Class 1 recommendation is using digoxin fab to reverse the effects of the offending agent **(Fig. 2)**. Class 2 recommendations advise using digoxin fab to treat yellow oleander toxicity **(Fig. 3)**. Extracorporeal hemoperfusion hemodialysis or plasmapheresis is designated as having no role in the management of the above-mentioned poisoning.[45]

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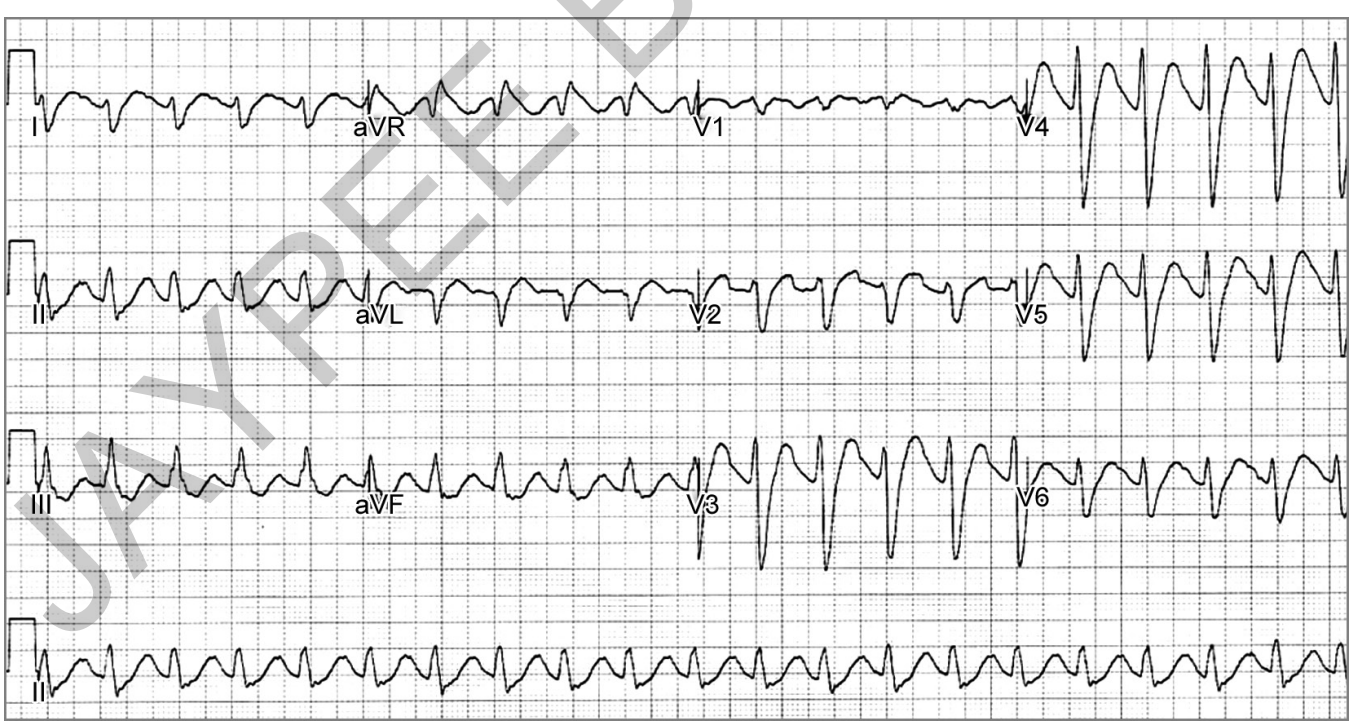

Fig. 1: TCA overdose. (TCA: tricyclic antidepressant)

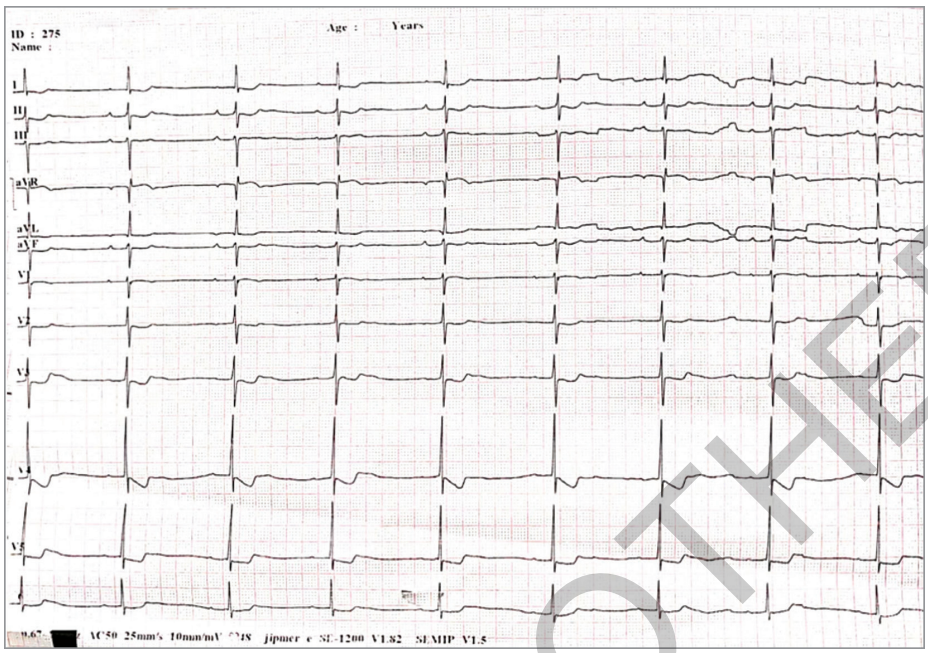

Fig. 2: Reverse tick digoxin effect.

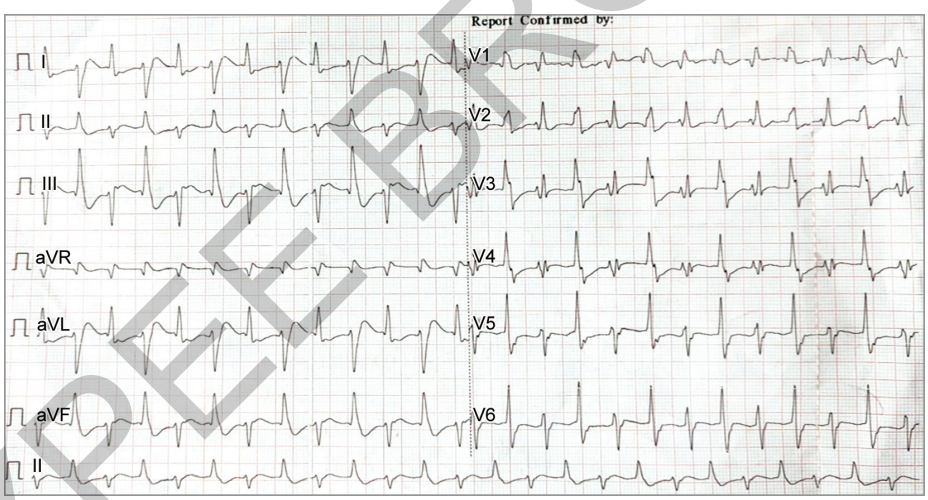

Fig. 3: Bidirectional VT of digoxin toxicity. (VT: ventricular tachycardia)

Local Anesthetic Systemic Toxicity

In local anesthetic systemic toxicity, we get CNS effects in 77–89% cases while (32–55%) will have cardiovascular symptoms. Bupivacaine is the most cardiotoxic local anesthetic because of its predilection to affect the cardiac calcium channels leading to conduction delays and reentry arrhythmias like ventricular fibrillation.[54] Acidemia and hypoxia lead to a worsening of the toxicity of local anesthetics.[55] Bradycardia is the first sign that the local anesthetic toxicity will lead to cardiovascular compromise. Class 1 recommendation is to use in vascular lipid emulsions of 20% at the dose of 1.5 mL/kg and then an infusion of 0.25 mL/kg/min for 30 minutes. Class 2 recommendations include using sodium bicarbonate for broad complex tachycardias and atropine for bradycardias. Avoidance of acidemia and hypoxia also precedes this step, with the American Heart Association (AHA) advising to use benzodiazepines to control seizures as a class 1 indication.[45]

Cyanide Toxicity

Cyanide is another mitochondrial toxin we need to keep in mind when dealing with cases that present with unexplained acidosis, cardiovascular collapse, and

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lactatemia. It is an obscure diagnosis.[56,57] Class 1 indication is the administration of hydroxocobalamin 5 g intravenous stat. Sodium nitrate is also a class 1 indication but the complications of methemoglobinemia have to be kept in mind during sodium nitrate administration.[45]

Beta-blocker Toxicity

Beta-blocker toxicity is a common cause of mortality in toxicology.[24] Patients can present with hypotension, bradycardia, and cardiovascular collapse. The AHA recommends the use of high-dose insulin therapy as a class 1 indication of hypotension along with vasopressor support. Glucagon is recommended as a class 2 recommendation for bradycardia management in this subset with no role for intravenous lipid emulsion.[45]

CONCLUSION

- Any discrepancies in paired occurrences, orthostatic hypotension, sudden onset of hypotension or hypertension, and suspicion of a toxin affecting the circulation should be thought of.
- ECGs are primordial in evaluating any case of poisoning.
- Preload augmentation and initiation of vasopressors are advised but early initiation of specific antidotal agents or high-dose insulin therapy is recommended at the earliest for better outcomes and to reduce the need for vasopressor support.
- Increasing evidence and recommendation for the use of extracorporeal systems in cases of refractory cardiogenic shock after maximal medical management.
- Tissue perfusion–based end points for resuscitation seem to be better and easier in continuous monitoring and also give a physiological basis for monitoring of the resuscitative efforts in circulatory failure.

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1.5 Disability Assessment in a Critically Ill Poisoned Patient

Manoj Goyal, Ritushree, Kathik, Ashish Bhalla

INTRODUCTION

Management of a patient with impaired consciousness (i.e., brain failure) represents one of the most challenging clinical scenarios and demands an urgent remedial action, more so as alteration of sensorium often indicates impending irreversible brain damage. Intoxication with drugs accounts for approximately one third of all patients presenting with coma to the emergency department.[1]

INITIAL RESUSCITATION

The first step in management of any critically ill poisoned patient (as is true for any patient presenting with altered sensorium irrespective of etiology) is quick assessment along with maintenance of airway, breathing, and circulation.[2] In any patient with stupor or coma, intubation is necessary to protect airway and prevent aspiration. Many drugs/poisons reduce ventilatory drive per se, resulting in CO_2 narcosis, further depressing the central nervous system (CNS). Often, patients in the emergency department are placed on high-flow oxygen therapy, resulting in adequate oxygen saturation values despite poor ventilatory drive. Thus, wherever possible, arterial blood gas measurement should be included as standard of care for management of patients with altered sensorium.

For maintenance of circulation, all patients with drug-induced coma and hypotension shall have two intravenous lines: One large-bore peripheral intravenous catheter and second intravenous line placed in either the peripheral or the central circulation.[3] For a poisoned patient, attention shall be given to specific poison while choosing the vasopressor agent if needed. Norepinephrine may be a better drug for hypotension secondary to poisoning.[3,4]

Many of the poisoned patients present with deep coma (opiates, sedative–hypnotics) and poor respiratory efforts. These patients need securing of the airway and ventilation as initial resuscitation. Seizures are common in organophosphate (OP) poisoning and opiate, sedative, and alcohol withdrawal and may be aborted. Benzodiazepines are the drug of choice for aborting and controlling toxic seizures.

ASSESSMENT AND DIFFERENTIAL DIAGNOSIS (TABLES 1 AND 2)

History

The first step in the evaluation of any patient with altered sensorium is to obtain good history. As most patients with altered sensorium cannot give history, it shall be obtained from relatives or friends. Whenever possible, try to obtain

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ISCCM Textbook of Toxicology

Treating the Critically Ill Poisoned Patient

This book on the treatment of critically ill poisoned patient is an attempt to fill the gap in a common and frequently encountered problem in critical care units in India. The spectrum of toxicological problems is unique to geographical regions and hence this book assumes much relevance given that there is limited content available on the common toxicological problems in India in a single publication. The chapters are written by intensivists for intensivists in training as well as those who have been practicing critical care. The book focuses on the approach to and management of the common toxicological problems in India. The first section provides an overview of the comprehensive evaluation of a poisoned patient and forms the platform for the subsequent sections. The second section is on specific poisons and deals with the common as well as the not-so-common toxicological conditions in India with a focus on critical care aspects. The third section deals with supportive therapy in poisoning with the application of the principles of organ support in toxicology. The final section is an evidence-based approach on the use of antidotes in poisoning.

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